The GALE ENCYCLOPEDIA of MENTAL DISORDERS

VOLUME

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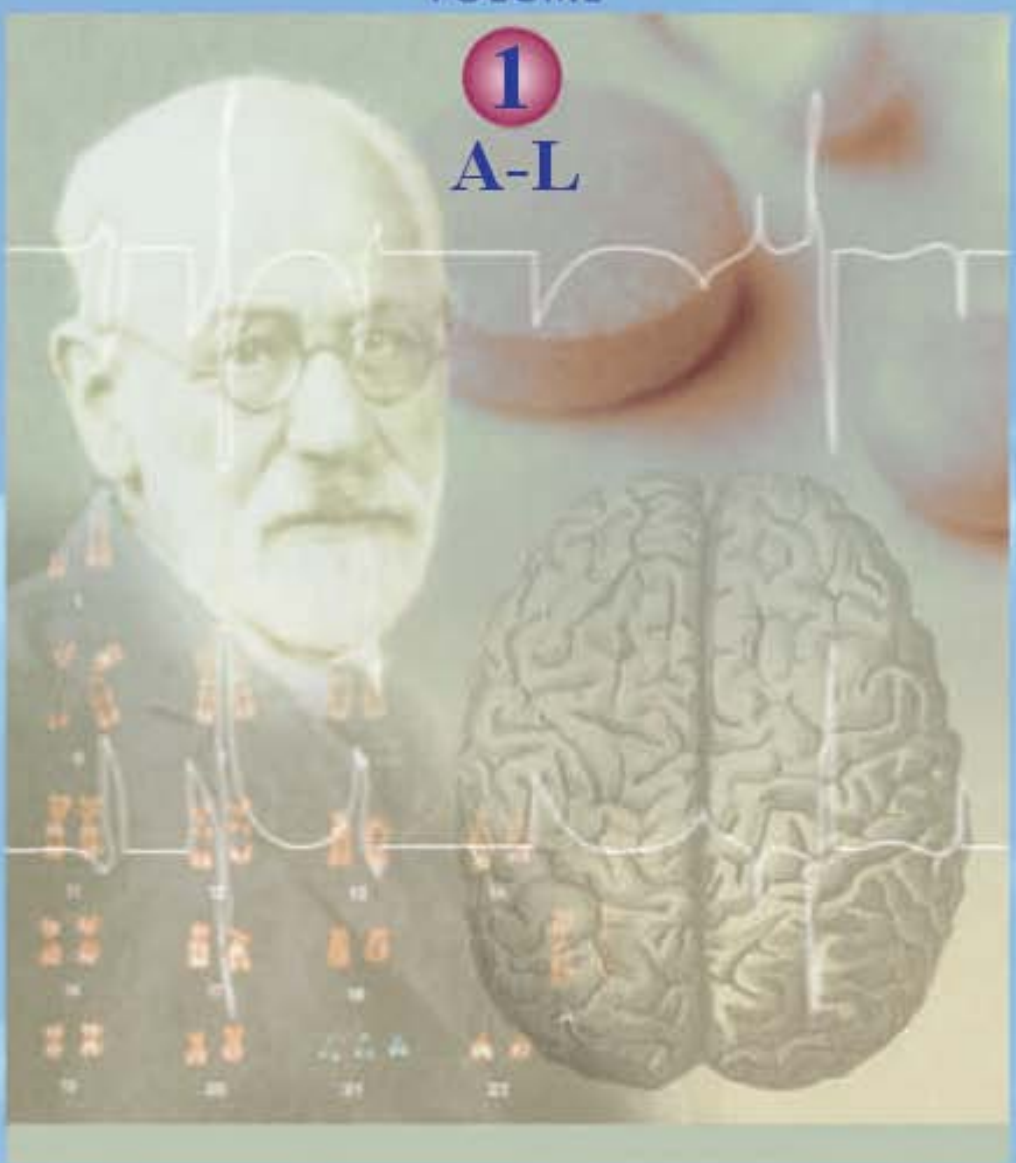

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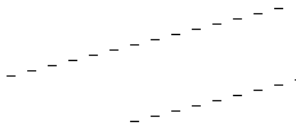

The Gale Encyclopedia of Mental Disorders

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PLEASE READ—IMPORTANT INFORMATION

The *Gale Encyclopedia of Mental Disorders* is a medical reference product designed to inform and educate readers about a wide variety of mental disorders, diagnostic techniques and tests, therapies, and psychiatric medications. The Gale Group believes the product to be comprehensive, but not necessarily definitive. It is intended to supplement, not replace, consultation with a physician or other health care practitioner. While the Gale Group has made substantial efforts to provide information that is accurate, comprehensive, and up-to-date, the Gale Group makes no representations or warranties of any kind, including without limitation, warranties of merchantability or fitness for a particular purpose, nor does it guarantee the accuracy, comprehensiveness, or timeliness of the information contained in this product. Readers should be aware that the universe of medical knowledge is constantly growing and changing, and that differences of medical opinion exist among authorities. Readers are also advised to seek professional diagnosis and treatment of any medical condition, and to discuss information obtained from this book with their health care provider.

INTRODUCTION

The *Gale Encyclopedia of Mental Disorders* is a valuable source of information for anyone who wants to learn more about mental disorders and their treatments. This collection of approximately 400 entries provides in-depth coverage of specific disorders recognized by the American Psychiatric Association (as well as some disorders not formally recognized as distinct disorders), diagnostic procedures and techniques, therapies, and psychiatric medications. In addition, entries have been included to facilitate understanding of related topics, such as Advance directives, Crisis housing, and Neurotransmitters.

This encyclopedia minimizes medical jargon and uses language that laypersons can understand, while still providing thorough coverage that will benefit health science students as well.

Entries follow a standardized format that provides information at a glance. Rubrics include:

Disorders	**Medications**
Definition	Definition
Description	Purpose
Causes and symptoms	Description
Demographics	Recommended dosage
Diagnosis	Precautions
Treatments	Side effects
Prognosis	Interactions
Prevention	Resources
Resources	

INCLUSION CRITERIA

A preliminary list of mental disorders and related topics was compiled from a wide variety of sources, including professional medical guides and textbooks, as well as consumer guides and encyclopedias. The advisory board, made up of professionals from a variety of health care fields including psychology, psychiatry, pharmacy, and social work, evaluated the topics and made suggestions for inclusion. Final selection of topics to include was made by the advisory board in conjunction with the Gale editor.

ABOUT THE CONTRIBUTORS

The essays were compiled by experienced medical writers, including physicians, pharmacists, and psychologists. The advisors reviewed the completed essays to ensure that they are appropriate, up-to-date, and accurate.

HOW TO USE THIS BOOK

The *Gale Encyclopedia of Mental Disorders* has been designed with ready reference in mind.

- Straight **alphabetical arrangement** of topics allows users to locate information quickly.
- **Bold-faced terms** within entries direct the reader to related articles.
- **Cross-references** placed throughout the encyclopedia direct readers from alternate names, drug brand names, and related topics to entries.
- A list of **key terms** is provided where appropriate to define unfamiliar terms or concepts. A **glossary** of key terms is also included at the back of Volume II.
- The **Resources** sections direct readers to additional sources of information on a topic.
- Valuable **contact information** for organizations and support groups is included with many of the disorder entries.
- A leaf graphic () inserted next to the entry title denotes entries about herbals (such as Ginkgo biloba) or dietary supplements (such as SAMe). These entries have the same rubrics as the medication entries; however, the graphic is to draw attention to the fact that these entries are not about prescription medications.
- A **Symptoms list** at the back of Volume II has been included *not* for diagnosis but to reveal patterns in

symptoms and disorders and to provide a starting point for research or discussion with a health care provider.

- A comprehensive **general index** guides readers to all topics mentioned in the text.

GRAPHICS

The *Gale Encyclopedia of Mental Disorders* contains 100 illustrations, photos, and tables. A color insert in each volume has been included to enhance certain photos shown in the text in black and white.

ADVISORY BOARD

A number of experts in medicine, psychology, psychiatry, and pharmacy provided invaluable assistance in the formulation of this encyclopedia. The members of the advisory board performed a myriad of duties, from defining the scope of coverage to reviewing individual entries for accuracy and accessibility. The editor would like to express appreciation to them for their time and for their contributions.

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A

Abnormal Involuntary Movement Scale

Definition

The Abnormal Involuntary Movement Scale (AIMS) is a rating scale that was designed in the 1970s to measure involuntary movements known as **tardive dyskinesia** (TD). TD is a disorder that sometimes develops as a side effect of long-term treatment with neuroleptic (antipsychotic) medications.

Purpose

Tardive dyskinesia is a syndrome characterized by abnormal involuntary movements of the patient's face, mouth, trunk, or limbs, which affects 20%–30% of patients who have been treated for months or years with neuroleptic medications. Patients who are older, are heavy smokers, or have diabetes mellitus are at higher risk of developing TD. The movements of the patient's limbs and trunk are sometimes called choreathetoid, which means a dance-like movement that repeats itself and has no rhythm. The AIMS test is used not only to detect tardive dyskinesia but also to follow the severity of a patient's TD over time. It is a valuable tool for clinicians who are monitoring the effects of long-term treatment with neuroleptic medications and also for researchers studying the effects of these drugs. The AIMS test is given every three to six months to monitor the patient for the development of TD. For most patients, TD develops three months after the initiation of neuroleptic therapy; in elderly patients, however, TD can develop after as little as one month.

Precautions

The AIMS test was originally developed for administration by trained clinicians. People who are not health care professionals, however, can also be taught to administer the test by completing a training seminar.

Description

The entire test can be completed in about 10 minutes. The AIMS test has a total of twelve items rating involuntary movements of various areas of the patient's body. These items are rated on a five-point scale of severity from 0–4. The scale is rated from 0 (none), 1 (minimal), 2 (mild), 3 (moderate), 4 (severe). Two of the 12 items refer to dental care. The patient must be calm and sitting in a firm chair that doesn't have arms, and the patient cannot have anything in his or her mouth. The clinician asks the patient about the condition of his or her teeth and dentures, or if he or she is having any pain or discomfort from dentures.

The remaining 10 items refer to body movements themselves. In this section of the test, the clinician or rater asks the patient about body movements. The rater also looks at the patient in order to note any unusual movements first-hand. The patient is asked if he or she has noticed any unusual movements of the mouth, face, hands or feet. If the patient says yes, the clinician then asks if the movements annoy the patient or interfere with daily activities. Next, the patient is observed for any movements while sitting in the chair with feet flat on the floor, knees separated slightly with the hands on the knees. The patient is asked to open his or her mouth and stick out the tongue twice while the rater watches. The patient is then asked to tap his or her thumb with each finger very rapidly for 10–15 seconds, the right hand first and then the left hand. Again the rater observes the patient's face and legs for any abnormal movements.

After the face and hands have been tested, the patient is then asked to flex (bend) and extend one arm at a time. The patient is then asked to stand up so that the rater can observe the entire body for movements. Next, the patient is asked to extend both arms in front of the body with the palms facing downward. The trunk, legs and mouth are again observed for signs of TD. The patient then walks a few paces, while his or her gait and hands are observed by the rater twice.

KEY TERMS

Choreathetoid movements—Repetitive dance-like movements that have no rhythm.

Clozapine—A newer antipsychotic medication that is often given to patients who are developing signs of tardive dyskinesia.

Neuroleptic—Another name for the older antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

Syndrome—A group of symptoms that together characterize a disease or disorder.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

Results

The total score on the AIMS test is not reported to the patient. A rating of 2 or higher on the AIMS scale, however, is evidence of tardive dyskinesia. If the patient has mild TD in two areas or moderate movements in one area, then he or she should be given a **diagnosis** of TD. The AIMS test is considered extremely reliable when it is given by experienced raters.

If the patient's score on the AIMS test suggests the diagnosis of TD, the clinician must consider whether the patient still needs to be on an antipsychotic medication. This question should be discussed with the patient and his or her family. If the patient requires ongoing treatment with antipsychotic drugs, the dose can often be lowered. A lower dosage should result in a lower level of TD symptoms. Another option is to place the patient on a trial dosage of **clozapine** (Clozaril), a newer antipsychotic medication that has fewer side effects than the older neuroleptics.

See also Medication-induced movement disorders; Schizophrenia

Resources

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American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 4th edition, text revised. Washington, DC: American Psychiatric Association, 2000.

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Jeste, Dilip V., M.D., and others. "Incidence of Tardive Dyskinesia in Early Stages of Low Dose Treatment With Typical Neuroleptics in Older Patients." *American Journal of Psychiatry* February 1999: 309-311.

Ondo, William G., M.D., and others. "Tetrabenazine Treatment for Tardive Dyskinesia: Assessment by Randomized Videotape Protocol." *American Journal of Psychiatry* August 1999: 1279-1281.

ORGANIZATIONS

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National Institute of Mental Health (NIMH). 6001 Executive Boulevard, Room 8184, Bethesda, MD, 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

Susan Hobbs, M.D.

Abuse

Definitions

Abuse is a complex psychosocial problem that affects large numbers of adults as well as children throughout the world. It is listed in the *Diagnostic and Statistic Manual of Mental Disorders* (*DSM-IV-TR*) under the heading of "Other Conditions That May Be a Focus of Clinical Attention." Although abuse was first defined with regard to children when it first received sustained attention in the 1950s, clinicians and researchers now recognize that adults can suffer abuse in a number of different circumstances. Abuse refers to harmful or injurious treatment of another human being that may include physical, sexual, verbal, psychological/emotional, intellectual, or spiritual maltreatment. Abuse may coexist

with **neglect**, which is defined as failure to meet a dependent person's basic physical and medical needs, emotional deprivation, and/or desertion. Neglect is sometimes described as passive abuse.

The costs of abuse to society run into billions of dollars annually in the United States alone. They include not only the direct costs of immediate medical and psychiatric treatment of abused people but also the indirect costs of learning difficulties, interrupted education, workplace absenteeism, and long-term health problems of abuse survivors.

Types of abuse

Physical

Physical abuse refers to striking or beating another person with the hands or an object, but may include assault with a knife, gun, or other weapon. Physical abuse also includes such behaviors as locking someone in a closet or other small space, depriving someone of sleep, burning, gagging, or tying them up, etc. Physical abuse of infants may include shaking them, dropping them on the floor, or throwing them against the wall or other hard object.

Sexual

Sexual abuse refers to inappropriate sexual contact between a child or an adult and someone who has some kind of family or professional authority over them. Sexual abuse may include verbal remarks, fondling or kissing, or attempted or completed intercourse. Sexual contact between a child and a biological relative is known as incest, although some therapists extend the term to cover sexual contact between a child and any trusted caregiver, including relatives by marriage. Girls are more likely than boys to be abused sexually; according to a conservative estimate, 38% of girls and 16% of boys are sexually abused before their eighteenth birthday.

Verbal

Verbal abuse refers to regular and consistent belittling, name-calling, labeling, or ridicule of a person; but it may also include spoken threats. It is one of the most difficult forms of abuse to prove because it does not leave physical scars or other evidence, but it is nonetheless hurtful. Verbal abuse may occur in schools or workplaces as well as in families.

Emotional/psychological

Emotional/psychological abuse covers a variety of behaviors that hurt or injure others even though no physical contact may be involved. In fact, emotional abuse is a stronger predictor than physical abuse of the likelihood of **suicide** attempts in later life. One form of emotional abuse involves the destruction of someone's pet or valued possession in order to cause pain. Another abusive behavior is emotional blackmail, such as threatening to commit suicide unless the other person does what is wanted. Other behaviors in this category include the silent treatment, shaming or humiliating someone in front of others, or punishing them for receiving an award or honor.

Intellectual/spiritual

Intellectual/spiritual abuse refers to such behaviors as punishing someone for having different intellectual interests or religious beliefs from others in the family, preventing them from attending worship services, ridiculing their opinions, and the like.

KEY TERMS

Cognitive restructuring—An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Flashback—The re-emergence of a traumatic memory as a vivid recollection of sounds, images, and sensations associated with the trauma. The person having the flashback typically feels as if he or she is reliving the event.

Incest—Unlawful sexual contact between persons who are biologically related. Many therapists, however, use the term to refer to inappropriate sexual contact between any members of a family, including stepparents and stepsiblings.

Stalking—The intentional pursuit or surveillance of another person, usually with the intent of forcing that person into a dating or marriage relationship. Stalking is now punishable as a crime in all 50 states.

Child abuse

Child abuse first attracted national attention in the United States in the 1950s, when a Denver pediatrician named C. Henry Kempe began publishing his findings regarding x-ray evidence of intentional injuries to small children. Kempe's research was followed by numerous investigations of other signs of child abuse and neglect, including **learning disorders**, malnutrition, failure to thrive, conduct disorders, emotional retardation, and sexually transmitted diseases in very young children.

Statistics from the mid-1990s indicate that although child abuse is found at all levels of income and educational achievement in the United States, children born into poor families are 12 times as likely to be abused as the children of middle-class or wealthy families, without regard to race or ethnic background. About 25% of children who are abused or neglected are younger than two years of age. Both sexes are equally affected. As of 2000, between 1,000 and 1,200 children die each year in the United States as the result of physical abuse. Of those who survive, 20% suffer permanent physical injury. Children who suffer from birth defects, developmental delays, or chronic illnesses have a higher risk of being abused by parents or other caregivers.

Abused adults

The women's movement of the 1970s led not only to greater recognition of domestic violence and other forms of abuse of adults, but also to research into the factors in the wider society that perpetuate abusive attitudes and behaviors. As of 2002, women are still more likely than men to be the targets of abuse in adult life.

Domestic violence

Domestic violence refers to the physical, emotional, and sexual abuse of a spouse or domestic partner. Early research into the problem of wife battering focused on middle-class couples, but it has since been recognized that spouse abuse occurs among wealthy professional couples as well. In addition, studies done in the late 1980s and 1990s indicate that domestic violence also occurs among gay and lesbian couples. It is estimated that four million women in the United States are involved in abusive marriages or relationships; moreover, most female murder victims are killed by their spouse or partner rather than by strangers.

Domestic violence illustrates the tendency of abusive people to attack anyone they perceive as vulnerable; most men who batter women also abuse their children; some battered women abuse their children; and abusive humans are frequently cruel to animals.

Elder abuse

Elder abuse has also become a subject of national concern in the last two decades. As older adults are living longer, many become dependent for years on adult caregivers, who may be either their own adult children or nursing home personnel. Care of the elderly can be extremely stressful, especially if the older adult is suffering from **dementia**. Elder abuse may include physical hitting or slapping; withholding their food or medications; tying them to their chair or bed; neglecting to bathe them or help them to the toilet; taking their personal possessions, including money or property; and restricting or cutting off their contacts with friends and relatives.

Abusive professional relationships

Adults can also be abused by sexually exploitative doctors, therapists, clergy, and other helping professionals. Although instances of this type of abuse were dismissed prior to the 1980s as consensual participation in sexual activity, most professionals now recognize that these cases actually reflect the practitioner's abuse of social and educational power. About 85% of sexual abuse cases in the professions involve male practitioners and female clients; another 12% involve male practitioners and male clients; and the remaining 3% involve female practitioners and either male or female clients. Ironically, many of these abusive relationships hurt women who sought professional help in order to deal with the effects of childhood abuse.

Stalking

Stalking, or the repeated pursuit or surveillance of another person by physical or electronic means, is now defined as a crime in all 50 states. Many cases of stalking are extensions of domestic violence, in that the stalker (usually a male) is attempting to track down a wife or girlfriend who left him. However, stalkers may also be casual acquaintances, workplace colleagues, or even total strangers. Stalking may include a number of abusive behaviors, including forced entry to the person's home, destruction of cars or other personal property, anonymous letters to the person's friends or employer, or repeated phone calls, letters, or e-mails. About 80% of stalking cases reported to police involve men stalking women.

Workplace bullying

Workplace bullying is, like stalking, increasingly recognized as interpersonal abuse. It should not be confused with sexual harassment or racial discrimination. Workplace bullying refers to verbal abuse of other workers, interfering with their work, withholding the equipment or other resources they need to do their job, or

This woman's husband had just beaten her. She took her children and left the house, and called police from a phone booth. She was afraid to return home. *(Nubar Alexanian/ CORBIS. Photo reproduced by permission.)*

invading their personal space, including touching them in a controlling manner. Half of all workplace bullies are women, and the majority (81%) are bosses or supervisors.

Causes of abuse

The causes of interpersonal abuse are complex and overlapping. Some of the most important factors are:

- Early learning experiences: This factor is sometimes described as the "life cycle" of abuse. Many abusive parents were themselves abused as children and have learned to see hurtful behavior as normal childrearing. At the other end of the life cycle, some adults who abuse their elderly parent are paying back the parent for abusing them in their early years.
- Ignorance of developmental timetables: Some parents have unrealistic expectations of children in terms of the appropriate age for toilet training, feeding themselves, and similar milestones, and attack their children for not meeting these expectations.
- Economic stress: Many caregivers cannot afford part-time day care for children or dependent elderly parents, which would relieve some of their emotional strain. Even middle-class families can be financially stressed if they find themselves responsible for the costs of caring for elderly parents before their own children are financially independent.
- Lack of social support or social resources: Caregivers who have the support of an extended family, religious group, or close friends and neighbors are less likely to lose their self-control under stress.
- Substance abuse: Alcohol and mood-altering drugs do not cause abuse directly, but they weaken or remove a person's inhibitions against violence toward others. In addition, the cost of a drug habit often gives a substance addict another reason for resenting the needs of the dependent person. A majority of workplace bullies are substance addicts.
- Mental disorders: Depression, **personality disorders**, dissociative disorders, and anxiety disorders can all affect parents' ability to care for their children appropriately. A small percentage of abusive parents or spouses are psychotic.
- Belief systems: Many men still think that they have a "right" to a relationship with a woman; and many people regard parents' rights over children as absolute.

- The role of bystanders: Research in the social sciences has shown that one factor that encourages abusers to continue their hurtful behavior is discovering that people who know about or suspect the abuse are reluctant to get involved. In most cases, bystanders are afraid of possible physical, social, or legal consequences for reporting abuse. The result, however, is that many abusers come to see themselves as invulnerable.

Aftereffects

Abuse affects all dimensions of human development and existence.

Physical and neurobiological

In addition to such direct results of trauma as broken bones or ruptured internal organs, physically abused children often display retarded physical growth and poor coordination. Malnutrition may slow the development of the **brain** as well as produce such dietary deficiency diseases as rickets. In both children and adults, repeated trauma produces changes in the neurochemistry of the brain that affect memory formation. Instead of memories being formed in the normal way, which allows them to be modified by later experiences and integrated into the person's ongoing life, traumatic memories are stored as chaotic fragments of emotion and sensation that are sealed off from ordinary consciousness. These traumatic memories may then erupt from time to time in the form of flashbacks.

Cognitive and emotional

Abused children develop distorted patterns of cognition (knowing) because they are stressed emotionally by abuse. As adults, they may suffer from cognitive distortions that make it hard for them to distinguish between normal occurrences and abnormal ones, or between important matters and relatively trivial ones. They often misinterpret other people's behavior and refuse to trust them. Emotional distortions include such patterns as being unable to handle strong feelings, or being unusually tolerant of behavior from others that most people would protest.

Social and educational

The cognitive and emotional aftereffects of abuse have a powerful impact on adult educational, social, and occupational functioning. Children who are abused are often in physical and emotional pain at school; they cannot concentrate on schoolwork, and consequently fall behind in their grades. They often find it hard to make or keep friends, and may be victimized by bullies or become bullies themselves. In adult life, abuse survivors are at risk of repeating childhood patterns through forming relationships with abusive spouses, employers, or professionals. Even though a survivor may consciously want to avoid re-abuse, the individual is often unconsciously attracted to people who remind him or her of the family of origin. Abused adults are also likely to fail to complete their education, or they accept employment that is significantly below their actual level of ability.

Treatment

Treatment of the aftereffects of abuse must be tailored to the needs of the specific individual, but usually involves a variety of long-term considerations that may include legal concerns, geographical relocation, and housing or employment as well as immediate medical or psychiatric care.

Medical and psychiatric

In addition to requiring immediate treatment for physical injuries, abused children and adults often need long-term **psychotherapy** in order to recover from specific mental disorders and to learn new ways of dealing with distorted thoughts and feelings. This approach to therapy is known as cognitive restructuring. Specific mental disorders that have been linked to childhood abuse include major depression, **bulimia nervosa**, **social phobia**, Munchausen syndrome by proxy, **generalized anxiety disorder**, **post-traumatic stress disorder**, **borderline personality disorder**, **dissociative amnesia**, and **dissociative identity disorder**. Abused adults may develop post-traumatic stress disorder, major depression, or substance abuse disorders. At present, researchers are focusing on genetic factors as a partial explanation of the fact that some people appear to react more intensely than others to being abused.

Legal considerations

Medical professionals and, increasingly, religious professionals as well, are required by law to report child abuse to law enforcement officials, usually a child protection agency. Physicians are granted immunity from lawsuits for making such reports.

Adults in abusive situations may encounter a variety of responses from law enforcement or the criminal justice system. In general, cases of spouse abuse, stalking, and sexual abuse by professionals are taken more seriously than they were two or three decades ago. Many communities now require police officers to arrest the aggressor in domestic violence situations, and a growing number of small towns as well as cities have shelters for family

members fleeing violent households. All major medical, educational, and legal professional societies, as well as mainstream religious bodies, have adopted strict codes of ethics, and have procedures in place for reporting cases of abuse by their members. Abuse in the workplace, however, is still a relatively new area of concern, and people affected by it have relatively few legal protections or resources as of 2002.

Prevention

Prevention of abuse requires long-term social changes in attitudes toward violence, gender roles, and the relationship of the family to other institutions. Research in the structure and function of the brain may help to develop more effective treatments for the aftereffects of abuse and possibly new appoaches to help break the intergenerational cycle of abuse. At present, preventive measures include protective removal of children or elders from abusive households; legal penalties for abusive spouses and professionals; and educating the public about the nature and causes of abuse.

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C. Henry Kempe National Center for the Prevention and Treatment of Child Abuse and Neglect. 1205 Oneida Street, Denver, CO 80220. (303) 321-3963.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

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Campaign Against Workplace Bullying. P. O. Box 1886, Benicia, CA 94510. <www.bullybusters.org>.

Rebecca J. Frey, Ph.D.

Acupressure *see* **Bodywork therapies**

Acupuncture

Definition

Acupuncture, one of the main forms of therapy in traditional Chinese medicine (TCM), has been practiced for at least 2,500 years. In acupuncture, certain points on the body associated with energy channels or meridians are stimulated by the insertion of fine needles. Unlike the hollow hypodermic needles used in mainstream medicine to give injections or draw blood, acupuncture needles are solid. The points can be needled between 15 and 90 degrees in range relative to the skin's surface, depending on treatment.

Acupuncture is thought to restore health by removing energy imbalances and blockages in the body. Practitioners of TCM believe that there is a vital force or energy called *qi* (pronounced "chee") that flows through the body, and between the skin surface and the internal organs, along channels or pathways called meridians. There are 12 major and 8 minor meridians. Qi regulates the spiritual, emotional, mental, and physical harmony of the body by keeping the forces of yin and yang in balance. Yang is a principle of heat, activity, brightness, outwardness, while yin represents coldness, passivity, darkness, interiority, etc. TCM does not try to eliminate either yin or yang, but to keep them in harmonious balance. Acupuncture may be used to raise or lower the level of yin or yang in a specific part of the body in order to restore the energy balance.

Acupuncture was virtually unknown in the United States prior to President Nixon's trip to China in 1972. A reporter for the *New York Times* named James Reston wrote a story for the newspaper about the doctors in Beijing who used acupuncture to relieve his pain following abdominal surgery. By 1993, Americans were making 12 million visits per year to acupuncturists, and spending $500 million annually on acupuncture treatments. By 1995, there were an estimated 10,000 certified acupuncturists practicing in the United States; as of 2000, there were 20,000. About a third of the credentialed acupuncturists in the United States as of 2002 are MDs.

Acupuncture's record of success has been sufficiently impressive to stimulate a number of research projects investigating its mechanisms as well as its efficacy. Research has been funded not only by the National Center for Complementary and Alternative Medicine (NCCAM), but also by the National Institute on Alcohol Abuse and Alcoholism (NIAAA), the National Institute of Dental Research, the National Institute of Neurological Disorders and Stroke (NINDS), and the National Institute on Drug Abuse. In 1997 a consensus panel of the National Institutes of Health (NIH) presented a landmark report in which it described acupuncture as

KEY TERMS

Cardiac tamponade—A condition in which blood leaking into the membrane surrounding the heart puts pressure on the heart muscle, preventing complete filling of the heart's chambers and normal heartbeat.

Electroacupuncture—A variation of acupuncture in which the practitioner stimulates the traditional acupuncture points electronically.

Endorphins—A group of peptide compounds released by the body in response to stress or traumatic injury. Endorphins react with opiate receptors in the brain to reduce or relieve pain.

Hyperemesis gravidarum—Uncontrollable nausea and vomiting associated with pregnancy. Acupuncture appears to be an effective treatment for women with this condition.

Meridians—In traditional Chinese medicine, a network of pathways or channels that convey qi (also sometimes spelled "ki"), or vital energy, through the body.

Moxibustion—A technique in traditional Chinese medicine that involves burning a *Moxa,* or cone of dried wormwood leaves, close to the skin to relieve pain. When used with acupuncture, the cone is placed on top of the needle at an acupuncture point and burned

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Opioids—Substances that reduce pain and may induce sleep. Some opioids are endogenous, which means that they are produced within the human body. Other opioids are produced by plants or formulated synthetically in the laboratory.

Pneumothorax—A condition in which air or gas is present in the chest cavity.

Qi—The Chinese term for energy, life force, or vital force.

Yin and yang—In traditional Chinese medicine and philosophy, a pair of opposing forces whose harmonious balance in the body is necessary to good health.

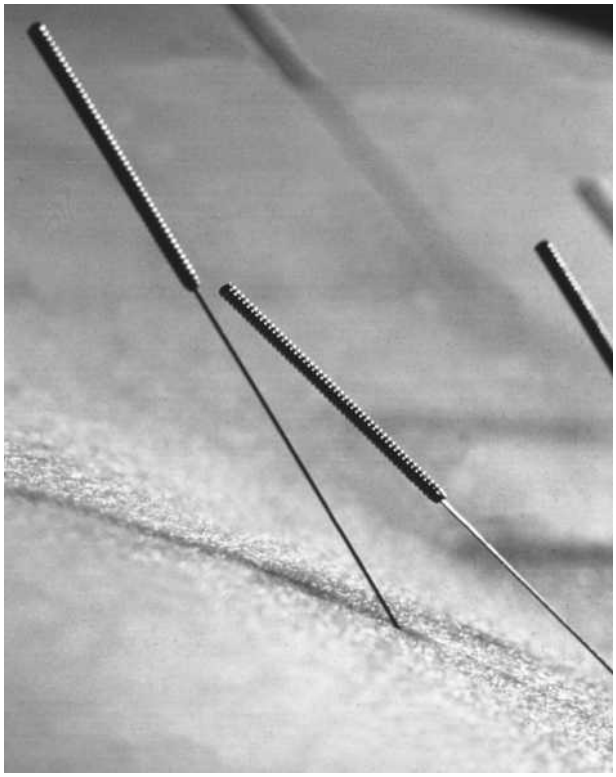
a sufficiently promising form of treatment to merit further study. In 2000, the British Medical Association (BMA) recommended that acupuncture should be made more readily available through the National Health Service (NHS), and that family doctors should be trained in some of its techniques.

Purpose

As already noted, the purpose of acupuncture in TCM is the rebalancing of opposing energy forces in different parts of the body. In Western terms, acupuncture is used most commonly as an adjunctive treatment for the relief of chronic or acute pain. In the United States, acupuncture is most widely used to treat pain associated with musculoskeletal disorders, but it has also been used in the treatment of substance abuse, and to relieve nausea and vomiting. A study done in 2001 showed that acupuncture was highly effective in stopping the intense vomiting associated with a condition in pregnant women known as hyperemesis gravidarum. In the past several years, acupuncture has been tried with a new patient population, namely children with chronic pain syndromes. One study of 30 young patients with disorders ranging from migraine headaches to endometriosis found that 70% felt that their symptoms had been relieved by acupuncture, and described themselves as "pleased" by the results of treatment. In addition to these disorders, acupuncture has been used in the United States to treat asthma, infertility, depression, anxiety, HIV infection, fibromyalgia, menstrual cramps, carpal tunnel syndrome, tennis elbow, pitcher's shoulder, chronic fatigue syndrome, and postoperative pain. It has even been used in veterinary medicine to treat chronic pain and prevent epileptic convulsions in animals. As of 2002, NCCAM is sponsoring research regarding the effectiveness of acupuncture in the rehabilitation of stroke patients.

The exact Western medicine mechanism by which acupuncture works is not known. Western researchers have suggested three basic explanations of acupuncture's efficacy in pain relief:

- Western studies have found evidence that the traditional acupuncture points conduct electromagnetic signals. Stimulating the acupuncture points causes these signals to be relayed to the **brain** at a higher than normal rate. These signals in turn cause the brain to release pain-relieving chemicals known as endorphins, and immune system cells to weak or injured parts of the body.
- Other studies have shown that acupuncture activates the release of opioids into the central nervous system. Opioids are also analgesic, or pain-relieving compounds.

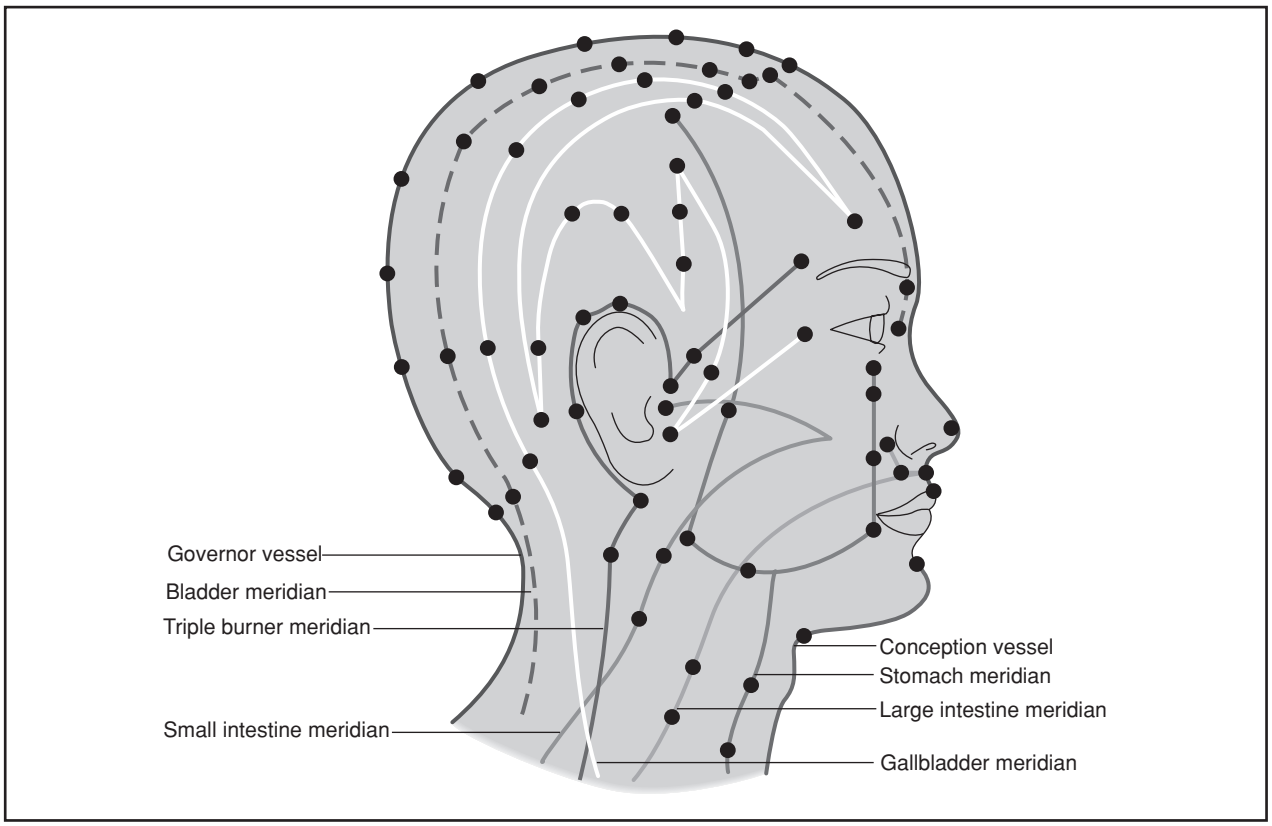


The purpose of acupuncture is to rebalance opposing energy forces in different parts of the body. In the United States, acupuncture is most widely used to treat pain associated with musculoskeletal disorders, but it has also been used in the treatment of substance abuse, and to relieve nausea and vomiting. *(Photo Researchers, Inc. Reproduced by permission.)*

- Acupuncture appears to alter the chemical balance of the brain itself by modifying the production and release of **neurotransmitters** and neurohormones. Acupuncture has been documented to affect certain involuntary body functions, including immune reactions, blood pressure, and body temperature.

In addition to its efficacy in relieving pain and other chronic conditions, acupuncture has gained in popularity because of several additional advantages:

- It lacks the side effects associated with many medications and surgical treatments in Western medicine.
- It is highly cost-effective; it may be used early in the course of a disease, potentially saving the patient the cost of hospitalizations, laboratory tests, and high-priced drugs.
- It can easily be combined with other forms of therapy, including psychotherapy.
- It is noninvasive.
- It carries relatively few risks.



Acupuncture sites and meridians on the face and neck. *(Illustration by Hans & Cassady, Inc.)*

Precautions

Although the risk of infection in acupuncture is minimal, patients should make sure that the acupuncturist uses sterile disposable needles. In the United States, the Food and Drug Administration (FDA) mandates the use of sterilized needles made from nontoxic materials. The needles must be clearly labeled as having their use restricted to qualified practitioners.

Patients should also inquire about the practitioner's credentials. Since acupuncture is now taught in over forty accredited medical schools and osteopathic colleges in the United States, patients who would prefer to be treated by an MD or an osteopath can obtain a list of licensed physicians who practice acupuncture in their area from the American Academy of Medical Acupuncture. With regard to nonphysician acupuncturists, 31 states have established training standards that acupuncturists must meet in order to be licensed in those states. In Great Britain, practitioners must qualify by passing a course offered by the British Acupuncture Accreditation Board.

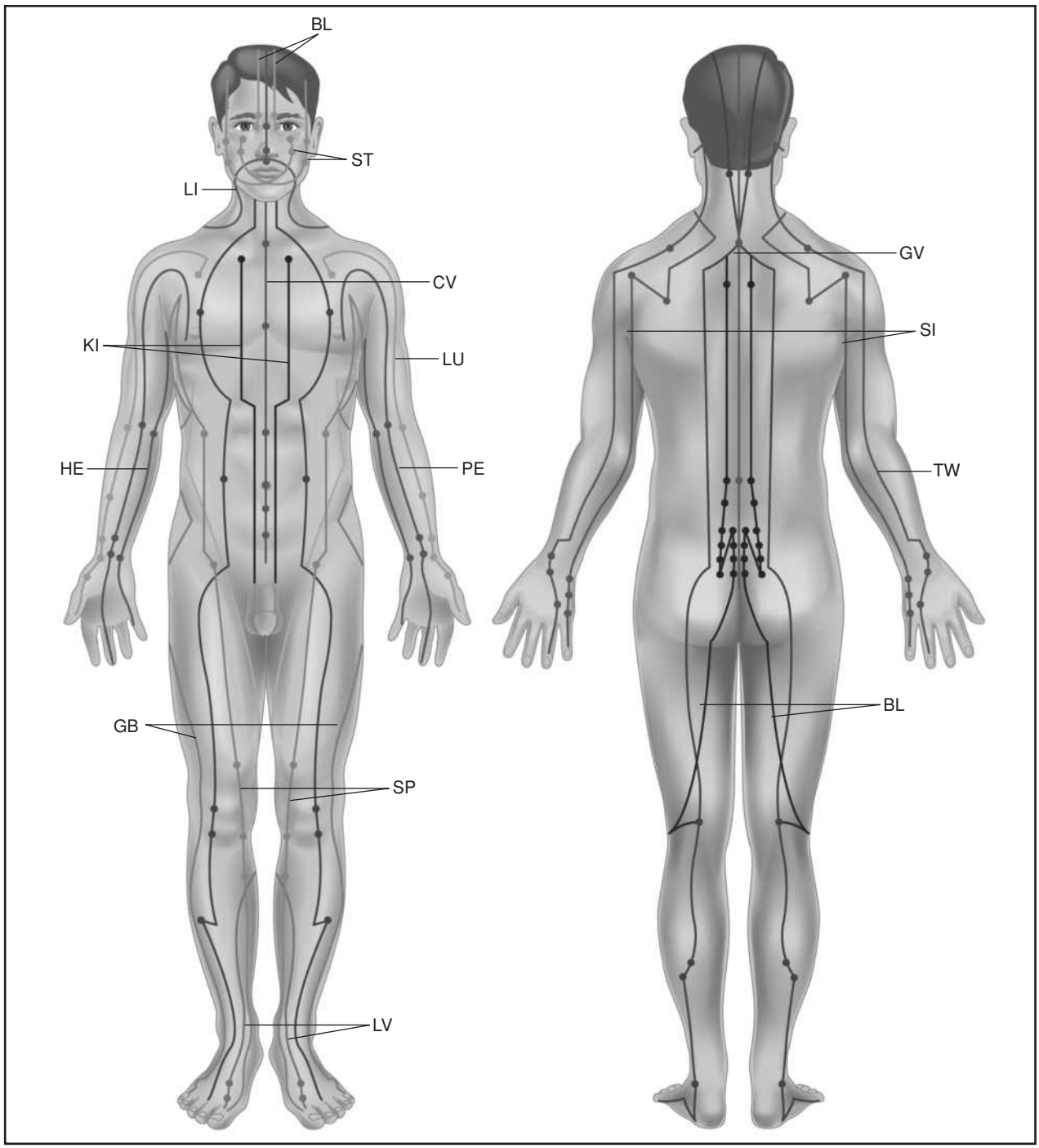
Patients seeking acupuncture treatment should provide the practitioner with the same information about their health conditions and other forms of treatment that they would give their primary care doctor. This information should include other alternative and complementary therapies, especially herbal remedies.

Acupuncture should not be used to treat severe traumatic injuries and other emergency conditions requiring immediate surgery. In addition, it does not appear to be useful in smoking cessation programs.

As is true with other forms of medical treatment, a minority of patients do not respond to acupuncture. The reasons for nonresponsiveness are not known at the present stage of research.

Description

In traditional Chinese medicine, acupuncture treatment begins with a thorough physical examination in which the practitioner evaluates the patient's skin color, vocal tone, and tongue color and coating. The practitioner then takes the patient's pulse at six locations and three depth levels on each wrist. These thirty-six pulse measurements will tell the practitioner where the qi in the patient's body might be blocked or unbalanced. After collecting this information, the acupuncturist will then identify the patterns of energy disturbance and the acupunc-



Traditional Chinese medicine teachings state that channels of energy flow throughout the body, and that disease is caused by too much or too little flow of energy along these channels. Points along the channels, called meridians, are manipulated in acupuncture. In the illustration, points are shown on the bladder (BL), conception vessel (CV), gall bladder (GB), governing vessel (GV), heart (HE), kidney (KI), large intestine (LI), liver (LV), lung (LU), pericardium (PE), small intestine (SI), spleen (SP), stomach (ST), and triple warmer (TW) meridians. *(Illustration by Electronic Illustrators Group.)*

ture points that should be stimulated to unblock the qi or restore harmony. Up to ten or twelve acupuncture needles will be inserted at strategic points along the relevant meridians. In traditional Chinese practice, the needles are twirled or rotated as they are inserted. Many patients feel nothing at all during this procedure, although others experience a prickling or mild aching sensation, and still others a feeling of warmth or heaviness.

The practitioner may combine acupuncture with moxibustion to increase the effectiveness of the treatment. Moxibustion is a technique in which the acupuncturist lights a small piece of wormwood, called a moxa, above the acupuncture point above the skin. When the patient begins to feel the warmth from the burning herb, it is removed. Cupping is another technique that is a method of stimulation of acupuncture points by applying suction through a metal, wood, or glass jar, and in which a partial vacuum has been created. Producing blood congestion at the site, the site is thus stimulated. The method is used for lower back pain, sprains, soft tissue injuries, as well as relieving fluid from the lungs in chronic bronchitis.

In addition to the traditional Chinese techniques of acupuncture, the following are also used in the United States:

- Electroacupuncture. In this form of acupuncture, the traditional acupuncture points are stimulated by an electronic device instead of a needle.
- Japanese meridian acupuncture. Japanese acupuncture uses thinner, smaller needles, and focuses on the meridians rather than on specific points along their course.
- Korean hand acupuncture. Traditional Korean medicine regards the hand as a "map" of the entire body, such that any part of the body can be treated by stimulating the corresponding point on the hand.
- Western medical acupuncture. Western physicians trained in this style of acupuncture insert needles into so-called trigger points in sore muscles, as well as into the traditional points used in Chinese medicine.
- Ear acupuncture. This technique regards the ear as having acupuncture points that correspond to other parts of the body. Ear acupuncture is often used to treat substance abuse and chronic pain syndromes.

A standard acupuncture treatment takes between 45 minutes to an hour and costs between $40 and $100, although initial appointments often cost more. Chronic conditions usually require 10 treatment sessions, but acute conditions or minor illnesses may require only one or two visits. Follow-up visits are often scheduled for patients with chronic pain. As of 2000, about 70%–80% of health insurers in the United States reimbursed patients for acupuncture treatments.

Preparation

Apart from a medical history and physical examination, no specific preparation is required for an acupuncture treatment. In addition to using sterile needles, licensed acupuncturists will wipe the skin over each acupuncture point with an antiseptic solution before inserting the needle.

Aftercare

No particular aftercare is required, as the needles should not draw blood when properly inserted. Many patients experience a feeling of relaxation or even a pleasant drowsiness after the treatment. Some patients report feeling energized.

Risks

Several American and British reports have concluded that the risks to the patient from an acupuncture treatment are minimal. Most complications from acupuncture fall into one of three categories: infections, most often from improperly sterilized needles; bruising or minor soft tissue injury; and injuries to muscle tissue. Serious side effects with sterilized needles are rare, although cases of pneumothorax and cardiac tamponade have been reported in the European literature. One American pediatrician estimates that the risk of serious injury from acupuncture performed by a licensed practitioner ranges between 1:10,000 and 1:100,000— or about the same degree of risk as a negative reaction to penicillin.

Normal results

Normal results from acupuncture are relief of pain and/or improvement of the condition being treated.

Abnormal results

Abnormal results from acupuncture include infection, a severe side effect, or worsening of the condition being treated.

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ORGANIZATIONS

American Academy of Medical Acupuncture/Medical Acupuncture Research Organization. 5820 Wilshire Boulevard, Suite 500, Los Angeles, CA 90036. (800) 521-2262 or (323) 937-5514. Fax: (323) 937-0959. <www.medicalacupuncture.org>.

American Association of Oriental Medicine. 433 Front Street, Catasaqua, PA 18032. (610) 266-1433. Fax: (610) 264-2768. <www.aaom.org>.

National Center for Complementary and Alternative Medicine (NCCAM) Clearinghouse. P.O. Box 7923, Gaithersburg, MD 20898. (888) 644-6226. TTY: (866) 464-3615. Fax: (866) 464-3616. <www.nccam.nih.gov>.

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Rebecca J. Frey, Ph.D.

Acute stress disorder

Definition

Acute stress disorder (ASD) is an anxiety disorder characterized by a cluster of dissociative and anxiety symptoms that occur within a month of a traumatic stressor. It is a relatively new diagnostic category and was added to the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* in 1994 to distinguish time-limited reactions to trauma from the farther-reaching and longer-lasting **post-traumatic stress disorder** (PTSD). Published by the American Psychiatric Association, the *DSM* contains diagnostic criteria, research findings, and treatment information for mental disorders. It is the primary reference for mental health professionals in the United States.

Description

ASD, like PTSD, begins with exposure to an extremely traumatic, horrifying, or terrifying event. Unlike PTSD, however, ASD emerges sooner and abates more quickly; it is also marked by more dissociative symptoms. If left untreated, however, ASD is likely to progress to PTSD. Because the two share many symptoms, some researchers and clinicians question the validity of maintaining separate diagnostic categories. Others explain them as two phases of an extended reaction to traumatic stress.

Causes and symptoms

Causes

The immediate cause of ASD is exposure to trauma—an extreme stressor involving a threat to life or the prospect of serious injury; witnessing an event that involves the death or serious injury of another person; or learning of the violent death or serious injury of a family member or close friend. The trauma's impact is determined by its cause, scope, and extent. Natural disasters (floods, earthquakes, hurricanes, etc.) or accidents (plane crashes, workplace explosions) are less traumatic than human acts of intentional cruelty or terrorism. Terrorist-inflicted trauma appears to produce particularly high rates of ASD and PTSD in survivors and bystanders.

Although most people define trauma in terms of events such as war, terrorist attacks, and other events that result in vast loss of life, the leading cause of stress-related mental disorders in the United States is motor vehicle accidents. Most Americans will be involved in a traffic accident at some point in their lives, and 25% of the population will be involved in accidents resulting in serious injuries. The National Comorbidity Survey of 1995 found that 9% of survivors of serious motor vehicle accidents developed ASD or PTSD.

Several factors influence a person's risk of developing ASD after trauma:

- Age—Older adults are less likely to develop ASD, possibly because they have had more experience coping with painful or stressful events.
- Previous exposure—People who were abused or experienced trauma as children are more likely to develop ASD (or PTSD) as adults, because these may produce long-lasting biochemical changes in the central nervous system.
- Biological vulnerability—Twin studies indicate that certain abnormalities in **brain** hormone levels and brain structure are inherited, and that these increase a person's susceptibility to ASD following exposure to trauma.
- Support networks—People who have a network of close friends and relatives are less likely to develop ASD.
- Perception and interpretation—People who feel inappropriate responsibility for the trauma, regard the event

KEY TERMS

Adjustment disorder—A disorder defined by the development of significant emotional or behavioral symptoms in response to a stressful event or series of events. Symptoms may include depressed mood, anxiety, and impairment of social and occupational functioning.

Depersonalization—A dissociative symptom in which the patient feels that his or her body is unreal, changing, or dissolving.

Derealization—A dissociative symptom in which the external environment is perceived as unreal or dreamlike.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Dissociative amnesia—A dissociative disorder characterized by loss of memory for a period or periods of time in the patient's life. May occur as a result of a traumatic event.

Exposure therapy—A form of cognitive-behavioral therapy in which patients suffering from phobias are exposed to their feared objects or situations while accompanied by the therapist. The length of exposure is gradually increased until the association between the feared situation and the patient's experienced panic symptoms is no longer present.

Flashback—The re-emergence of a traumatic memory as a vivid recollection of sounds, images, and sensations associated with the trauma. The person having the flashback typically feels as if he or she is reliving the event.

Hyperarousal—A symptom of traumatic stress characterized by abnormally intense reactions to stimuli. A heightened startle response is one sign of hyperarousal.

Hypervigilance—A state of abnormally intense wariness or watchfulness that is found in survivors of trauma or long-term abuse. Hypervigilance is sometimes described as "being on red alert all the time."

Personalization—The tendency to refer large-scale events or general patterns of events to the self in inappropriate ways. For example, a person who regards the loss of a friend or relative in an accident as punishment for having quarreled with them before the accident is said to be personalizing the event. Personalization increases a person's risk of developing ASD or PTSD after a traumatic event.

Psychic numbing—An inability to respond emotionally with normal intensity to people or situations; this affects positive emotions as well as fear or anger.

Sequela (plural, sequelae)—An abnormal condition resulting from a previous disease or disorder. An episode of depression is a common sequela of acute stress disorder.

Supportive—An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or educational approaches to treatment.

Survivor's guilt—A psychological reaction in trauma survivors that takes the form of guilt feelings for having survived or escaped a trauma without serious injury when others did not.

Therapeutic writing—A treatment technique in which patients are asked to set down in writing an account of the traumatic event and their emotional responses to it.

as punishment for personal wrongdoing, or have generally negative or pessimistic worldviews are more likely to develop ASD than those who do not personalize the trauma or are able to maintain a balanced view of life.

Symptoms

Acute stress disorder may be diagnosed in patients who (A) lived through or witnessed a traumatic event to which they (B) responded with intense fear, horror, or helplessness, and are (C) currently experiencing three or more of the following dissociative symptoms:

- psychic numbing
- being dazed or less aware of surroundings
- derealization
- depersonalization
- dissociative amnesia

Other symptoms that indicate ASD are:

- Reexperiencing the trauma in recurrent dreams, images, thoughts, illusions, or flashbacks; or intense distress when exposed to reminders of the trauma.

- A marked tendency to avoid people, places, objects, conversations, and other stimuli reminiscent of the trauma (many people who develop ASD after a traffic accident, for example, refuse to drive a car for a period of time).
- Hyperarousal or anxiety, including sleep problems, irritability, inability to concentrate, an unusually intense startle response, hypervigilance, and physical restlessness (pacing the floor, fidgeting, etc.).
- Significantly impaired social functions and/or the inability to do necessary tasks, including seeking help.
- Symptoms last for a minimum of two days and a maximum of four weeks, and occur within four weeks of the traumatic event.
- The symptoms are not caused by a substance (medication or drug of abuse) or by a general medical condition; do not meet the criteria of a **brief psychotic disorder**; and do not represent the worsening of a mental disorder that the person had before the traumatic event.

People with ASD may also show symptoms of depression including difficulty enjoying activities that they previously found pleasurable; difficulty in concentrating; and survivor's guilt at having survived an accident or escaping serious injury when others did not. The *DSM-IV-TR* (revised edition published in 2000) notes that people diagnosed with ASD "often perceive themselves to have greater responsibility for the consequences of the trauma than is warranted," and may feel that they will not live out their normal lifespans. Many symptoms of ASD are also found in patients with PTSD.

Demographics

Acute responses to traumatic stressors are far more widespread in the general United States population than was first thought in 1980, when PTSD was introduced as a diagnostic category in the *DSM-III*. The National Comorbidity Survey, a major epidemiological study conducted between 1990 and 1992, estimated that the lifetime prevalence among adult Americans is 7.8%, with women (10.4%) twice as likely as men (5%) to be diagnosed with trauma-related stress disorders at some point in their lives. These figures represent only a small proportion of adults who have experienced at least one traumatic event—60.7% of men and 51.2% of women respectively. More than 10% of the men and 6% of the women reported experiencing *four or more* types of trauma in their lives.

The prevalence of ASD by itself in the general United States population is not known. A few studies of people exposed to traumatic events found rates of ASD between 14% and 33%. Some groups are at greater risk of developing ASD or PTSD, including people living in depressed urban areas or on Native American reservations (23%) and victims of violent crimes (58%).

Diagnosis

ASD symptoms develop within a month after the traumatic event; it is still unknown, however, why some trauma survivors develop symptoms more rapidly than others. Delayed symptoms are often triggered by a situation that resembles the original trauma.

ASD is usually diagnosed by matching the patient's symptoms to the *DSM-IV-TR* criteria. The patient may also meet the criteria for a major depressive episode or **major depressive disorder**. A person who has been exposed to a traumatic stressor and has developed symptoms that do not meet the criteria for ASD may be diagnosed as having an **adjustment disorder**.

As of 2002, there are no diagnostic interviews or questionnaires in widespread use for diagnosing ASD, although screening instruments specific to the disorder are being developed. A group of Australian clinicians has developed a 19-item Acute Stress Disorder Scale, which appears to be effective in diagnosing ASD but frequently makes false-positive predictions of PTSD. The authors of the scale recommend that its use should be followed by a careful clinical evaluation.

Treatments

Therapy for ASD requires the use of several treatment modalities because the disorder affects systems of belief and meaning, interpersonal relationships, and occupational functioning as well as physical well-being.

Medications

Medications are usually limited to those necessary for treating individual symptoms. **Clonidine** is given for hyperarousal; **propranolol**, **clonazepam**, or **alprazolam** for anxiety and panic reactions; **fluoxetine** for avoidance symptoms; and **trazodone** or topiramate for **insomnia** and nightmares. Antidepressants may be prescribed if ASD progresses to PTSD. These medications may include selective serotonin reuptake inhibitors (SSRIs), monoamine oxidase inhibitors (MAOIs), or tricyclic antidepressants.

Psychotherapy

Cognitive-behavioral therapy, exposure therapy, therapeutic writing (journaling), and supportive therapy have been found effective in treating ASD. One variant of cognitive-behavioral therapy called psychoeducation-

Immediate crisis intervention after a tragedy or natural disaster may help victims express their feelings and fears and may help them to avoid developing acute stress disorder and post-traumatic stress disorder. Here, victims of a 1993 flood in Missouri receive counseling. *(Najlah Feanny-Hicks. Corbis/SABA. Photo reproduced by permission.)*

al therapy appears to be three to four times as effective as supportive therapy in preventing ASD from progressing to PTSD. This treatment combines cognitive restructuring of the traumatic event with exposure to disturbing images and techniques for anxiety management. In addition, it can help patients identify and reinforce positive aspects of their experience. For example, some people find new strengths or talents within themselves in times of crisis, or discover new spiritual resources.

Group and family therapies also appear to help patients with ASD reinforce effective strategies for coping with the trauma, and may reduce the risk of social isolation as a reaction to the trauma. They give patients opportunities to describe what happened and how they responded; they also let patients receive warmth and caring from their listeners, and help put memories of the event into a coherent narrative, allowing them to integrate the trauma into their overall lives.

Critical incident stress management (CISM) is a comprehensive crisis-intervention system in which a team of specially trained practitioners comes to the site of a traumatic event and provides several different forms of assistance, including one-on-one crisis support; crisis management briefing, which is a 45–75-minute **intervention** for groups of people affected by the traumatic event; and critical incident stress debriefing, which is a structured group discussion of the event. CISM appears to be particularly helpful in preventing burnout and ASD in emergency service personnel, rescue personnel, police, and other caregivers who are involved in treating survivors of a traumatic event.

Alternative and complementary treatments

Many mainstream practitioners recommend holistic or naturopathic approaches to recovery from ASD, including good nutrition with appropriate dietary supplements and regular exercise. **Yoga** and some forms of body work or massage therapy are helpful in treating the muscular soreness and stiffness that is often a side effect of the anxiety and insomnia related to ASD. Hydrotherapy is often helpful for post-traumatic muscular aches and cramps. A skilled naturopath may also recommend peppermint or other herbal preparations to calm the patient's digestive tract. In addition, prayer, **meditation**, or counseling with a spiritual advisor have been

found to be helpful in treating patients with ASD whose belief systems have been affected by the traumatic event.

Diagnosis and treatment of ASD in children

Very little is known about the prevalence of ASD or PTSD in children, and even less is known how effectively medications and **psychotherapy** treat these disorders in this age group. There are as yet no standardized screens or diagnostic interviews in widespread use for assessing either ASD or PTSD in children, although a Child Post-traumatic Stress Reaction Index was published in 1992. One preliminary study recommends the cautious use of low doses of **imipramine** for treating children with ASD, but notes that research in this area has barely begun.

Prognosis

Untreated ASD is highly likely to progress to PTSD in children as well as in adults. One team of Australian researchers found that 80% of persons diagnosed with ASD met criteria for PTSD six months later; 75% met criteria for PTSD two years after the traumatic event.

Clinicians in Norway have compiled a list of four "early response" variables that appear to be effective predictors of ASD's progressing to PTSD:

- the degree of the patient's sleep disturbance
- a strong startle reaction
- the degree of the patient's social withdrawal
- fear or phobia related to the site of the traumatic event

In addition to developing PTSD, people diagnosed with ASD are at increased risk of developing a major depressive disorder, particularly if their emotional responses to the trauma were marked by intense despair and hopelessness. Other sequelae may include neglect of personal needs for health or safety; and impulsive or needlessly risky behavior.

Prevention

Some forms of trauma, such as natural disasters and accidents, can never be completely eliminated from human life. Traumas caused by human intention would require major social changes to reduce their frequency and severity, but given the increasing prevalence of trauma-related stress disorders around the world, these long-term changes are worth the effort. In the short run, educating people—particularly those in the helping professions—about the signs of critical incident stress may prevent some cases of exposure to trauma from developing into ASD and progressing to full-blown PTSD.

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Anxiety Disorders Association of America. 11900 Parklawn Dr., Ste. 100, Rockville, MD 20852. (301) 231-9350.

International Society for Traumatic Stress Studies. 60 Revere Drive, Suite 500, Northbrook, IL 60062. Telephone: (847) 480-9028. Fax: (847) 480-9282. <www.istss.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

Society for Traumatic Stress Studies. 60 Revere Dr., Ste. 500, Northbrook, IL 60062. (708) 480-9080.

Rebecca J. Frey, Ph.D.

Adapin *see* **Doxepin**

Addiction

Definition

Most definitions refer to addiction as the compulsive need to use a habit-forming substance, or an irresistible urge to engage in a behavior. Two other important defining features of addiction are tolerance, the increasing need for more of the substance to obtain the same effect, and withdrawal, the unpleasant symptoms that arise when an addict is prevented from using the chosen substance.

Description

The term addiction has come to refer to a wide and complex range of behaviors. While addiction most commonly refers to compulsive use of substances, including alcohol, prescription and illegal drugs, cigarettes, and food, it is also used to describe excessive indulgence in activities such as work, exercise, shopping, sex, the Internet, and gambling.

Causes and symptoms

Causes

Some experts describe the range of behaviors designated as addictive in terms of five interrelated concepts: patterns, habits, compulsions, impulse control disorders, and physical addiction. There is ongoing controversy as to whether addictions constitute true physical disease in the same sense that diabetes and hypertension are considered physical diseases. Indeed, the most prevalent model of substance dependence today is the so-called disease model. This model, first introduced in the late 1940s by E. M. Jellinek, was adopted in 1956 by the American Medical Association. Since that time, the disease model of alcoholism and drug addiction has been well accepted throughout the world.

Other experts disagree with the analogy between substance abuse and physical disease. They believe that addictive behaviors can be better understood as problematic habits or behavior patterns that have been learned in accordance with the principles that guide all learning. To these experts, addictive behaviors are maladaptive habits and behavior patterns that can be "unlearned" and replaced with new, alternative, more healthful behaviors. According to learning theorists, one's past and present experiences, environment, family history, peer group influences, and individual beliefs and expectations, determine who will or will not become addicted to a substance or behavior.

Psychodynamic theorists believe that addicts suffer from an inability to soothe themselves or comfortably manage the emotions of day-to-day life. Feelings such as anxiety, depression, shame, discomfort in social situations, and anger are often believed to be causes of substance abuse. In this sense, many experts believe that addicts self-medicate, that is, use destructive substances to ease their painful emotions.

Disease model adherents believe that the compulsion to use is genetically and physiologically based and that, while the disease can be arrested, the disease is progressive and, if unchecked, fatal. Researchers have found the sons of alcoholics to be twice as prone to alcoholism as other people. Among pairs of identical twins, if one is alcoholic, there is a 60% chance that the other will be also. In spite of an apparent inherited tendency toward alcoholism, the fact that the majority of people with alcoholic parents do not become alcoholics themselves demonstrates the influence of psychosocial factors, including personality factors and a variety of environmental stressors, such as occupational or marital problems.

Symptoms

Both disease model and learning model adherents agree that initial positive consequences of substance abuse or addictive activities are what initially "hook," and then later keep, the addict addicted. Addicts describe feelings of euphoria when using their substance or engaging in their activity of choice. Many experts believe that these substances and activities affect **neurotransmitters** in the **brain**. Use causes an increase in endorphin levels, which is believed to be one of the chief causes of the "high" sensation experienced by addicts.

As the addict continues to use, his or her body adjusts to the substance and tolerance develops. Increasing amounts of the substance are needed to produce the same effect. Levels of substances that addicts routinely ingest would be lethal to a non-addict.

Over time, physical symptoms of dependence strengthen. Failure to use leads to withdrawal symptoms, which include flu-like aches and pains, digestive upset, and, in severe cases, **seizures**, and hallucinatory-like sensations, such as the feeling of bugs crawling on the skin. Damage to various organs of the body, including the brain and liver, can lead to serious and even fatal illness as well as mental symptoms such as **dementia**. Severe disruption of social and family relationships, and of the ability to maintain a steady job, are also symptoms of the addictive process.

Demographics

According to a 1999 national survey, about 14.8 million Americans used an illicit drug at least once in the

month prior to the survey, and the chances of receiving a **diagnosis** of substance abuse or dependence at some point in one's life is 16.7% for people over age 18. The lifetime chances of developing alcohol abuse or dependence is 13.8%; for nonalcohol substances, 6.2%. As of 1995, 6.1% of the population age 12 and older currently used illicit drugs. The most commonly used substances are alcohol and cigarettes, as well as marijuana, hashish, and cocaine. Unfortunately, substance abuse has been on the rise among children and adolescents since 1993.

According to findings of the National Institute of Drug Abuse, overall use of drugs in the United States has decreased by 50% during the past 20 years. However, drug use among adolescents has increased during the past 10 years.

Addiction is more common among men than women, and the use of drugs other than alcohol is skewed even further in that direction. Substance abuse is higher among the unemployed and the less educated. Most current illicit drug users are white. It is estimated that 9.6 million whites (75% of all users), 1.9 million African Americans (15% of users), and 1.0 million Hispanics (8% of users) were using illicit drugs in 1995.

Diagnosis

Substance abuse and dependence are among the psychological disorders categorized as major clinical syndromes (known as "Axis 1") in the American Psychiatric Association's ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*). Alcohol, classified as a depressant, is the most frequently abused psychoactive substance. Alcohol abuse and dependence affect more than 20 million Americans—about 13% of the adult population. An alcoholic has been defined as a person whose drinking impairs his or her life adjustment, affecting health, personal relationships, and/or work.

When blood alcohol level reaches 0.1%, a person is considered intoxicated. Judgment and other rational processes are impaired, as are motor coordination, speech, and vision. Alcohol abuse, according to the *DSM-IV-TR*, progresses through a series of stages from social drinking to chronic alcoholism. Danger signs that indicate the probable onset of a drinking problem include frequent desire to drink, increasing alcohol consumption, memory lapses (blackouts), and morning drinking. Other symptoms include attempts to hide alcohol from family and colleagues, and attempts to drink in secret. Among the most acute reactions to alcohol are four conditions referred to as alcoholic psychoses: alcohol idiosyncratic intoxication (an acute reaction in persons with an abnormally low tolerance for alcohol); alcohol withdrawal **delirium** (delirium tremens); **hallucinations**; and Korsakoff's **psychosis**, an irreversible brain disorder involving severe memory loss.

Other substance abuse disorders are diagnosed by looking for patterns of compulsive use, frequency of use, increasing tolerance, and withdrawal symptoms when the substance is unavailable or the individual tries to stop using.

Treatments

Pharmacologic

Addictions are notoriously difficult to treat. Physical addictions alter a person's brain chemistry in ways that make it difficult to be exposed to the addictive substance again without relapsing. Some medications, such as Antabuse (**disulfiram**), have shown limited effectiveness in treating alcohol addiction. Substitute medications, such as **methadone**, a drug that blocks the euphoric effect of opiates, have also shown mixed results. When an addicted individual is using a substance to self-medicate for depression, anxiety, and other uncomfortable emotions, prescription medications can be an effective treatment.

Psychological and psychosocial

It is a commonly held belief by many professionals that people with addictive disorders cannot be treated effectively by conventional outpatient **psychotherapy**. Substance abusers are often presumed to have severe personality problems and to be very resistant to treatment, to lack the motivation to change, or to be just too much trouble in an outpatient office setting. Unfortunately, these beliefs may create a self-fulfilling prophecy. Many of the negative behaviors and personality problems associated with chronic substance use disappear when use of the substance stops. While some substance abusers do, in fact, have other mental disorders, they represent only a minority of the addicted population.

Most treatment for addictive behaviors is provided not by practicing clinicians (psychiatrists, psychologists, and **social workers**), but rather by specialized addiction treatment programs and clinics. These programs rely upon confrontational tactics and re-education as their primary approaches, often employing former or recovering addicts to treat newly admitted addicts.

Some addicts are helped by the combination of individual, group, and family treatment. In family treatment (or **family therapy**), "enabling behaviors" can be addressed and changed. Enabling behaviors are the actions of family members who assist the addict in maintaining active addiction, including providing money, food, and shelter. Residential settings may be effective in initially assisting the addicted individual to stay away

from the many "cues," including people, places, and things, that formed the setting for their substance use.

During the past several decades, alternatives to the complete abstinence model (the generally accepted model in the United States) have arisen. Controlled use programs allow addicted individuals to reduce their use without committing to complete abstinence. This alternative is highly controversial. The generally accepted position is that only by complete abstinence can an addicted individual recover. The effectiveness of addiction treatment based on behavioral and other psychotherapeutic methods, however, is well documented. Among these are motivation-enhancing strategies, relapse-prevention strategies using cognitive-behavioral approaches, solution-oriented and other brief therapy technques, and harm-reduction approaches.

Self-help groups such as Alcoholics Anonymous and Narcotics Anonymous have also developed widespread popularity. The approach of one addict helping another to stay "clean," without professional **intervention**, has had tremendous acceptance in the United States and other countries.

Prognosis

Relapse and recidivism are, unfortunately, very common. Interestingly, a classic study shows that people addicted to different substances show very similar patterns of relapse. Whatever the addictive substances, data show that about two-thirds of all relapses occur within the first 90 days following treatment. Many consider recovery to be an ongoing, lifelong process. Because the use of addictive substances alters brain chemistry, cravings can persist for many years. For this reason, the predominating belief is that recovery is only possible by commitment to complete abstinence from all substance use.

Prevention

Prevention approaches are most effectively targeted at young teenagers between the ages of 11 and 13. It is during these years that most young people are likely to experiment with drugs and alcohol. Hence, reducing experimentation during this critical period holds promise for reducing the number of adults with addictive disease. Effective prevention programs focus on addressing the concerns of young people with regard to the effects of drugs. Training older adolescents to help younger adolescents resist peer pressure has shown considerable effectiveness in preventing experimentation.

See also Alcohol and related disorders; Amphetamines and related disorders; Anti-anxiety drugs and abuse-related disorders; Barbiturates; Caffeine and related disorders; Cannabis and related disorders; Denial; Disease concept of chemical dependency; Dual diagnosis; Internet addiction disorder; Nicotine and related disorders; Opioids and related disorders; Relapse and relapse prevention; Sedatives and related disorders; Self-help groups; Substance abuse and related disorders; Support groups; Wernicke-Korsakoff syndrome

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Adjustment disorder

Definition

An adjustment disorder is a type of mental disorder resulting from maladaptive, or unhealthy, responses to stressful or psychologically distressing life events. This low level of adaptation then leads to the development of emotional or behavioral symptoms.

Description

Often, a person experiences a stressful event as one that changes his or her world in some fundamental way.

An adjustment disorder represents significant difficulty in adjusting to the new reality.

The stressful events that precipitate an adjustment disorder vary widely. They may include the loss of a job; the end of a romantic relationship; a life transition such as a career change or retirement; or a serious accident or sickness. Some are acute "one-time" stressors, such as relocating to a new area, while others are chronic, such as caring for a child with **mental retardation**.

Psychiatrists have disagreed about the validity of the **diagnosis** of adjustment disorder, largely because of its lack of specificity. What qualifies as a stressful event, and what is an abnormal response to it? While adjustment disorders are more difficult to quantify than other mental disorders, many researchers consider the category a useful one for two reasons: 1) an adjustment disorder may be an early sign of a major mental disorder and allow for early treatment and **intervention**; 2) adjustment disorders are "situational" or "reactive"; they do not imply that the patient has an underlying **brain** disease.

KEY TERMS

Cognitive-behavioral therapy—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

Group therapy—Group interaction designed to provide support, correction through feedback, constructive criticism, and a forum for consultation and reference.

Interpersonal therapy—An approach that includes psychoeducation about the sick role, and emphasis on the present and improving interpersonal dynamics and relationships. Interpersonal therapy is effective in treating adjustment disorders related to physical illness.

Psychosocial—A term that refers to the emotional and social aspects of psychological disorders.

Solution-focused therapy—A type of therapy that involves concrete goals and an emphasis on future direction rather than past experiences.

Stressor—A stimulus or event that provokes a stress response in an organism. Stressors can be categorized as acute or chronic, and as external or internal to the organism.

Support group—A group whose primary purpose is the provision of empathy and emotional support for its members. Support groups are less formal and less goal-directed than group therapy.

Causes and symptoms

Causes

The ***Diagnostic and Statistical Manual of Mental Disorders***, which is the basic reference work consulted by mental health professionals, included an important change in its most recent version, the *DSM-IV-TR*, with regard to the criteria for adjustment disorder. In the previous edition, the identifiable stressor was described as being "psychosocial," a category that excludes physical illnesses and natural disasters. In the *DSM-IV-TR*, the word "psychosocial" was deleted in order to make the point that any stressful event can lead to an adjustment disorder. It is important to recognize, however, that while adjustment disorders are triggered by external stressors, the symptoms result from the person's interpretation of and adaptation to the stressful event or circumstances. Beliefs, perceptions, fears, and expectations influence the development of an adjustment disorder.

People with chronic physical illnesses appear to have an increased risk of developing adjustment disorders, particularly one with depressed mood. This connection has been demonstrated among cancer patients. The relationship between chronic pain (as is commonly experienced by cancer patients) and depressive symptoms is still being studied.

Symptoms

DSM-IV-TR states that the symptoms of an adjustment disorder must appear within three months of a stressor; and that they must meet at least one of the following criteria: 1) the distress is greater than what would be expected in response to that particular stressor; 2) the patient experiences significant impairment in social relationships or in occupational or academic settings. Moreover, the symptoms cannot represent bereavement, as normally experienced after the death of a loved one.

DSM-IV-TR specifies six subtypes of adjustment disorder, each with its own predominant symptoms:

- With depressed mood: The chief manifestations are feelings of sadness and depression, with a sense of accompanying hopelessness. The patient may be tearful and have uncontrollable bouts of crying.
- With anxiety: The patient is troubled by feelings of apprehension, nervousness, and worry. He or she may also feel jittery and unable to control his or her thoughts of doom. Children with this subtype may express fears of separation from parents or other significant people, and refuse to go to sleep alone or attend school.

- With mixed anxiety and depressed mood: The patient has a combination of symptoms from the previous two subtypes.
- With disturbance of conduct: This subtype involves such noticeable behavioral changes as shoplifting, truancy, reckless driving, aggressive outbursts, or sexual promiscuity. The patient disregards the rights of others or previously followed rules of conduct with little concern, guilt or remorse.
- With mixed disturbance of emotions and conduct: The patient exhibits sudden changes in behavior combined with feelings of depression or anxiety. He or she may feel or express guilt about the behavior, but then repeat it shortly thereafter.
- Unspecified: This subtype covers patients who are adjusting poorly to **stress** but who do not fit into the other categories. These patients may complain of physical illness and pull away from social contact.

Adjustment disorders may lead to **suicide** or suicidal thinking. They may also complicate the treatment of other diseases when, for instance, a sufferer loses interest in taking medication as prescribed or adhering to **diets** or exercise regimens.

An adjustment disorder can occur at any stage of life.

Demographics

Adjustment disorder appears to be fairly common in the American population; recent figures estimate that 5%–20% of adults seeking outpatient psychological treatment suffer from one of the subtypes of this disorder. As many as 70% of children in psychiatric inpatient settings may be diagnosed with an adjustment disorder. In a 1991 questionnaire that was sent to child psychiatrists, 55% admitted to giving children the diagnosis of an adjustment disorder to avoid the **stigma** associated with other disorders.

Women are diagnosed with adjustment disorder twice as often as men, while in clinical samples of children and adolescents, boys and girls were equally likely to be diagnosed with an adjustment disorder. Nolen-Hoeksema, a researcher who has conducted numerous studies on gender differences in depression, has argued that women over the age of 15 exhibit a more depressive temperament than men. She theorizes that women are more likely to respond to depression in ways that make the disorder worse and prolong it. Her findings appear to have some applicability to adjustment disorder with depressed mood.

There are no current studies of differences in the frequency of adjustment disorder in different racial or ethnic groups. There is, however, some potential for bias in diagnosis, particularly when the diagnostic criteria concern abnormal responses to stressors. *DSM-IV-TR* specifies that clinicians must take a patient's cultural background into account when evaluating his or her responses to stressors.

Diagnosis

Adjustment disorders are almost always diagnosed as the result of an interview with a **psychiatrist**. The psychiatrist will take a history, including identification of the stressor that has triggered the adjustment disorder, and evaluate the patient's responses to the stressor. The patient's primary physician may give him or her a thorough physical examination to rule out a previously undiagnosed medical illness.

The American Psychiatric Association considers adjustment disorder to be a residual category, meaning that the diagnosis is given only when an individual does not meet the criteria for a major mental disorder. For example, if a person fits the more stringent criteria for **major depressive disorder**, the diagnosis of adjustment disorder is not given. If the patient is diagnosed with an adjustment disorder but continues to have symptoms for more than six months after the stressor and its consequences have ceased, the diagnosis is changed to another mental disorder. The one exception to this time limit is situations in which the stressor itself is chronic or has enduring consequences. In that case, the adjustment disorder would be considered chronic and the diagnosis could stand beyond six months.

The diagnosis of adjustment disorder represents a particular challenge to clinicians because it has no checklist of specific and observable symptoms. The diagnosis is instead based on a broad range of emotional and behavioral symptoms that can vary widely in appearance and severity. The lack of a diagnostic checklist does in fact distinguish adjustment disorders from either **post-traumatic stress disorder** or **acute stress disorder**. All three require the presence of a stressor, but the latter two define the extreme stressor and specific patterns of symptoms. With adjustment disorder, the stressor may be any event that is significant to the patient, and the disorder may take very different forms in different patients.

Adjustment disorders must also be distinguished from **personality disorders**, which are caused by enduring personality traits that are inflexible and cause impairment. A personality disorder that has not yet surfaced may be made worse by a stressor and may mimic an adjustment disorder. A clinician must separate relatively stable traits in a patient's personality from passing disturbances. In some cases, however, the patient may be given both diagnoses. Again, it is important for psychia-

trists to be sensitive to the role of cultural factors in the presentation of the patient's symptoms.

If the stressor is a physical illness, diagnosis is further complicated. It is important to recognize the difference between an adjustment disorder and the direct physiological effects of a general medical condition (such as the usual temporary functional impairment associated with chemotherapy). This distinction can be clarified through communication with the patient's physician or by education about the medical condition and its treatment. For some individuals, however, both may occur and reinforce each other.

Treatments

There have been few research studies of significant scope to compare the efficacy of different treatments for adjustment disorder. The relative lack of outcome studies is partially due to the lack of specificity in the diagnosis itself. Because there is such variability in the types of stressors involved in adjustment disorders, it has proven difficult to design effective studies. As a result, there is no consensus regarding the most effective treatments for adjustment disorder.

Psychological and social interventions

There are, however, guidelines for effective treatment of people with adjustment disorders. Effective treatments include stress-reduction approaches; therapies that teach coping strategies for stressors that cannot be reduced or removed; and those that help patients build support networks of friends, family, and people in similar circumstances. **Psychodynamic psychotherapy** may be helpful in clarifying and interpreting the meaning of the stressor for a particular patient. For example, if the person is suffering from cancer, he or she may become more dependent on others, which may be threatening for people who place a high value on self-sufficiency. By exploring those feelings, the patient can then begin to recognize all that is not lost and regain a sense of self-worth.

Therapies that encourage the patient to express the fear, anxiety, rage, helplessness and hopelessness of dealing with the stressful situation may be helpful. These approaches include journaling, certain types of art therapy, and movement or dance therapy. **Support groups** and **group therapy** allow patients to gain perspective on the adversity and establish relationships with others who share their problem. Psychoeducation and medical crisis counseling can assist individuals and families facing stress caused by a medical illness.

Such types of brief therapy as **family therapy**, **cognitive-behavioral therapy**, solution-focused therapy, and **interpersonal therapy** have all met with some success in treating adjustment disorder.

Medications

Clinicians do not agree on the role of medications in treating adjustment disorder. Some argue that medication is not necessary for adjustment disorders because of their brief duration. In addition, they maintain that medications may be counterproductive by undercutting the patient's sense of responsibility and his or her motivation to find effective solutions. At the other end of the spectrum, other clinicians maintain that medication by itself is the best form of treatment, particularly for patients with medical conditions, those who are terminally ill, and those resistant to **psychotherapy**. Others advocate a middle ground of treatment that combines medication and psychotherapy.

Alternative therapies

Spiritual and religious counseling can be helpful, particularly for people coping with existential issues related to physical illness.

Some herbal remedies appear to be helpful to some patients with adjustment disorders. For adjustment disorder with anxiety, a randomized controlled trial found that patients receiving Euphytose (an herbal preparation containing a combination of plant extracts including Crataegus, Ballota, Passiflora, Valeriana, Cola, and Paullinia) showed significant improvement over patients taking a placebo.

Prognosis

Most adults who are diagnosed with adjustment disorder have a favorable prognosis. For most people, an adjustment disorder is temporary and will either resolve by itself or respond to treatment. For some, however, the stressor will remain chronic and the symptoms may worsen. Still other patients may develop a major depressive disorder even in the absence of an additional stressor.

Studies have been conducted to follow up on patients five years after their initial diagnosis. At that time, 71% of adults were completely well with no residual symptoms, while 21% had developed a major depressive disorder or alcoholism. For children aged 8–13, adjustment disorder did not predict future psychiatric disturbances. For adolescents, the prognosis is grimmer. After five years, 43% had developed a major psychiatric disorder, often of far greater severity. These disorders included **schizophrenia**, **schizoaffective disorder**, major depression, substance use disorders, or personality disorders. In contrast with adults, the adolescents' behav-

ioral symptoms and the type of adjustment disorder predicted future mental disorders.

Researchers have noted that once an adjustment disorder is diagnosed, psychotherapy, medication or both can prevent the development of a more serious mental disorder. Effective treatment is critical, as adjustment disorder is associated with an increased risk of suicide attempts, completed suicide, substance abuse, and various unexplained physical complaints. Patients with chronic stressors may require ongoing treatment for continued symptom management. While patients may not become symptom-free, treatment can halt the progression toward a more serious mental disorder by enhancing the patient's ability to cope.

Prevention

In many cases, there is little possibility of preventing the stressors that trigger adjustment disorders. One preventive strategy that is helpful to many patients, however, is learning to be proactive in managing ordinary life stress, and maximizing their problem-solving abilities when they are not in crisis.

See also Anxiety-reduction techniques; Bodywork therapies; Cognitive retraining techniques; Generalized anxiety disorder; Cognitive problem-solving skills training

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National Institute of Mental Health. 6001 Executive Boulevard, Rm. 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://nimh.nih.gov>.

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Advance directives

Definition

An advance directive is a written document in which people clearly specify how medical decisions affecting them are to be made if they are unable to make them, or to authorize a specific person to make such decisions for them. These documents are sometimes called "living wills." Psychiatric advance directives serve the same purpose as general medical advance directives, but are written by mental health consumers as a set of directions for others to follow, made in advance of an injury, psychiatric illness, or crisis.

Description

Many consumers of mental health services know which treatments work best for them and, over the past several years, their opinions have become increasingly valued by those providing services. However, when a mental health consumer becomes unable to make decisions or to give **informed consent** for treatments offered, others (including family, friends, judges, or care providers) make the decisions for the individual in crisis. In these kinds of crisis situations, advance directives may be beneficial for the person receiving care, because the advance directive is a legal document that may protect him or her from unwanted treatment.

Psychiatric advance directives usually fall into two categories: instruction directives and agent-driven directives.

Instruction directives

An instruction directive is a written document that specifies which treatments an individual does and does not want, in the case that that individual becomes unable to make decisions about his or her care. These documents may indicate the affected individual's preferences about many aspects of treatment, including:

- people who should be contacted at a time of psychiatric crisis
- activities that reduce (and heighten) anxiety for the individual
- effective alternatives to restraint or seclusion for the individual
- acceptable and unacceptable medications and dosages
- other interventions that might be considered during a time of crisis (such as **electroconvulsive therapy**)

Agent-driven directives

An agent-driven directive may also be called a durable power of attorney. This directive is a signed, dated, and witnessed document that authorizes a designated person (usually a family member or close friend) to act as an agent or proxy. This empowers the proxy to make medical decisions for a person when the person is deemed unable to make these decisions him/herself. Such a power of attorney frequently includes the person's stated preferences in regard to treatment. Several states do not allow any of the following people to act as a person's proxy:

- the person's physician, or other health care provider
- the staff of health care facilities that is providing the person's care
- guardians (often called conservators) of the person's financial affairs
- employees of federal agencies financially responsible for a person's care
- any person that serves as agent or proxy for 10 people or more. The person who is to act as the proxy should be familiar with the individual's expressed wishes about care, and should understand how to work within the mental health system.

These two distinct documents may, in some cases, be combined into one form.

Special concerns

In the United States, each state has laws about general medical advance directives and how those laws apply to psychiatric advance directives; a few states exclude psychiatric advance directives from their statutes. The specific form the advance directive should take, the language it should use, and the number of witnesses required to make the document legal and binding vary from state to state. In general, according to the National Mental Health Association, physicians and other health care professionals are expected to comply with the instructions of an advance directive, as long as those instructions are within the guidelines of accepted medical practice. Currently, however, few laws require providers to comply with an advance directive. It is recommended that people speak to their attorney or physician to ensure that their wishes are communicated in a form that is legally acceptable in their state.

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Advance Directive Training Project. Resource Center, Inc. Albany, NY. (518) 463-9242. <www.peer-resource.org>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850. Web site: <http://www.psych.org/>.

Judge David L. Bazelon Center for Mental Health Law. Washington, D.C. (202) 467-5730. <www.bazelon.org/advdir.html>.

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National Mental Health Association. (Produces a *Psychiatric Advance Directives Toolkit*). (800) 969-6642.

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Affect

Definition

Affect is a psychological term for an observable expression of emotion.

Description

A person's affect is the expression of emotion or feelings displayed to others through facial expressions, hand gestures, voice tone, and other emotional signs such as laughter or tears. Individual affect fluctuates according to emotional state. What is considered a normal range of affect, called the *broad affect*, varies from culture to culture, and even within a culture. Certain individuals may gesture prolifically while talking, and display dramatic facial expressions in reaction to social situations or other

stimuli. Others may show little outward response to social environments or interactions, expressing a narrow range of emotions to the outside world.

People with psychological disorders may display variations in their affect. A *restricted* or *constricted affect* describes a mild restriction in the range or intensity of display of feelings. As the reduction in display of emotion becomes more severe, the term *blunted affect* may be applied. The absence of any exhibition of emotions is described as *flat affect* where the voice is monotone, the face expressionless, and the body immobile. *Labile affect* describes emotional instability or dramatic mood swings. When the outward display of emotion is out of context for the situation, such as laughter while describing pain or sadness, the affect is termed "inappropriate."

See also Borderline personality disorder; Depression and depressive disorders; Major depressive disorder; Schizophrenia

Agoraphobia

Definition

Agoraphobia is an anxiety disorder characterized by intense fear related to being in situations from which escape might be difficult or embarrassing (i.e., being on a bus or train), or in which help might not be available in the event of a **panic attack** or panic symptoms. Panic is defined as extreme and unreasonable fear and anxiety.

According to the handbook used by mental health professionals to diagnose mental disorders, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision, also known as the *DSM-IV-TR*, patients with agoraphobia are typically afraid of such symptoms as feeling dizzy, having an attack of diarrhea, fainting, or "going crazy."

The word "agoraphobia" comes from two Greek words that mean "fear" and "marketplace." The anxiety associated with agoraphobia leads to avoidance of situations that involve being outside one's home alone, being in crowds, being on a bridge, or traveling by car or public transportation. Agoraphobia may intensify to the point that it interferes with a person's ability to take a job outside the home or to carry out such ordinary errands and activities as picking up groceries or going out to a movie.

Description

The close association in agoraphobia between fear of being outside one's home and fear of having panic symptoms is reflected in *DSM-IV-TR* classification of two separate disorders: **panic disorder** (PD) with agoraphobia, and agoraphobia without PD. PD is essentially characterized by sudden attacks of fear and panic. There may be no known reason for the occurrence of panic attacks; they are frequently triggered by fear-producing events or thoughts, such as driving, or being in an elevator. PD is believed due to an abnormal activation of the body's hormonal system, causing a sudden "fight-or-flight" response.

The chief distinction between PD with agoraphobia and agoraphobia without PD is that patients who are diagnosed with PD with agoraphobia meet all criteria for PD; in agoraphobia without PD, patients are afraid of panic-like symptoms in public places, rather than full-blown panic attacks.

People with agoraphobia appear to suffer from two distinct types of anxiety— panic, and the anticipatory anxiety related to fear of future panic attacks. Patients with agoraphobia are sometimes able to endure being in the situations they fear by "gritting their teeth," or by having a friend or relative accompany them.

In the United States' diagnostic system, the symptoms of agoraphobia can be similar to those of specific phobia and **social phobia**. In agoraphobia and specific phobia, the focus is fear itself; with social phobia, the person's focus is on how others are perceiving him/her. Patients diagnosed with agoraphobia tend to be more afraid of their own internal physical sensations and similar cues than of the reactions of others per se. In cases of specific phobia, the person fears very specific situations, whereas in agoraphobia, the person generally fears a variety of situations (being outside of the home alone, or traveling on public transportation including a bus, train, or automobile, for example). An example of a patient diagnosed with a specific phobia rather than agoraphobia would be the person whose fear is triggered only by being in a bus, rather than a car or taxi. The fear of the bus is more specific than the agoraphobic's fear of traveling on public transportation in general. The *DSM-IV-TR* remarks that the differential **diagnosis** of agoraphobia "can be difficult because all of these conditions are characterized by avoidance of specific situations."

Causes and symptoms

Causes

GENETIC. As of 2002, the causes of agoraphobia are complex and not completely understood. It has been known for some years that anxiety disorders tend to run in families. Recent research has confirmed earlier hypotheses that there is a genetic component to agoraphobia, and that it can be separated from susceptibility to PD. In 2001

KEY TERMS

Associationism—A theory about human learning that explains complex psychological phenomena in terms of coincidental relationships. For example, a person with agoraphobia who is afraid of riding in a car may have had a panic attack in a car on one occasion and has learned to associate cars with the physical symptoms of a panic attack.

Ayurvedic medicine—The traditional medical system of India. Ayurvedic treatments include diet, exercises, herbal treatments, meditation, massage, breathing techniques, and exposure to sunlight.

Behavioral inhibition—A set of behaviors that appear in early infancy that are displayed when the child is confronted with a new situation or unfamiliar people. These behaviors include moving around, crying, and general irritability, followed by withdrawing, and seeking comfort from a familiar person. These behaviors are associated with an increased risk of social phobia and panic disorder in later life. Behavioral inhibition in children appears to be linked to anxiety and mood disorders in their parents.

Cognitive restructuring—An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

Exposure therapy—A form of cognitive-behavioral therapy in which patients suffering from phobias are exposed to their feared objects or situations while accompanied by the therapist. The length of exposure is gradually increased until the association between the feared situation and the patient's experienced panic symptoms is no longer present.

Paresthesias—Abnormal sensations of tingling or "pins and needles." Paresthesias are a common panic-like symptom associated with agoraphobia.

Phobia—Irrational fear of places, things, or situations that lead to avoidance.

Simple phobia—An older term for specific phobia.

Specific phobia—A type of phobia in which the object or situation that arouses fear is clearly identifiable and limited. An older term for specific phobia is simple phobia.

a team of Yale geneticists reported the discovery of a genetic locus on human chomosome 3 that governs a person's risk of developing agoraphobia. PD was found to be associated with two loci: one on human chromosome 1 and the other on chromosome 11q. The researchers concluded that agoraphobia and PD are common; they are both inheritable anxiety disorders that share some, but not all, of their genetic loci for susceptibility.

INNATE TEMPERAMENT. A number of researchers have pointed to inborn temperament as a broad vulnerability factor in the development of anxiety and mood disorders. In other words, a person's natural disposition or temperament may become a factor in developing a number of mood or anxiety disorders. Some people seem more sensitive throughout their lives to events, but upbringing and life history are also important factors in determining who will develop these disorders. Children who manifest what is known as "behavioral inhibition" in early infancy are at increased risk for developing more than one anxiety disorder in adult life—particularly if the inhibition remains over time. (Behavioral inhibition refers to a group of behaviors that are displayed when the child is confronted with a new situation or unfamiliar people.) These behaviors include moving around, crying, and general irritability, followed by withdrawing, seeking comfort from a familiar person, and stopping what one is doing when one notices the new person or situation. Children of depressed or anxious parents are more likely to develop behavioral inhibition.

PHYSIOLOGICAL REACTIONS TO ILLNESS. Another factor in the development of PD and agoraphobia appears to be a history of respiratory disease. Some researchers have hypothesized that repeated episodes of respiratory disease would predispose a child to PD by making breathing difficult and lowering the threshold for feeling suffocated. It is also possible that respiratory diseases could generate fearful beliefs in the child's mind that would lead him or her to exaggerate the significance of respiratory symptoms.

LIFE EVENTS. About 42% of patients diagnosed with agoraphobia report histories of real or feared separation from their parents or other caretakers in childhood. This statistic has been interpreted to mean that agoraphobia in adults is the aftermath of unresolved childhood separation anxiety. The fact that many patients diagnosed with agoraphobia report that their first episode occurred after the death of a loved one, and the observation that other agoraphobics feel safe in going out as long as someone is

with them, have been taken as supportive evidence of the separation anxiety hypothesis.

LEARNED BEHAVIOR. There are also theories about human learning that explain agoraphobia. It is thought that a person's initial experience of panic-like symptoms in a specific situation— for example, being alone in a subway station— may lead the person to associate physical symptoms of panic with all subway stations. Avoiding all subway stations would then reduce the level of the person's discomfort. Unfortunately, the avoidance strengthens the phobia because the person is unlikely to have the opportunity to test whether subway stations actually cause uncomfortable physical sensations. One treatment modality—exposure therapy—is based on the premise that phobias can be "unlearned" by reversing the pattern of avoidance.

SOCIAL FACTORS RELATED TO GENDER. Gender role socialization has been suggested as an explanation for the fact that the majority of patients with agoraphobia are women. One form of this hypothesis maintains that some parents still teach girls to be fearful and timid about venturing out in public. Another version relates agoraphobia to the mother-daughter relationship, maintaining that mothers tend to give daughters mixed messages about becoming separate individuals. As a result, girls grow up with a more fragile sense of self, and may stay within the physical boundaries of their home because they lack a firm sense of their internal psychological boundaries.

Symptoms

The symptoms of an episode of agoraphobia may include any or all of the following:

- trembling
- breaking out in a sweat
- heart palpitations
- paresthesias (tingling or "pins and needles" sensations in the hands or feet)
- nausea
- fatigue
- rapid pulse or breathing rate
- a sense of impending doom

In most cases, the person with agoraphobia feels some relief from the symptoms after he or she has left the precipitating situation or returned home.

Demographics

In general, phobias are the most common mental disorders in the general United States population, affecting about 7% of adults, or 6.4 million Americans. Agoraphobia is one of the most common phobias, affecting between 2.7% and 5.8% of American adults. The onset of symptoms is most likely to occur between age 15 and age 35.The lifetime prevalence of agoraphobia is estimated at 5%–12%. Like most phobias, agoraphobia is two to four times more common in women than in men.

The incidence of agoraphobia appears to be similar across races and ethnic groups in the U.S.

Diagnosis

The differential diagnosis of agoraphobia is described differently in *DSM-IV-TR* and in *ICD-10,* the European diagnostic manual. The U.S. diagnostic manual specifies that agoraphobia must be defined in relation to PD, and that the diagnoses of **specific phobias** and social phobias are the next to consider. The *DSM-IV-TR* also specifies that the patient's symptoms must not be related to substance abuse; and if they are related to a general medical condition, they must have excessive symptoms usually associated with that condition. For example, a person with Crohn's disease has realistic concerns about an attack of diarrhea in a public place and should not be diagnosed with agoraphobia unless the fear of losing bowel control is clearly exaggerated. The *DSM-IV-TR* does not require a person to experience agoraphobia within a set number of circumstances in order to meet the diagnostic criteria.

In contrast, the European diagnostic manual primarily distinguishes between agoraphobia and delusional or obsessive disorders, and depressive episodes. In addition, *ICD-10* specifies that the patient's anxiety must be restricted to or occur primarily within two out of four specific situations: crowds; public places; traveling alone; or traveling away from home. The primary area of agreement between the American and European diagnostic manuals is that both specify avoidance of the feared situation as a diagnostic criterion.

Diagnosis of agoraphobia is usually made by a physician after careful exclusion of other mental disorders and physical conditions or diseases that might be related to the patient's fears. Head injury, pneumonia, and withdrawal from certain medications can produce some of the symptoms of a panic attack. In addition, the physician may ask about caffeine intake as a possible dietary factor. As of 2002, there are no laboratory tests or diagnostic **imaging studies** that can be used to diagnose agoraphobia.

Furthermore, there are no widely used diagnostic interviews or screening instruments specifically for agoraphobia. One self-report questionnaire, however, is under development by Dutch researchers who recently

reported on its validity. The test is called the Agoraphobic Self-Statements Questionnaire, or ASQ, and is intended to evaluate thinking processes in patients with agoraphobia, as distinct from their emotional responses.

Treatments

Treatment of agoraphobia usually consists of medication plus **cognitive-behavioral therapy** (CBT). The physician may also recommend an alternative form of treatment for the anxiety symptoms associated with agoraphobia. Some patients may be advised to cut down on or give up coffee or tea, as the caffeine in these beverages can be contribute to their panic symptoms.

Medications

Medications that have been used with patients diagnosed with agoraphobia include the benzodiazepine tranquilizers, the MAO inhibitors (MAOIs), tricyclic antidepressants (TCAs), and the selective serotonin uptake inhibitors, or SSRIs. In the past few years, the SSRIs have come to be regarded as the first-choice medication treatment because they have fewer side effects. The benzodiazepines have the disadvantage of increasing the symptoms of agoraphobia when they are withdrawn, as well as interfering with CBT. (Benzodiazepines can decrease mental sharpness, making it difficult for patients taking these medications to focus in therapy sessions.) The MAO inhibitors require patients to follow certain dietary guidelines. For example, they must exclude aged cheeses, red wine, and certain types of beans. TCAs may produce such side effects as blurred vision, constipation, dry mouth, and drowsiness.

Psychotherapy

CBT is regarded as the most effective psychotherapeutic treatment for agoraphobia. The specific CBT approach that seems to work best with agoraphobia is exposure therapy. Exposure therapy is based on undoing the association that the patient originally formed between the panic symptoms and the feared situation. By being repeatedly exposed to the feared location or situation, the patient gradually learns that he or she is not in danger, and the anxiety symptoms fade away. The therapist typically explains the procedure of exposure therapy to the patient and reassures him or her that the exposure can be stopped at any time that his or her limits of toleration have been reached. The patient is then exposed in the course of a number of treatment sessions to the feared situation, usually for a slightly longer period each time. A typical course of exposure therapy takes about 12 weeks.

On the other hand, one group of German researchers reported good results in treating patients with agoraphobia with individual high-density exposure therapy. The patients were exposed to their respective feared situations for an entire day for two–three weeks. One year later, the patients had maintained their improvement.

Exposure treatment for agoraphobia may be combined with cognitive restructuring. This form of cognitive behavioral therapy teaches patients to observe the thoughts that they have in the feared situation, such as, "I'll die if I have to go into that railroad station," and replace these thoughts with positive statements. In this example, the patient with agoraphobia might say to him- or herself, "I'll be just fine when I go in there to buy my ticket."

Although insight-oriented therapies have generally been considered relatively ineffective in treating agoraphobia, a recent trial of brief **psychodynamic psychotherapy** in patients with PD with agoraphobia indicates that this form of treatment may also be beneficial. Of the 21 patients who participated in the 24-session course of treatment (twice weekly for 12 weeks), 16 experienced remission of their agoraphobia. There were no relapses at six-month follow-up.

Alternative and complementary treatments

Patients diagnosed with agoraphobia have reported that alternative therapies, such as **hypnotherapy** and music therapy, were helpful in relieving symptoms of anxiety and panic. Ayurvedic medicine, **yoga**, religious practice, and guided imagery **meditation** have also been helpful.

Prognosis

The prognosis for untreated agoraphobia is considered poor by most European as well as most American physicians. The *DSM-IV-TR* remarks that little is known about the course of agoraphobia without PD, but that anecdotal evidence indicates that it may persist for years with patients becoming increasingly impaired. The *ICD-10* refers to agoraphobia as "the most incapacitating of the phobic disorders," to the point that some patients become completely housebound. With proper treatment, however, 90% of patients diagnosed with agoraphobia can recover and resume a normal life.

Prevention

As of this writing in 2002, the genetic factors that appear to be implicated in the development of agoraphobia cannot be prevented. On the other hand, recent recognition of the link between anxiety and mood disorders in parents and vulnerability to phobic disorders in their chil-

dren may help to identify children at risk and to develop appropriate preventive strategies for them.

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Anxiety Disorders Association of America. 11900 Parklawn Drive, Suite 100, Rockville, MD 20852-2624. (301) 231-9350. <www.adaa.org>.

Ayurvedic and Naturopathic Medical Clinic. 2115 112th Ave NE, Bellevue, WA 98004. (425) 453-8022. <www.ayurvedicscience.com>.

Freedom From Fear. 308 Seaview Avenue, Staten Island, NY 10305 (718) 351-1717. <www.freedomfromfear.com>.

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AIMS *see* **Abnormal Involuntary Movement Scale**

Akineton *see* **Biperiden**

Alcohol and related disorders

Definition

Alcoholism is defined as alcohol seeking and consumption behavior that is harmful. Long-term and uncontrollable harmful consumption can cause alcohol-related disorders that include: **antisocial personality disorder**, mood disorders (bipolar and major depression) and anxiety disorders.

Description

Alcoholism is the popular term for the disorder recognized by the American Psychiatric Association (APA) as alcohol dependence. The hallmarks of this disorder are **addiction** to alcohol, inability to stop drinking, and repeated interpersonal, school- or work-related problems

KEY TERMS

Antisocial personality disorder—Disorder characterized by behavior pattern of disregard for others' rights. People with this disorder often deceive and manipulate, or their behavior might include aggression to people or animals or property destruction, for example. This disorder has also been called sociopathy or psychopathy.

Blackout—A period of loss of consciousness or memory.

Delirium tremens—Serious alcohol withdrawal symptoms that must be treated in a hospital and that may include shaking, delirium, and hallucinations.

Detoxification—A process in which the body is allowed to free itself of a drug while the symptoms of withdrawal are treated. It is the primary step in any treatment program for drug or alcohol abuse.

Euphoria—A feeling or state of well-being or elation.

Intoxication—Condition of being drunk.

Relapse—A person experiences a relapse when he or she re-engages in a behavior that is harmful and that he or she was trying to change or eliminate. Relapse is a common occurrence after treatment for many disorders, including addictions and eating disorders.

Thiamin—A B-vitamin that is essential to normal metabolism and nerve function, and whose absorption is affected by alcoholism.

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

Wernicke's syndrome—A group of symptoms that appears in some people who are dependent on alcohol. Due to low levels of thiamin, the syndrome results in disordered eye movements, very poor balance and difficulty walking.

Wernicke-Korsakoff syndrome—Group of symptoms that appears in people who are dependent on alcohol. The syndrome is due to a thiamin deficiency, and severely affects one's memory, preventing new learning from taking place.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

that can be directly attributed to the use of alcohol. Alcoholism can have serious consequences, affecting an individual's health and personal life, as well as impacting society at large.

Alcohol dependence is a complex disorder that includes the social and interpersonal issues mentioned above, and also includes biological elements, as well. These elements are related to tolerance and withdrawal, cognitive (thinking) problems that include craving, and behavioral abnormalities including the impaired ability to stop drinking. Withdrawal is a term that refers to the symptoms that occur when a person dependent on a substance stops taking that substance for a period of time; withdrawal symptoms vary in type and severity depending on the substance, but alcohol withdrawal symptoms can include shaking, irritability, and nausea. Tolerance is a reduced response to the alcohol consumed and can be acute or chronic. Acute tolerance occurs during a single episode of drinking and is greater when blood alcohol concentration rises. Chronic tolerance occurs over the long term when there is greater resistance to the intoxicating effects of alcohol, and, as a result, the affected person has to drink more to achieve desired effect.

The APA also recognizes another alcohol use disorder called alcohol abuse. Alcohol abuse is similar to dependence in that the use of alcohol is impairing the affected person's ability to achieve goals and fulfill responsibilities, and his or her interpersonal relationships are affected by the alcohol abuse. However, unlike a person with dependence, a person diagnosed with alcohol abuse does not experience tolerance or, when not drinking, withdrawal symptoms. People who abuse alcohol can become dependent on the substance over time.

Alcohol-related disorders are groups of disorders that can result in persons who are long-term users of alcohol. These disorders can affect the person's metabolism, gastrointestinal tract, nervous system, bone marrow (the matter in bones that forms essential blood cells) and can cause endocrine (hormone) problems. Additionally, alcoholism can result in nutritional deficiencies. Some common alcohol-related medical disorders include vitamin deficiencies, alterations in sugar and fat levels in blood, hepatitis, fatty liver, cirrhosis, esophagitis (inflammation of the esophagus), gastritis (inflammation of the lining of the stomach), **dementia**, abnormal heart rates and rhythms, lowered platelets (cells important for forming a clot), leukopenia (decrease in the number of white

blood cells that are important for body defenses and immunity), and testicular atrophy (shrinking of the testicles). People with anxiety, depression, or **bipolar disorder** may consume alcohol for temporary relief from their symptoms. Others, such as people with antisocial personality disorder, may use alcohol as part of a **dual diagnosis** of criminality and substance dependence.

Causes and symptoms

Causes

The cause of alcoholism is related to behavioral, biological, and genetic factors.

Behaviorally, alcohol consumption is related to internal or external feedback. Internal feedback is the internal state a person experiences during and after alcohol consumption. External feedback is made up of the cues that other people send the person when he or she drinks. Internal states pertaining to alcohol can include shame or hangover. Alcohol-related external cues can include reprimands, criticism, or encouragement. People may drink to the point of dependence because of peer pressure, acceptance in a peer group, or because drinking is related to specific moods (easygoing, relaxed, calm, sociable) that are related to the formation of intimate relationships.

Biologically, repeated use of alcohol can impair the **brain** levels of a "pleasure" neurotransmitter called dopamine. **Neurotransmitters** are chemicals in the brain that pass impulses from one nerve cell to the next. When a person is dependent on alcohol, his or her brain areas that produce dopamine become depleted and the individual can no longer enjoy the pleasures of everyday life—his or her brain chemistry is rearranged to depend on alcohol for transient euphoria (state of happiness).

Genetic studies have isolated a gene that causes alcohol dependence and that is usually transmitted from affected fathers to sons. Other genetic studies have demonstrated that close relatives of an alcoholic are four times more likely to become alcoholics themselves. Furthermore, this risk holds true even for children who were adopted away from their biological families at birth and raised in a non-alcoholic adoptive family, with no knowledge of their biological family's difficulties with alcohol.

Symptoms

ALCOHOL DEPENDENCE. Individuals who are alcohol-dependent compulsively drink ethanol (the chemical name for alcohol) to the level of intoxication. Intoxication occurs at blood alcohol levels of 50 to 150 mg/dl and is characterized by euphoria at first, and then if blood concentrations of alcohol continue to rise, a person can become explosively combative. Neurologically, acute intoxication causes impaired thinking, incoordination, slow or irregular eye movements, and impaired vision. As the person repeatedly drinks, the body develops a reduced response to ethanol called tolerance.

People with chronic tolerance may apparently be sober (not intoxicated) even after consumption of alcohol that could cause death in non-drinkers. People with alcohol dependence may also develop alcoholic blackouts after large amounts of ethanol consumption. These blackouts are typically characterized by **amnesia** (loss of memory) lasting several hours without impaired consciousness. In other words, people experiencing blackouts appear to be conscious, but will not remember their actions during the blackouts after the intoxication has worn off.

People with alcohol dependence also develop alcohol withdrawal (a state of non-drinking) syndrome. The nervous system adapts to chronic ethanol exposure by increasing the activity of nerve cell mechanisms that counteract alcohol's depressant effects. Therefore, when drinking is abruptly reduced, the affected person develops disordered perceptions, **seizures**, tremor (often accompanied by irritability, nausea, and vomiting). Tremor of the hands called "morning shakes," usually occurs in the morning due to overnight abstinence. The most serious manifestation of alcohol withdrawal syndrome is delirium tremens, which occurs in approximately 5% of people dependent on alcohol. Delirium tremens consists of agitation, disorientation, **insomnia**, **hallucinations**, **delusions**, intense sweating, fever, and increased heart rate (tachycardia). This state is a medical emergency because it can be fatal, and affected persons must be immediately hospitalized and treated with medications that control vital physiological functions.

The APA publishes a manual for mental health professionals called the ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM*. This manual lists criteria that each disorder must meet for **diagnosis**. The criteria are symptoms that must be present so that the diagnosis can be made. Alcohol dependence can be diagnosed if three or more of the following symptoms are present:

- tolerance
- withdrawal
- denial of problem
- preoccupation with seeking alcohol
- drinking is the focal point of person's life (using takes up most of the person's time)
- continued use despite problems

ALCOHOL ABUSE. In order for a person to be diagnosed with alcohol abuse, one of the following four criteria must be met. Because of drinking, a person repeatedly:

- fails to live up to his or her most important responsibilities
- physically endangers him- or herself, or others (for example, by drinking when driving)
- gets into trouble with the law
- experiences difficulties in relationships or jobs

Demographics

The lifetime prevalence in the general population for alcoholism is between 9.4% and 14.1%. The disorder occurs twice as often in males than in females. Alcoholism and alcohol abuse affect 20% or more of hospitalized and ambulatory patients (those receiving care on an outpatient basis). Alcoholism can develop in all people of all races and socioeconomic classes. Approximately two-thirds of Americans older than 14 years drink alcohol. People who drink excessive amounts of alcohol account for about half of the total alcohol consumed, and account for almost all the socioeconomic and medical complications of alcoholism at an annual cost of $100 billion. Alcoholism ranks third in the United States as a preventable disease and accounts for 5% of the total deaths in the U.S. amounting to about 100,000 people dying annually.

Diagnosis

The diagnosis of alcoholism can either be based on medical and/or psychological conditions. With a long-term history of abusive drinking, medical conditions can result, and these could lead the physician to suspect a patient's alcoholism. These medical conditions may include organ complications such as: cirrhosis (liver), hepatitis (liver), pancreatitis (pancreas), peripheral neuropathy (nervous system) or cardiomyopathy (heart). Additionally, recurrent trauma, resulting in bone fractures, **fatigue**, depression, sexual dysfunction, fluctuating blood pressure, and **sleep disorders** may prompt the clinician to further assess for alcoholism.

Psychological diagnosis can be accomplished through a clinical interview and history (biopsychosocial assessment), and from a choice of many standardized alcohol use tests. The biopsychosocial assessment is an extensive interview conducted by the clinician. During the interview, the clinician will ask the patient about many areas of life, including childhood, education, and medical history. One very simple tool for beginning the diagnosis of alcoholism is called the CAGE questionnaire. It consists of four questions, with the first letters of each key word spelling out the word CAGE:

- Have you ever tried to *Cut* down on your drinking?
- Have you ever been *Annoyed* by anyone's comments about your drinking?
- Have you ever felt *Guilty* about your drinking?
- Do you ever need an *Eye-opener* (a morning drink of alcohol) to start the day?

Other, longer lists of questions exist to help determine the severity and effects of a person's alcohol use. Given the recent research pointing to a genetic basis for alcoholism, the doctor will also attempt to ascertain whether anyone else in the person's family has ever suffered from alcoholism.

Diagnosis is sometimes facilitated when family members call the attention of a physician to a loved one's difficulties with alcohol.

Treatments

Comprehensive treatment for alcohol dependence has two components: **detoxification** and rehabilitation.

Detoxification

The goal of detoxification is to rid the patient's body of the toxic effects of alcohol. Because the person's body has become accustomed to alcohol, the person will need to be supported as he or she goes through withdrawal. Withdrawal will be different for different patients, depending on the severity of the alcoholism, as measured by the quantity of alcohol ingested daily and the length of time the patient has been dependent on alcohol. Withdrawal symptoms can range from mild to life-threatening. Mild withdrawal symptoms include nausea, achiness, diarrhea, difficulty sleeping, sweatiness, anxiety, and trembling. This phase is usually over in about three to five days. More severe effects of withdrawal can include hallucinations (in which a patient sees, hears, or feels something that is not actually real), seizures, a strong craving for alcohol, confusion, fever, fast heart rate, high blood pressure, and delirium (a fluctuating level of consciousness). Patients at highest risk for delirium tremens are those with other medical problems, including malnutrition, liver disease, or Wernicke's syndrome. Delirium tremens usually begins about three to five days after the patient's last drink, progressing from the more mild symptoms to the more severe, and may last a number of days.

Patients going through mild withdrawal are simply monitored carefully to make sure that more severe symptoms do not develop. No medications are necessary,

however. Treatment of a patient suffering the more severe effects of withdrawal may require the use of sedative medications to relieve the discomfort of withdrawal and to avoid the potentially life-threatening complications of high blood pressure, fast heart rate, and seizures. Benzodiazepines are medications that ease tension by slowing down the central nervous system and may be helpful in those patients suffering from hallucinations. Because of the patient's nausea, fluids may need to be given through a vein (intravenously), along with some necessary sugars and salts. It is crucial that thiamin be included in the fluids, because thiamin is usually quite low in patients with alcohol dependence, and deficiency of thiamin is responsible for **Wernicke-Korsakoff syndrome**.

Rehabilitation

After cessation of drinking has been accomplished, the next steps involve helping the patient stay healthy and avoid relapsing. (Relapse occurs when a patient returns to old behaviors that he or she was trying to change.) This phase of treatment is referred to as rehabilitation. The best programs incorporate the family into the therapy, because the family has undoubtedly been severely affected by the patient's drinking. Some therapists believe that family members, in an effort to deal with their loved one's drinking problem, sometimes develop patterns of behavior that accidentally support or "enable" the patient's drinking. This situation is referred to as "co-dependence," and must be addressed in order to treat a person's alcoholism successfully.

PSYCHOLOGICAL THERAPIES. **Psychotherapy** helps affected individuals to anticipate, understand, recognize, and prevent relapse. Behavioral therapy approaches typically include community-centered **support groups**, meetings such as Alcoholics Anonymous (AA), **cognitive-behavioral therapy** (CBT), and Motivated Enhancement Therapy (MET). CBT focuses on teaching alcoholics recognition and coping skills for craving states and high-risk situations that precipitate or trigger relapsing behaviors. MET can motivate patients to use their personal resources to initiate changes in behavior. Many people recovering from substance dependence find peer-led support groups helpful in helping them avoid relapse.

MEDICATIONS. Two medications called **naltrexone** (Revia) and acamprosate can help decrease craving states in alcoholics. In combination with psychotherapy, these medications can help reduce relapse. Another medication called **disulfiram** (Antabuse) affects the metabolism of alcohol and causes unpleasant effects in patients who consume alcohol while taking the medication. Antabuse should only be taken by people who are committed to recovery and understand that they are to avoid all contact with alcohol or alcohol-containing products. People who have alcohol dependence along with other disorders, such as depression, can work with their physician to determine if medication might be a feasible treatment option for them.

ADDITIONAL TREATMENTS. Alternative treatments can be a helpful adjunct for the alcoholic patient, once the medical danger of withdrawal has passed. Because many alcoholics have very stressful lives (whether because of or leading to the alcoholism is sometimes a matter of debate), many of the treatments for alcoholism involve managing and relieving **stress**. These include massage, **meditation**, and **hypnotherapy**. The malnutrition of long-term alcohol use is addressed by nutrition-oriented practitioners and dietitians with careful attention to a healthy diet and the use of nutritional supplements such as vitamins A, B complex, and C, as well as certain fatty acids, amino acids, zinc, magnesium, and selenium. **Acupuncture** is believed to decrease both withdrawal symptoms and to help improve a patient's chances for continued recovery from alcoholism.

Prognosis

Most people who use alcohol start to drink during adolescence or early adulthood. Approximately 50% of male drinkers have alcohol-related problems such as fighting, blackouts, or legal problems during their early drinking years, usually late teens or early twenties. People who cannot control their drinking behaviors will tend to accumulate drinking-related problems and become dependent on alcohol. Approximately 30% to 60% of alcoholics maintain about one year of sobriety with psychotherapeutic interventions alone. About 20% of alcoholics can achieve long-term abstinence without any type of active treatment.

Prevention

Good prevention includes education and a knowledge of family (genetic) propensity. If alcohol dependence is present in a close family member, then relatives should know and be discouraged to drink alcohol-containing beverages. Education of older children and young teenagers concerning the negative effects and consequences of drinking alcohol may help to decrease or recognize problems before start or worsen.

See also Addiction; Denial; Diets; Disease concept of chemical dependency; Nutrition and mental health; Nutrition counseling; Relapse and relapse prevention; Self-help groups; Substance-induced anxiety disorder; Substance-induced psychotic disorder

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ORGANIZATIONS

National Institute on Alcohol Abuse and Alcoholism. 6000 Executive Boulevard-Willco Building, Bethesda, Maryland 20892-7003. <http://www.niaaa.nih.gov>.

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Alcoholics Anonymous *see* **Self-help groups**

Alcohol-induced persisting amnestic disorder *see* **Wernicke-Korsakoff syndrome**

Alprazolam

Definition

Alprazolam is a tranquilizer. It belongs to a group of drugs called benzodiazepines. In the United States alprazolam is sold under brand name Xanax.

Purpose

The United States Food and Drug Administration has approved alprazolam to treat anxiety, **panic disorder**, and anxiety associated with depression. Occasionally alprazolam is used to treat alcohol withdrawal, but it is not FDA-approved for this use, and is not normally the first drug tried in treating alcohol withdrawal symptoms.

Description

Alprazolam is classified as a benzodiazepine. Benzodiazepines are sedative-hypnotic drugs that help to relieve nervousness, tension, and other anxiety symptoms by slowing the central nervous system. To do this, they block the effects of a specific chemical involved in the transmission of nerve impulses in the **brain**, decreasing the excitement level of the nerve cells.

KEY TERMS

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

All benzodiazepines cause sedation, including drowsiness and reduced mental alertness. However, one benefit of alprazolam is that it causes somewhat less drowsiness than many other benzodiazepine drugs.

Alprazolam comes in 0.25-mg, 0.5-mg, 1-mg and 2-mg tablets, and 1-mg/ml solution.

Recommended dosage

The recommended initial adult dose for anxiety is 0.25–0.5 milligrams (mg) taken three times daily. This dosage may be increased every three to four days to a maximum total of 4 mg daily. Dosage for alcohol withdrawal usually totals from 2–2.5 mg daily given in several small doses throughout the day.

The starting dose for treating panic disorder is 0.5 mg three times daily. This dosage may be increased every three to four days until the total daily dosage ranges from 2–10 mg. The total amount should be divided in at least three even daily doses. Average doses for anxiety associated with depression range from 2.5–3 mg daily divided into even doses.

Precautions

Alprazolam should not be used by patients who are pregnant, have narrow angle glaucoma, take ketoconazole or itraconazole, or those who are allergic to this or any other benzodiazepine drug. The dose of alprazolam must be carefully regulated and individualized in the elderly (over age 60), people with liver or kidney disease, and those taking other medications used to treat mental disorders.

Because alprazolam is a nervous system and respiratory depressant, it should not be taken with other similar depressants, such as alcohol, other sedatives, sleeping

pills, or tranquilizers. People taking this drug should not drive, operate dangerous machinery, or engage in hazardous activities that require mental alertness at least until they see how the drug affects them.

Alprazolam should be used under close physician supervision in patients with history of substance abuse. Like other benzodiazepines, alprazolam can be habit-forming. Risk and severity of dependence appears greater in patients taking doses larger than 4 mg daily. However, smaller doses may cause dependence if alprazolam is taken longer than 12 weeks.

Suddenly discontinuing alprazolam after several weeks may cause uncomfortable symptoms of withdrawal. Withdrawal symptoms in people who have taken alprazolam three months or longer may include **seizures**, anxiety, nervousness, and headache. Patients should discuss with their doctor how to gradually discontinue alprazolam use to avoid such symptoms.

Side effects

The most common side effects of alprazolam include sedation, dizziness, drowsiness, **insomnia**, and nervousness. The intensity of these side effects usually declines gradually and subsides in about eight weeks. A drop in blood pressure and an increase in heart rate may also occur in people who are taking alprazolam.

Decreased sex drive, menstrual disorders, and both weight gain and weight loss has been associated with alprazolam use. People who experience the side effects of stomach upset, nausea, vomiting, and dry mouth should eat frequent, small meals and/or chew sugarless gum. Alprazolam has been associated with both diarrhea and constipation, as well as tremor, muscle cramps, vision disturbances, and rash.

Interactions

Alprazolam interacts with a long list of other medications. Anyone starting this drug should review the other medications they are taking with their physician and pharmacist for possible interactions. The most severe interactions occur with antifungal medications, such as ketoconazole, itraconazole, and fluconazole. These are associated with alprazolam toxicity (excessive sedation, **fatigue**, slurred speech, slowed reactions and other types of psychomotor impairment).

Estrogens (female hormones), erythromycin (an antibiotic), **fluoxetine** (Prozac, Sarafem), cimetidine (Tagamet), isoniazid, and **disulfiram** (Antabuse) can increase the effects of alprazolam. **Carbamazepine** can make alprazolam less effective. When alprazolam is combined with other sedative drugs (tranquilizers, sleeping pills) or alcohol, its depressants effects are more intense. These combinations should be avoided.

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Alzheimer's disease

Definition

Alzheimer's disease, or AD, is a progressive, incurable disease of the **brain** caused by the degeneration and eventual death of neurons (nerve cells) in several areas of the brain.

Description

Patients with AD first lose such mental functions as short-term memory and the ability to learn new things. In the later stages of AD they gradually lose control over their sense of orientation, their emotions, and other aspects of behavior. End-stage AD is characterized by loss of control of body functions, an increased likelihood of **seizures**, loss of the ability to eat or swallow, and eventual death from infection or malnutrition. Alzheimer's disease is the most common cause of **dementia** (loss of cognitive abilities) in the elderly; it is thought to be responsible for 50%–70% of cases of dementia in the United States.

Alzheimer's disease was first identified in 1906 by a German **psychiatrist** and neuroanatomist named Alois Alzheimer. He was studying slides prepared from the brain of a fifty-one-year-old woman, known as Frau D., who had died after several years of dementia with symptoms that did not fit the definition of any brain disorder known at the time. Alzheimer was the first to describe the plaques and neurofibrillary tangles that are now used to identify AD at autopsy. Plaques are clumps or clusters of dead or dying nerve cells and other cellular debris found in the brains of patients with Alzheimer's disease. Neurofibrillary tangles are the accumulations of twisted protein fragments found inside the nerve cells in the brains of Alzheimer's patients. Because dementia had

been associated with elderly people and Frau D. had been middle-aged, AD was first known as presenile dementia and was thought to be a very rare disorder. It was not until the early 1950s that researchers at St. Elizabeth's Hospital in Washington, DC, came to recognize that AD is the single most common cause of dementia in adults.

Alzheimer's disease is now considered a very serious public health problem because of the growing numbers of people who are affected by it, the increasing length of their lives, and the direct and indirect costs of their care. It is estimated that four million people in the United States had AD as of 2000, with 360,000–400,000 new cases identified every year. One person in ten over the age of 65 has AD, and nearly 50% of those over 85 have the disease. Unless a cure or preventive treatment is discovered, 14 million Americans will have Alzheimer's by 2050. Very few people are wealthy enough to cover the cost of caring for an Alzheimer's patient in the seven–10 years that typically extend between the beginning of the person's dependency and death. The average lifetime cost of caring for one patient with AD is estimated at $174,000. The costs of laboratory tests, physicians' visits, medications, nursing services, home care, and adult day care come to $114.4 billion per year in the United States alone. This sum is greater than the combined annual budgets of six Federal departments (Commerce, Education, Justice, Labor, Energy, and Interior).

The problem is expected to be complicated in future years by the fact that the so-called "baby boomer" generation is better nourished and better educated than the generation now at risk for AD. When the baby boomers are old enough to be at risk for late-onset Alzheimer's, they are expected to live longer than the average Alzheimer's patient does in 2002. Public health researchers who are making future projections about the impact of AD in the mid-twenty-first century point out that a treatment that would delay the onset of the disease would reduce the overall prevalence of AD. One study estimates that a therapy that would delay the onset of Alzheimer's by only one year would save the United States $9 billion by 2007. The second approach, that of discovering a treatment for people who already have Alzheimer's, would alter the proportion of mild cases to those considered moderate or severe. The researchers conclude by stating: "None of our models predicts less than a threefold rise in the total number of persons with Alzheimer's disease between 2000 and 2050."

Types of Alzheimer's disease

As of 2002, some researchers think that Alzheimer's may be more accurately described as a group or family of diseases rather than a single disease. Moreover, more recent research is helping to differentiate Alzheimer's disease from other less common causes of dementia. In particular, some cases of dementia that were formerly thought to have been related to AD are now known to have been caused by Pick's disease or Lewy body dementia. Pick's disease is a rare type of dementia that affects certain areas of the brain and is characterized by a progressive loss of social skills, language, and memory. Lewy body dementia is a type of dementia in which the brain has characteristic Lewy bodies—areas of injury found on damaged nerve cells in certain parts of the brain.

Physicians now recognize three different forms of Alzheimer's disease.

EARLY-ONSET AD. Early-onset AD is a rare form of Alzheimer's found in fewer than 10% of AD patients. This group of patients, however, develops more of the brain abnormalities associated with AD than patients with the late-onset form. In addition, patients with early-onset Alzheimer's are more likely to develop myoclonus (a condition in which a muscle or group of muscles has sudden spasms or twitching).

LATE-ONSET AD. Late-onset AD is the most common form of the disease; its symptoms usually begin to appear after age 65. Late-onset Alzheimer's, which may or may not be affected by genetic variables, is also called sporadic Alzheimer's disease because it does not necessarily run in families.

FAMILIAL ALZHEIMER'S DISEASE (FAD). Familial Alzheimer's disease, or FAD, is a rare form that is entirely inherited. FAD accounts for fewer than 5% of all cases of AD. It has a very early onset, often in the patient's 40s, and there is a clear family history of the disease.

Stages

Health care professionals use the term "insidious" to describe Alzheimer's, which means that it is very gradual in onset. Many times people recognize the first symptoms of the disorder in a friend or family member only in hindsight. In addition, the present generation of people old enough to be at risk for Alzheimer's is the first generation in history to know what the **diagnosis** means; there are therefore very powerful emotional reasons for attributing the early signs of AD to normal aging, job **stress**, adjusting to retirement, and other less troubling factors. The insidious onset of Alzheimer's is a characteristic, however, that allows doctors to distinguish it from other causes of dementia, including **vascular dementia**.

EARLY-STAGE ALZHEIMER'S. Early-stage Alzheimer's may begin almost imperceptibly. The first symptoms usually include short-term memory loss, temporary

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Agitation—Excessive restlessness or emotional disturbance that is often associated with anxiety or psychosis. Agitation may be associated with middle-stage Alzheimer's disease.

Agnosia—Loss of the ability to recognize familiar people, places, and objects.

Amygdala—An almond-shaped brain structure in the limbic system that is activated in stressful situations to trigger the emotion of fear. It is thought that the emotional overreactions in Alzheimer's patients are related to the destruction of neurons in the amygdala.

Amyloid—A waxy translucent substance composed mostly of protein, that forms plaques (abnormal deposits) in the brain during the progression of Alzheimer's disease.

Aphasia—Loss of language abilities.

Apolipoprotein E—A protein that transports cholesterol through the body. One form of this protein, apoE4, is associated with a 60% risk of late-onset AD.

Apraxia—Inability to perform purposeful movements that is not caused by paralysis or loss of feeling.

Beta amyloid protein—A starchy substance that builds up in the brains of people with AD to form the plaques that are characteristic of the disease. Beta amyloid is formed when amyloid precursor protein, or APP, is not broken down properly by the body.

Bleomycin hydrolase—An enzyme involved in the body's processing of amyloid precursor protein. If the gene that governs production of BH mutates, the APP accumulates, producing the plaques in the brains of patients with AD.

Brain stem—The part of the brain that is continuous with the spinal cord and controls most basic life functions. It is the last part of the brain that is destroyed by Alzheimer's disease.

Cholinesterase inhibitors—A group of medications given to slow the progression of Alzheimer's disease.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness. It can be distinguished from dementia by its relatively sudden onset and variation in the severity of the symptoms.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Down syndrome—A genetic disorder characterized by an extra chromosome 21 (trisomy 21), mental retardation, and susceptibility to early-onset Alzheimer's disease.

Gingko—A shade tree native to China with fan-shaped leaves and fleshy seeds with edible kernels. Gingko extract is being studied as a possible complementary or adjunctive treatment for Alzheimer's.

Hallucination—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not real-

episodes of spatial disorientation, groping for words while one is speaking, minor problems with arithmetic, and small errors of judgment. For example, the person may light a stove burner under a saucepan before noticing that he has forgotten to put the food or water in the pan first, or he may have difficulty balancing a checkbook as quickly as he used to. At this stage in the disease, however, the patient can usually keep up with most activities of daily life. Although some persons at this point can still operate a motor vehicle safely, it is advisable to consult a physician about possible impairment behind the wheel. Many patients with early-stage AD voluntarily give up their driver's licenses for their own safety and that of other drivers on the roads.

MIDDLE-STAGE ALZHEIMER'S. In the middle stage, which typically begins two to three years after onset, the person begins to lose awareness of his or her cognitive deficits. Memory lapses are more frequent and the person begins to have more severe problems with language. Unlike early-stage AD, the problems caused by loss of cognitive functioning are impossible to ignore. The middle stage of AD is the point at which the behavioral and psychiatric symptoms that are so stressful to caregivers

KEY TERMS CONTINUED

ly present. Hallucinations can also affect the senses of smell, touch, and taste.

Hippocampus—A part of the brain that is involved in memory formation and learning. The hippocampus is shaped like a curved ridge and belongs to an organ system called the limbic system.

Insidious—Proceeding gradually and inconspicuously but with serious effect.

Lewy bodies—Areas of injury found on damaged nerve cells in certain parts of the brain associated with dementia. Lewy body dementia was first ecognized in the 1980s and is now distinguished from Alzheimer's disease.

Mild cognitive impairment (MCI)—A transitional phase of memory loss in older people that precedes dementia or Alzheimer's disease.

Multi-infarct dementia—Dementia caused by damage to brain tissue resulting from a series of blood clots or clogs in the blood vessels. It is also called vascular dementia.

Myoclonus—An abrupt spasm or twitching in a muscle or group of muscles. It is more common in early-onset AD than in late-onset Alzheimer's.

Neurofibrillary tangles—Accumulations of twisted protein fragments found inside the nerve cells in the brains of Alzheimer's patients.

Neurotransmitters—Chemicals that carry nerve impulses from one nerve cell to another. Alzheimer's disease causes a drop in the production of several important neurotransmitters.

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness. It may be related in some way to Lewy body dementia.

Pick's disease—A rare type of primary dementia that affects the frontal lobes of the brain. It is characterized by a progressive loss of social skills, language, and memory, leading to personality changes and sometimes loss of moral judgment.

Plaques—Clumps or clusters of beta amyloid fragments, dead or dying nerve cells, and other cellular debris, found in the brains of patients with Alzheimer's disease.

Polygenic—A trait or disorder that is determined by a group of genes acting together. Most human characteristics, including height, weight, and general body build, are polygenic. Schizophrenia and late-onset Alzheimer's disease are considered polygenic disorders.

Post mortem—After death. The definitive diagnosis of Alzheimer's disease can be made only after the patient's death.

Presenile dementia—An older name for Alzheimer's disease.

Pseudodementia—A term for a depression with symptoms resembling those of dementia. The term "dementia of depression" is now preferred.

Systolic—Referring to the rhythmic contraction of the heart (systole), when the blood in the chambers of the heart is forced out. Systolic blood pressure is blood pressure measured during this phase.

Tau protein—A protein that is involved in maintaining the internal structure of nerve cells. The tau protein is damaged in Alzheimer's disease and ends up forming the neurofibrillary tangles.

often begin— the agitation, wandering, temper outbursts, depression, and disorientation. The patient is at high risk for falls and similar accidents. In addition, the patient becomes increasingly unable to understand simple instructions or to follow a conversation, and begins to lose his or her basic sense of personal identity.

END-STAGE ALZHEIMER'S. End-stage Alzheimer's is marked by the loss of the ability to walk and eventually even to sit up. Patients may be able to use a wheelchair for awhile but eventually become completely bedridden. They lose bladder and bowel control. When the disease begins to affect the patient's brain stem, the basic processes of digestion, respiration, and excretion shut down. Patients usually stop eating at this point and sleep most of the time. The hands and feet begin to feel cold, the breathing becomes shallow, and the patient is generally unresponsive to caregivers. Eventually the patient's breathing simply stops.

Causes and symptoms

Causes

Evidence has accumulated that Alzheimer's disease is multifactorial— that is, it is caused by a combination of several genetic and environmental factors.

GENETIC. Early-onset AD is caused by a defect in one of three genes known as APP, presenilin-1, and presenilin-2, found on human chromosomes 21, 14, and 1, respectively. Early-onset AD is also associated with Down syndrome, in that people with trisomy 21 (three forms of human chromosome 21 instead of a pair) often develop this form of Alzheimer's. The brains of people with Down syndrome age prematurely, so that those who develop early-onset AD are often only in their late 40s or early 50s when the symptoms of the disease first appear.

Genetic research indicates that late-onset Alzheimer's disease is a polygenic disorder; that is, its development is influenced by more than one gene. It has been known since 1993 that a specific form of a gene for apolipoprotein E (apoE4) on human chromosome 19 is a genetic risk factor for late-onset AD. People who have the apoE4 gene from one parent have a 50% chance of developing AD; a 90% chance if they inherited the gene from both parents. They are also likely to develop AD earlier in life. One of the remaining puzzles about this particular gene, however, is that it is not a consistent marker for AD. In other words, some people who have the apoE4 gene do not develop Alzheimer's, and some who do not have the gene do develop the disorder. In 1998 another gene on chromosome 12 that controls the production of bleomycin hydrolase (BH, an enzyme involved in the body's processing of amyloid precursor protein) was identified as a second genetic risk factor that acts independently of the APOE gene. In December 2000, three separate research studies reported that a gene on chromosome 10 that may affect the processing of a particular protein is also involved in the development of late-onset AD.

Familial Alzheimer's disease appears to be related to abnormal genes on human chromosomes 21 and 14.

NEUROBIOLOGICAL. Investigators since Alois Alzheimer's time have studied the abnormalities found at autopsy in the brains of patients with AD. One abnormality is plaques, or clumps, of a starchy protein called beta amyloid. Beta amyloid is formed when a substance called amyloid precursor protein, or APP, fails to be metabolized properly in the body. APP is a substance found in many parts of the body, but its precise function is not yet known. Following the formation of beta amyloid, pieces of it then stick to one another and gradually build up into plaques. The other abnormal finding is neurofibrillary tangles, which are twisted threads formed from parts of the dying nerve cell called the tau protein, which was discovered in 1986. If the tau protein is damaged by the addition of molecules of phosphorus, a process called hyperphosphorylation, it forms filaments that twist around each other to form the neurofibrillary tangles. As the plaques and tangles accumulate in the brain, they cause the nerve cells to wither and eventually die. As the nerve cells die, the affected parts of the brain start to shrink in size. It is not known as of 2002, however, whether the plaques and tangles are causes of AD or results of it. The relationship between the plaques and the tangles is another question that has not yet been answered. Although the plaques usually appear in brain tissue before the tangles, it is not clear that they cause the tangles. There are other brain disorders, such as Pick's disease, in which tangles appear in the brain cells without plaques.

Another nervous system abnormality in AD is the lowered level of **neurotransmitters** produced by the cells in the brain. Neurotransmitters are chemicals that carry nerve impulses across the small gaps (synapses) between nerve cells. The neurotransmitters whose production is affected by Alzheimer's include serotonin, norepinephrine, and acetylcholine. Many of the behavioral and psychiatric problems associated with AD are thought to result from the inadequate supply of these neurotransmitters.

ENVIRONMENTAL. Researchers have been studying the possibility that certain chemicals or other toxins in the environment may have a role in causing or triggering AD. The environmental factors that have been considered include aluminum, zinc, toxins in contaminated food, and viruses. Although there is little evidence as of 2002 that AD is caused by a virus or other infectious agent, the possibility cannot be completely excluded.

Other hypotheses about the causes of Alzheimer's include damage caused by oxidation, estrogen deficiency, and inflammation. All of these possibilities are presently under investigation.

RISK FACTORS. A number of factors have been identified that increase a person's risk of developing Alzheimer's:

- Age. The risk of developing AD rises after age 65, and rises sharply after age 75. While 1% of the population has AD at age 65, almost 50% of those over 85 have it.
- Sex. Women are more likely to develop AD than men. As of 2002, however, it is not known whether women are more susceptible to the disorder, or more likely to develop it because they live longer than men, on average.
- Family history of AD.
- Having Down syndrome.
- History of head injury.

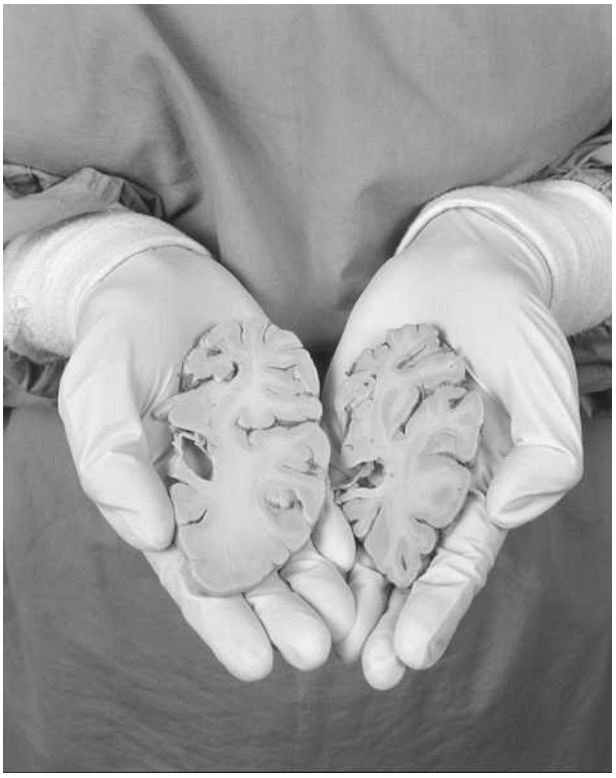
- Substances in the environment. Higher-than-average amounts of aluminum have been found in the brains of patients with Alzheimer's. Some researchers in the late 1990s thought that exposure to aluminum might be a risk factor for the disorder. It now appears that the levels of aluminum in the brains of patients are a result rather than a cause of AD.
- Low occupational attainment and education level. Studies have found a clear correlation between employment in jobs that are not mentally challenging and an increased risk of AD. In addition, taking less rather than more challenging jobs as one grows older is associated with a higher risk of AD.
- High systolic blood pressure.
- High blood cholesterol levels. When both high systolic blood pressure and high cholesterol are present, the risk of developing AD increases by a factor of 3.5.
- Mild cognitive impairment (MCI). Mild cognitive impairment is a transitional decline in cognitive functioning that precedes the onset of AD. MCI is characterized primarily by memory loss while other cognitive functions remain intact. People with MCI are at higher risk for AD than people who do not develop the condition; 12% of people with mild cognitive impairment develop Alzheimer's disease each year, compared with 1–2% per year of people without MCI. After four years, 40% of people diagnosed with mild cognitive impairment have clear symptoms of Alzheimer's disease.

Symptoms

The symptoms of Alzheimer's can be grouped into three categories: cognitive deficits, or losses of brain function related to memory and learning; behavioral and psychiatric symptoms of dementia, or BPSD; and problems with activities of daily life, or ADLs.

COGNITIVE DEFICITS. There are four major symptoms of loss of cognitive capacities in Alzheimer's:

- **Amnesia**. Amnesia refers to memory impairment; however, loss of short-term memory also means that the patient loses his or her sense of time as well.
- Aphasia. Aphasia refers to loss of language function. The person may not remember the names of objects and may use words like "thing" or "it" instead; they may echo what other people say or repeat a word or phrase over and over. On occasion the person may lose the ability to speak except for curse words.
- Apraxia. Apraxia is the loss of the ability to perform voluntary movements in the absence of paralysis. A person with apraxia, for example, may have trouble putting on a hospital gown or brushing his or her teeth.



A brain segment affected by Alzheimer's disease on the right compared with a healthy brain segment (left). The brain affected by disease appears shrunken, and the fissures are noticeably larger. *(Simon Fraser/ MRC Unit, Newcastle General Hospital/ Science Photo Library. Photo Researchers, Inc. Reproduced by permission.)*

- Agnosia. Agnosia comes from a Latin word that means "to not know", and refers to inability to recognize familiar places and people. Patients with agnosia may even fail to recognize their own face in a mirror.

NEUROPSYCHIATRIC SYMPTOMS. Symptoms associated with BPSD include:

- Depression. Depression associated with AD is thought to result from the lowered production of the neurotransmitter serotonin. Depression in AD can be treated with medication, usually with one of the selective serotonin reuptake inhibitors, or SSRIs.
- **Delusions**. A delusion is a false belief that a person maintains even when presented with contrary evidence. For example, patients with AD may say that a person is stealing their things when they cannot remember where they have put them. Suspicions of other people caused by delusions can sometimes be treated with medication.
- Wandering. This behavior may result from becoming disoriented and getting lost, but sometimes people with AD wander for no apparent reason. The Alzheimer's Association in Chicago has a Safe Return Hotline (list-

ed under "Resources," below) that can be contacted for information about registering a patient with AD. If the registered patient should wander from home, the Safe Return Hotline can help identify him or her and return them to their family or nursing home.

- **Hallucinations**. Like delusions, hallucinations in AD patients are thought to be related to the deterioration of the patient's brain tissue. In a hallucination, the patient has a sensory experience that is real to him or her but not to other people. Hallucinations can affect any of the senses, but most are either visual or auditory. For example, a patient with AD may say that he or she sees little Martians in the corner of the room, or that he or she hears the voice of a long-dead parent calling to them. Hallucinations are sometimes caused by medications that the patient may be taking.
- Aggression. Aggression refers to hitting, shoving, pushing, or threatening behavior.
- Agitation. Agitation refers to emotionally excited behavior (screaming, shouting, cursing, pacing, fidgeting, etc.) that is disruptive or unsafe. Agitation may result from the changes in the patient's brain tissue, or it may be a symptom of depression associated with Alzheimer's disease.

For most of the twentieth century, studies of Alzheimer's patients focused on the cognitive symptoms of the disorder. It was not until the 1980s and 1990s that researchers began to look more closely at the behavioral and psychiatric symptoms of AD. Such methods of standardized assessment of these symptoms as the neuropsychiatric inventory are very recent developments.

PROBLEMS WITH ACTIVITIES OF DAILY LIVING (ADLS). Needing help with ADLs, or personal care activities that are part of everyday living, is among the earliest symptoms of Alzheimer's. The functions that are often affected include:

- eating, including simple cooking and washing dishes
- bathing, showering, or shaving
- grooming and dressing in clothing appropriate to the weather and activity
- toileting
- other aspects of personal hygiene (brushing teeth or cleaning dentures, washing hair, etc.)
- shopping for groceries and other necessary items

Health care professionals usually assess the ADLs of a patient diagnosed with Alzheimer's in order to determine what type of care is needed.

Demographics

Some demographic statistics in the developed countries have already been cited in the context of risk factors for AD and public health concerns related to the disorder.

Relatively little is known about the demographics of AD and other forms of dementia in the developing countries. Alzheimer's Disease International, which is based in London, supports a group of researchers called the 10/66 Dementia Research Group. The 10/66 group is trying to correct the global imbalance of AD research; as of 2001, fewer than 10% of all population-based research studies of AD and related forms of dementia has been directed toward the 66% of people with these disorders who live outside the developed countries. Of the estimated 18 million people in the world with dementia, 12 million live in China, India, Latin America, and other developing nations.

Diagnosis

As of 2002, the diagnosis of AD is essentially a process of exclusion. The only definitive diagnosis of Alzheimer's is made post mortem (after death), by performing an autopsy and examining the patient's brain tissue. There are no present tests that can be done on a living person to make the diagnosis of AD more than probable.

Diagnostic evaluation of AD

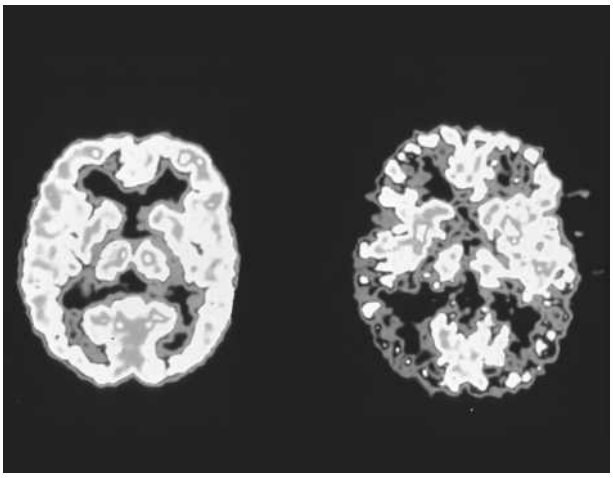
At present, the diagnostic process includes the following components:

- Clinical interview. In the absence of laboratory tests or **imaging studies** that can provide definite diagnoses, the physician must rely on his or her clinical judgment. In evaluating the patient, the doctor will assess signs of cognitive impairment other than short-term memory loss. In most cases, the doctor will ask a family member or close friend of the patient about the suddenness of symptom onset and the length of time that the patient seems to have been impaired.
- Physical examination. The patient will be given a complete physical and have blood and urine samples taken to rule out vitamin deficiencies, head trauma, tertiary syphilis, thyroid disorders, and other possible causes of dementia. The doctor will also review all the medications that the patient is taking (including alternative remedies) in order to exclude reversible dementia caused by drug interactions.
- Neurological examination. In early AD, the neurological findings are usually normal. If the patient appears to have had a **stroke**, he or she will be referred for a more thorough assessment by a neurologist.

- Tests of cognitive function. The patient will be given the mini-mental status examination (MMSE) and such other tests of cognitive function as the clock test or verbal fluency tests. The MMSE is a screening test and should not be used by itself to make the diagnosis of AD. In addition, the MMSE is not very sensitive in detecting cognitive impairment in people who previously functioned at a high level and were well educated. It is possible for a well-educated person to score a perfect 30 on the MMSE and still suffer cognitive impairment. The clock test is a test in which patients are asked to draw a clock face. Sometimes, patients will also be asked to include a specific time on the clock, such as 3:20. Patients with Alzheimer's often draw the face of the clock with numbers out of order, or all of the hour markers in a portion of the clock face instead of evenly spaced around the face, and often have difficulty adding the clock hands.
- Neuropsychiatric evaluation. A neuropsychiatric examination may be given to determine the pattern of the patient's cognitive impairment and probe his or her level of functioning more deeply. The patient may be asked to write a sample check, to describe how they answer the phone, to interpret sample traffic signs, and to look at a shopping list and pick out the items on the list from a display.
- Diagnostic imaging. Imaging studies are useful in detecting such causes of dementia as a previously undiagnosed brain tumor or abnormal brain structure. Scans can show doctors that certain areas of the brain have lost tissue (as happens in AD), and can strengthen a physician's suspicion of a patient's AD diagnosis, but scans cannot diagnose AD on their own. Scans are used more to rule out other possible diagnoses and to confirm a suspected diagnosis. CT (**computed tomography**) scans are commonly performed, as well as MRI (**magnetic resonance imaging**) scans in patients who are having problems with gait or balance. PET (**positron emission tomography**) and SPECT (**single photon emission computed tomography**) scans can be used to evaluate patterns of glucose (sugar) metabolism in the brain and to differentiate the patterns that are characteristic of Alzheimer's from those associated with vascular dementia and Pick's disease. PET scans are more precise than SPECT scans, but their cost is prohibitive.

Ethical considerations

With regard to genetic factors, tests are now available for the apoE4 gene implicated in late-onset Alzheimer's, but the American College of Medical Genetics and the American Neurological Association do not recommend these tests as of 2002. One reason is that the test results are not conclusive— about 20% of people who eventually develop AD do not carry this gene. Another important reason is the ethical implications of testing for a disease that presently has no cure, in terms of the psychological consequences for patients and their families, and the possible loss of health insurance for people found to be carrying the gene. These considerations may change, however, if researchers discover better treatments for primary dementia, more effective preventive methods, or more reliable genetic markers for AD.



Colored positron emission tomography (PET) brain scans comparing a normal brain (left) with the brain of a person with Alzheimer's disease. *(Photo Researchers, Inc. Reproduced by permission.) See color insert for color version of photo.*

Treatments

At present the mainstay of Alzheimer's treatment is medication, both to slow symptom progression and to manage the behavioral and psychiatric symptoms of AD.

Medications to slow symptom progression

The medications most commonly given to delay the progression of symptoms in Alzheimer's are a group of drugs called cholinesterase inhibitors. These drugs were approved by the FDA over a decade ago. They work by slowing down the body's destruction of the neurotransmitter acetylcholine.

The cholinesterase inhibitors include:

- **Tacrine** (Cognex). This drug is the oldest cholinesterase inhibitor in use. It is used less often than newer agents because it must be taken four times a day and may cause liver damage.
- **Donepezil** (Aricept). This drug is the one used most commonly as of 2002 to treat AD. It has fewer side effects than tacrine and can be given in one daily dose.

- **Rivastigmine** (Exelon). This drug is taken twice daily.
- **Galantamine** (Reminyl). This is the newest cholinesterase inhibitor, approved in late 2001. It acts on an additional acetylcholine receptor.

None of these medications provide more than modest benefits to patients with AD: they slow the progression of symptoms for about six months to a year in one-third to one-half of patients with AD. In addition, the cholinesterase inhibitors have side effects, most commonly nausea, vomiting, diarrhea, muscle cramps, and sleep disturbances.

Medications for BPSD

Medications are also prescribed to manage the behavioral and psychiatric symptoms of AD, which are often quite stressful for caregivers if the patient is being cared for at home. These medications are usually prescribed for specific symptoms:

- Delusions: Antipsychotic drugs, usually **haloperidol** (Haldol) or **risperidone** (Risperdal).
- Agitation: Short-term anti-anxiety drugs, usually **lorazepam** (Ativan) or **buspirone** (BuSpar).
- Depression: One of the selective serotonin reuptake inhibitors (SSRIs), at half the dosage for a young adult.
- Pain: Acetaminophen or a very low dose of codeine.

In general, older patients require lower dosages than those given to younger adults. Patients with AD are also more susceptible to the side effects of medications. For these reasons, physicians often recommend making changes in the patient's environment to reduce the behavioral symptoms before trying medications.

Alternative and complementary treatments

Some complementary therapies have been shown to benefit patients with Alzheimer's.

NATUROPATHY. A naturopathic approach to Alzheimer's includes supplementing antioxidant vitamins (vitamins A, E, and C) in the patient's diet, along with adding carotenoids, small amounts of selenium and zinc, and thiamin. Botanical supplements that have been said to improve cognitive function include Huperzine A, a Chinese tea, and an extract made from *Gingko biloba*, a tree that is native to China and is said to be the world's oldest living deciduous tree. GBE, or gingko biloba extract, is the most frequently used herbal medicine in Europe. It is available in Germany by prescription and in an over-the-counter form; and has been approved by the German Commission E for dementia-related memory loss. Gingko extract is thought to work in a manner similar to the cholinesterase inhibitors. At present the National Center for Complementary and Alternative Medicine (NCCAM) is conducting studies of gingko extract as a treatment for Alzheimer's.

MUSIC THERAPY. Music therapy has been found to calm agitated patients with Alzheimer's, to improve mood, and to enhance their long-term memory. Old familiar songs are particularly effective in improving recall. In other studies, music therapy has been shown to reduce sensations of chronic pain in patients with AD.

Prognosis

There is no cure for Alzheimer's disease as of 2002. The prognosis is progressive loss of mental and bodily functions leading to death within seven to ten years. Some patients, however, die within three years of diagnosis and others may survive for as long as fifteen.

Prevention

Researchers are considering several different strategies to prevent Alzheimer's, ranging from development of a vaccine to prevent the formation of beta amyloid plaques to finding a drug that would stop the conversion of APP to beta amyloid. As of 2002, the vaccine, which was originally developed and tested on mice, does not appear to have any serious side effects in humans. It is presently being tested in Phase II trials on human subjects.

See also Dementia; Mini mental state examination (MMSE)

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ORGANIZATIONS

Alzheimer's Association. 919 North Michigan Avenue, Suite 1100, Chicago, IL 60611-1676. (800) 272-3900 or (312) 335-8700. Fax: (312) 335-1110. <www.alz.org>.

Alzheimer's Disease Education and Referral (ADEAR) Center. P. O. Box 8250, Silver Spring, MD 20907-8250. (800) 438-4380. <www.alzheimers.org>.

Alzheimer's Disease International. 45–46 Lower Marsh, London SE1 7RG, UK. (+44) 20-7620-3011. Fax: (+44) 20-7401-7351. <www.alz.co.uk>.

National Center for Complementary and Alternative Medicine (NCCAM) Clearinghouse. P.O. Box 7923, Gaithersburg, MD 20898. (888) 644-6226. TTY: (866) 464-3615. Fax: (866) 464-3616. <www.nccam.nih.gov>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

National Institute of Neurological Disorders and Stroke (NINDS). Building 31, Room 8A06, 9000 Rockville Pike, Bethesda, MD 20892. (301) 496-5751. <www.ninds.nih.gov>.

OTHER

Safe Return Hotline. (888) 572-8566. This hotline provides information about registering a patient with AD with the Alzheimer's Association as a means of identification in the event that he or she wanders away from home.

Rebecca J. Frey, Ph.D.

Amantadine

Definition

Amantadine is a synthetic antiviral agent that also has strong antiparkinsonian properties. It is sold in the United States under the brand name Symmetrel, and is also available under its generic name.

Purpose

Amantadine is used to treat a group of side effects (called parkinsonian side effects) that include tremors, difficulty walking, and slack muscle tone. These side effects may occur in patients who are taking antipsychotic medications used to treat mental disorders such as **schizophrenia**. An unrelated use of amantadine is in the treatment of viral infections of some strains of influenza A.

Description

Some medicines, called antipsychotic drugs, that are used to treat schizophrenia and other mental disorders can cause side effects similar to the symptoms of Parkinson's disease. The patient does not have Parkinson's disease, but he or she may experience shaking in muscles while at rest, difficulty with voluntary movements, and poor muscle tone. These symptoms are similar to the symptoms of Parkinson's disease.

One way to eliminate these undesirable side effects is to stop taking the antipsychotic medicine. Unfortunately, the symptoms of the original mental disorder usually come back, so in most cases simply stopping the antipsychotic medication is not a reasonable option. Some drugs such as amantadine that control the symptoms of Parkinson's disease also control the parkinsonian side effects of antipsychotic medicines.

Amantadine works by restoring the chemical balance between dopamine and acetylcholine, two neurotransmitter chemicals in the **brain**. Taking amantadine along with the antipsychotic medicine helps to control symptoms of the mental disorder, while reducing parkinsonian side effects. Amantadine is in the same family of drugs (commonly known as anticholinergic drugs) as **biperiden** and **trihexyphenidyl**.

Recommended dosage

Amantadine is available in 100-mg tablets and capsules, as well as a syrup containing 50 mg of amantadine in each teaspoonful. For the treatment of drug-induced parkinsonian side effects, amantadine is usually given in a dose of 100 mg orally twice a day. Some patients may need a total daily dose as high as 300 mg. Patients who are taking other antiparkinsonian drugs at the same time may require lower daily doses of amantadine (100 mg daily, for example).

People with kidney disease or who are on hemodialysis must have their doses lowered. In these patients, doses may range from 100 mg daily to as little as 200 mg every seven days.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Parkinsonian—Related to symptoms associated with Parkinson's disease, a nervous system disorder characterized by abnormal muscle movement of the tongue, face, and neck, inability to walk or move quickly, walking in a shuffling manner, restlessness, and/or tremors.

Precautions

Amantadine increases the amount of the neurotransmitter dopamine (a central nervous system stimulant) in the brain. Because of this, patients with a history of epilepsy or other seizure disorders should be carefully monitored while taking this drug. This is especially true in the elderly and in patients with kidney disease. Amantadine may cause visual disturbances and affect mental alertness and coordination. People should not

operate dangerous machinery or motor vehicles while taking this drug.

Side effects

Five to ten percent of patients taking amantadine may experience the following nervous system side effects:

- dizziness or lightheadedness
- insomnia
- nervousness or anxiety
- impaired concentration

One to five percent of patients taking amantadine may experience the following nervous system side effects:

- irritability or agitation
- depression
- confusion
- lack of coordination
- sleepiness or nightmares
- fatigue
- headache

In addition, up to 1% of patients may experience **hallucinations**, euphoria (excitement), extreme forgetfulness, aggressive behavior, personality changes, or **seizures**. Seizures are the most serious of all the side effects associated with amantadine.

Gastrointestinal side effects may also occur in patients taking amantadine. Five to ten percent of people taking this drug experience nausea and up to 5% have dry mouth, loss of appetite, constipation, and vomiting. In most situations, amantadine may be continued and these side effects treated symptomatically.

One to five percent of patients taking amantadine have also reported a bluish coloring of their skin (usually on the legs) that is associated with enlargement of the blood vessels (called livedo reticularis). This side effect usually appears within one month to one year of starting the drug and subsides within weeks to months after the drug is discontinued. People who think they may be experiencing this or other side effects from any medication should tell their physician.

Interactions

Taking amantadine along with other drugs used to treat parkinsonian side effects may cause increased confusion or even hallucinations. The combination of amantadine and central nervous system stimulants (such as **amphetamines** or decongestants) may cause increased central nervous stimulation or increase the likelihood of seizures.

Resources

BOOKS

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Ambien *see* **Zolpidem**

Amitriptyline

Definition

Amitriptyline is a medication used to treat various forms of depression, pain associated with the nerves (neuropathic pain), and to prevent migraine headaches. It is sold in the United States under the brand names Elavil and Endep.

Purpose

Amitriptyline helps relieve depression and pain. This medication, usually given at bedtime, also helps patients sleep better.

Description

This medication is one of several tricyclic antidepressants, so-called because of the three-ring chemical structure common to these drugs. Amitriptyline acts to block reabsorption of **neurotransmitters** (chemicals that transmit nerve messages in the **brain**). Amitriptyline and the other tricyclic antidepressants are primarily used to treat mental depression but are increasingly being replaced by a newer and more effective group of antidepressant drugs called selective serotonin reuptake inhibitors (SSRIs). Amitriptyline is sometimes prescribed to help treat pain associated with cancer. In addition, it is sometimes prescribed for various types of chronic pain. Tablets are available in 10, 25, 50, 70, and 150 mg.

KEY TERMS

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Methylphenidate—A mild central nervous system stimulant that is used to treat hyperactivity.

Monoamine oxidase inhibitors (MAOIs)—A group of antidepressant drugs that decreases the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Urinary retention—Excessive storage of urine in the body.

Recommended dosage

The usual adult dose for pain management ranges from 10 mg to 150 mg at bedtime. Patients are generally started on a low dose and the amount may be increased as needed. Side effects, such as a dry mouth and drowsiness, may make it difficult to increase the dose in older adults. Bedtime dosing helps the patient sleep. Doctors generally prescribe 75–150 mg for depression. It is given at bedtime or in divided doses during the day. It may take 30 days for the patient to feel less depressed. Pain relief is usually noticed sooner than the mood change. Teens and older adults usually receive a lower dose. If the nightly dose is missed, it should not be taken the next morning. Taking amitriptyline during waking hours could result in noticeable side effects. Patients should check with their doctor if the daily dose is missed. Those on more than one dose per day should take a missed dose as soon as it is remembered but should not take two doses at the same time. While amitriptyline is usually administered orally, injectable amitriptyline is available. It should not be used in this form long-term; patients should switch to tablets as soon as possible.

Precautions

Patients should not stop taking this medication suddenly. The dose should gradually be decreased, then discontinued. If the drug is stopped abruptly, the patient may experience headache, nausea, or discomfort throughout the body, and a worsening of original symptoms. The effects of the medication last for three to seven days after it has been stopped, and older patients usually are more prone to some side effects such as drowsiness, dizziness, mental confusion, blurry vision, dry mouth, difficulty urinating, and constipation. Taking a lower dose may help resolve these problems. Patients may need to stop this medication before surgery.

Amitriptyline should not be given to anyone with allergies to the drug or to patients recovering from a heart attack. Patients taking the monoamine oxidase inhibitors (MAOIs) Parnate (**tranylcypromine**) and Nardil (**phenelzine**) —different types of antidepressants—should not use amitriptyline in combination. It should be administered with caution to patients with glaucoma, **seizures**, urinary retention, overactive thyroid, poor liver or kidney function, alcoholism, asthma, digestive disorders, enlarged prostate, seizures, or heart disease. This medication should not be given to children under 12 years of age. Pregnant women should discuss the risks and benefits of this medication with their doctor as fetal deformities have been associated with taking this drug during pregnancy. Women should not breast feed while using amitriptyline.

Side effects

Common side effects include dry mouth, drowsiness, constipation, and dizziness or lightheadedness when standing. Patients can suck on ice cubes or sugarless hard candy to combat the dry mouth. Increased fiber in the diet and additional fluids may help relieve constipation. Dizziness is usually caused by a drop in blood pressure when suddenly changing position. Patients should slowly rise from a sitting or lying position if dizziness is noticed. Amitriptyline may increase the risk of falls in older adults. Patients should not drive or operate machinery or appliances while under the influence of this drug. Alcohol and other central nervous system depressants can increase drowsiness. Amitriptyline may also produce blurry vision, irregular or fast heartbeat, high or low blood pressure, palpitations, and an increase or decrease in a diabetic patient's blood sugar levels. Patients' skin may become more sensitive to the sun and thus direct sunlight should be avoided by wearing protective clothing and the application of sunscreen with a protective factor of 15 or higher.

Amitriptyline may increase appetite, cause weight gain, or produce an unpleasant taste in the mouth. It may also cause diarrhea, vomiting, or heartburn. Taking this medication with food may decrease digestive side effects. Other less likely side effects include muscle tremors, nervousness, impaired sexual function, sweating, rash, itching, hair loss, ringing in the ears, or changes in the makeup of the patient's blood. Patients with **schizophrenia** may develop an increase in psychiatric symptoms.

Interactions

Patients should always tell all doctors and dentists that they are taking this medication. It may decrease the

effectiveness of some drugs used to treat high blood pressure and should not be taken with other antidepressants, epinephrine and other adrenaline-type drugs, or **methylphenidate**. Patients should not take over-the-counter medications without checking with their doctor. For instance, amitriptyline should not be taken with Tagamet (cimetidine) or Neosynephrine. Patients taking this drug should avoid the dietary supplements **St. John's wort**, belladonna, henbane, and scopolia. Black tea may decrease the absorption of this drug. Patients should ingest the drug and tea at least two hours apart.

See also Depression and depressive disorders

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Amnesia

Definition

Amnesia is a partial or total loss of memory.

Description

There are numerous causes of amnesia, including **stroke**, injury to the **brain**, surgery, alcoholism, encephalitis (inflammation of the brain), and **electroconvulsive therapy** (ECT, a treatment for various mental disorders in which electricity is sent to the brain through electrodes).

Contrary to the popular notion of amnesia—in which a person suffers a severe blow to the head, for example, and cannot recall his or her past life and experiences—the principal symptom of amnesia is the inability to retain new information, beginning at the point at which the amnesia began. The capacity to recall past experiences may vary, depending on the severity of the amnesia.

There are two types of amnesia: retrograde and anterograde. Retrograde amnesia refers to the loss of memory of one's past, and can vary from person to person. Some retain virtually full recall of things that happened prior to the onset of amnesia; others forget only their recent past, and still others lose all memory of their past lives. Anterograde amnesia refers to the inability to recall events or facts introduced since the amnesia began.

Amnesia is not always obvious to the casual observer—motor skills such as tying shoelaces and bike riding are retained, as is the ability to read and comprehend the meaning of words. Because of this phenomenon, researchers have suggested that there is more than one area of the brain used to store memory. General knowledge and perceptual skills may be stored in a memory separate from the one used to store personal facts.

Childhood amnesia, a term coined by Anna Freud in the late 1940s, refers to the fact that most people cannot recall childhood experiences during the first three to five years of life. It has been suggested that this type of amnesia occurs because children and adults organize memories in different ways based on their brain's physical development. Others believe children begin remembering facts and events once they have accumulated enough experience to be able to relate experiences to each other.

See also Amnestic disorders; Dissociative amnesia; Dissociative fugue

Amnestic disorders

Definition

The amnestic disorders are a group of disorders that involve loss of memories previously established, loss of the ability to create new memories, or loss of the ability to learn new information. As defined by the mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (2000), also known as *DSM-IV-TR*, the amnestic disorders result from two basic causes: general medical conditions that produce memory disturbances; and exposure to a chemical (drug of abuse, medication, or environmental toxin). An amnestic disorder whose cause cannot be definitely established may be given the **diagnosis** of amnestic disorder not otherwise specified.

KEY TERMS

Anterograde amnesia—Amnesia for events that occurred after a physical injury or emotional trauma but before the present moment.

Confabulation—In psychiatry, the filling-in of gaps in memory with false information that the patient believes to be true. It is not deliberate telling of lies.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Factitious disorder—A type of mental disturbance in which patients intentionally act physically or mentally ill without obvious benefits. It is distinguished from malingering by the absence of an obvious motive, and from conversion disorder by intentional production of symptoms.

Hypnotic—A type of medication that induces sleep.

Korsakoff's syndrome—A disorder of the central nervous system resulting from long-term thiamin deficiency. It is characterized by amnesia, confusion, confabulation, and unsteady gait; and is most commonly seen in alcoholics.

Malingering—Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

Orientation—In psychiatry, the ability to locate oneself in one's environment with respect to time, place and people.

Retrograde amnesia—Amnesia for events that occurred before a traumatic injury.

Thiamin—A B-vitamin that is essential to normal metabolism and nerve function, and whose absorption is affected by alcoholism.

Description

The amnestic disorders are characterized by problems with memory function. There is a range of symptoms associated with the amnestic disorders, as well as differences in the severity of symptoms. Some people experience difficulty recalling events that happened or facts that they learned before the onset of the amnestic disorder. This type of **amnesia** is called retrograde amnesia. Other people experience the inability to learn new facts or retain new memories, which is called anterograde amnesia. People with amnestic disorders do not usually forget all of their personal history and their identity, although memory loss of this degree of severity occurs in rare instances in patients with dissociative disorders.

Causes and symptoms

Causes

In general, amnestic disorders are caused by structural or chemical damage to parts of the **brain**. Problems remembering previously learned information vary widely according to the location and the severity of brain damage. The ability to learn and remember new information, however, is always affected in an amnestic disorder.

Amnestic disorder due to a general medical condition can be caused by head trauma, tumors, **stroke**, or cerebrovascular disease (disease affecting the blood vessels in the brain). Substance-induced amnestic disorder can be caused by alcoholism, long-term heavy drug use, or exposure to such toxins as lead, mercury, carbon monoxide, and certain insecticides. In cases of amnestic disorder caused by alcoholism, it is thought that the root of the disorder is a vitamin deficiency that is commonly associated with alcoholism, known as Korsakoff's syndrome. The causes of transient global amnesia, or TGA, are unclear.

Symptoms

In addition to problems with information recall and the formation of new memories, people with amnestic disorders are often disoriented with respect to time and space, which means that they are unable to tell an examiner where they are or what day of the week it is. Most patients with amnestic disorders lack insight into their loss of memory, which means that they will deny that

there is anything wrong with their memory in spite of evidence to the contrary. Others will admit that they have a memory problem but have no apparent emotional reaction to their condition. Some persons with amnestic disorders undergo a personality change; they may appear apathetic or bland, as if the distinctive features of their personality have been washed out of them.

Some people experiencing amnestic disorders confabulate, which means that they fill in memory gaps with false information that they believe to be true. Confabulation should not be confused with intentional lying. It is much more common in patients with temporary amnestic disorders than it is in people with long-term amnestic disorders.

Transient global amnesia (TGA) is characterized by episodes during which the patient is unable to create new memories or learn new information, and sometimes is unable to recall past memories. The episodes occur suddenly and are generally short. Patients with TGA often appear confused or bewildered.

Demographics

The overall incidence of the amnestic disorders is difficult to estimate. Amnestic disorders related to head injuries may affect people in any age group. Alcohol-induced amnestic disorder is most common in people over the age of 40 with histories of prolonged heavy alcohol use. Amnestic disorders resulting from the abuse of drugs other than alcohol are most common in people between the ages of 20 and 40. Transient global amnesia usually appears in people over 50. Only 3% of people who experience transient global amnesia have symptoms that recur within a year.

Diagnosis

Amnestic disorders may be self-reported, if the patient has retained insight into his or her memory problems. More often, however, the disorder is diagnosed because a friend, relative, employer, or acquaintance of the patient has become concerned about the memory loss or recognizes that the patient is confabulating, and takes the patient to a doctor for evaluation. Patients who are disoriented, or whose amnesia is associated with head trauma or substance abuse, may be taken to a hospital emergency room.

The doctor will first examine the patient for signs or symptoms of traumatic injury, substance abuse, or a general medical condition. He or she may order **imaging studies** to identify specific areas of brain injury, or laboratory tests of blood and urine samples to determine exposure to environmental toxins or recent consumption of alcohol or drugs of abuse. If general medical conditions and substance abuse are ruled out, the doctor may administer a brief test of the patient's cognitive status, such as the **mini-mental state examination** or MMSE. The MMSE is often used to evaluate a patient for **dementia**, which is characterized by several disturbances in cognitive functioning (speech problems, problems in recognizing a person's face, etc.) that are not present in amnestic disorders. The doctor may also test the patient's ability to repeat a string of numbers (the so-called digit span test) in order to rule out **delirium**. Patients with an amnestic disorder can usually pay attention well enough to repeat a sequence of numbers whereas patients with delirium have difficulty focusing or shifting their attention. In some cases the patient may also be examined by a neurologist (a doctor who specializes in disorders of the central nervous system)

If there is no evidence of a medical condition or substance use that would explain the patient's memory problems, the doctor may test the patient's memory several times in order to rule out **malingering** or a **factitious disorder**. Patients who are faking the symptoms of an amnestic disorder will usually give inconsistent answers to memory tests if they are tested more than once.

DSM-IV-TR specifies three general categories of amnestic disorders. These are: amnestic disorder due to a general medical condition, substance-induced persisting amnestic disorder, and amnestic disorder not otherwise specified. The basic criterion for diagnosing an amnestic disorder is the development of problems remembering information or events that the patient previously knew, or inability to learn new information or remember new events. In addition, the memory disturbance must be sufficiently severe to affect the patient's social and occupational functioning, and to represent a noticeable decline from the patient's previous level of functioning. *DSM-IV-TR* also specifies that the memory problems cannot occur only during delirium, dementia, substance use or withdrawal.

Treatments

There are no treatments that have been proved effective in most cases of amnestic disorder, as of 2002. Many patients recover slowly over time, and sometimes recover memories that were formed before the onset of the amnestic disorder. Patients generally recover from transient global amnesia without treatment. In people judged to have the signs that often lead to alcohol-induced persisting amnestic disorder, treatment with thiamin may stop the disorder from developing.

Prognosis

Amnestic disorders caused by alcoholism do not generally improve significantly over time, although in a small number of cases the patient's condition improves completely. In many cases the symptoms are severe, and in some cases warrant long-term care for the patient to make sure his or her daily needs are met. Other substance-induced amnestic disorders have a variable rate of recovery, although in many cases full recovery does eventually occur. Transient global amnesia usually resolves fully.

Prevention

Amnestic disorders resulting from trauma are not generally considered preventable. Avoiding exposure to environmental toxins, refraining from abuse of alcohol or other substances, and maintaining a balanced diet may help to prevent some forms of amnestic disorders.

See also Dissociative amnesia

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Amobarbitol *see* **Barbiturates**

Amoxapine

Definition

Amoxapine is an oral tricyclic antidepressant. Formerly sold in the United States under the brand name Asendin, it is now manufactured and sold only under its generic name.

Purpose

Amoxapine is used primarily to treat depression and to treat the combination of symptoms of anxiety and depression. Like most antidepressants of this chemical and pharmacological class, amoxapine has also been used in limited numbers of patients to treat **panic disorder**, **obsessive-compulsive disorder**, **attention-deficit/hyperactivity disorder**, **enuresis** (bed-wetting), eating disorders such as **bulimia nervosa**, cocaine dependency, and the depressive phase of bipolar (manic-depressive) disorder. It has also been used to support smoking cessation programs.

Description

Tricyclic antidepressants act to change the balance of naturally occurring chemicals in the **brain** that regulate the transmission of nerve impulses between cells. Amoxapine acts primarily by increasing the concentration of norepinephrine and serotonin (both chemicals that stimulate nerve cells) and, to a lesser extent, by blocking the action of another brain chemical, acetylcholine. Amoxapine shares most of the properties of other tricyclic antidepressants, such as **amitriptyline**, **clomipramine**, **desipramine**, **imipramine**, **nortriptyline**, **protriptyline**, and **trimipramine**. Studies comparing amoxapine with these other drugs have shown that amoxapine is no more or less effective than other antidepressants of its type. Its choice for treatment is as much a function of physician preference as any other factor.

The therapeutic effects of amoxapine, like other antidepressants, appear slowly. Maximum benefit is often not evident for at least two weeks after starting the drug. People taking amoxapine should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Recommended dosage

As with any antidepressant, amoxapine must be adjusted by the physician to produce the desired therapeutic effect. Amoxapine is available as 25-mg, 50-mg, 100-mg, and 150-mg oral tablets. Therapy is usually started at 100 to 150 mg per day and increased to 200 to 300 mg daily

by the end of the first week. If no improvement is seen at this dose after two weeks, the physician may increase the dose up to 400 mg per day in outpatients and up to 600 mg per day in hospitalized patients. Doses up to 300 mg may be given in single or divided doses. Doses of more than 300 mg per day should be divided in two or three doses daily.

Because of changes in drug metabolism of older patients, starting at about age 60, the initial dose of amoxapine should be adjusted downward to 50 to 75 mg per day and increased to 100 to 150 mg daily by the end of the first week. Some older patients may require up to 300 mg daily, but doses should never be increased beyond that.

Precautions

Like all tricyclic antidepressants, amoxapine should be used cautiously and with close physician supervision in people, especially the elderly, who have benign prostatic hypertrophy, urinary retention, and glaucoma, especially angle-closure glaucoma (the most severe form). Before starting treatment, people with these conditions should discuss the relative risks and benefits of treatment with their doctors to help determine if amoxapine is the right antidepressant for them.

A common problem with tricyclic antidepressants is sedation (drowsiness, lack of physical and mental alertness). This side effect is especially noticeable early in therapy. In most patients, sedation decreases or disappears entirely with time, but until then patients taking amoxapine should not perform hazardous activities requiring mental alertness or coordination. The sedative effect is increased when amoxapine is taken with other central nervous system depressants, such as alcoholic beverages, sleeping medications, other sedatives, or antihistamines. It may be dangerous to take amoxapine in combination with these substances. Amoxapine may increase the possibility of having **seizures**. Patients should tell their physician if they have a history of seizures, including seizures brought on by the abuse of drugs or alcohol. These people should use amoxapine only with caution and be closely monitored by their physician.

Amoxapine may increase heart rate and stress on the heart. It may be dangerous for people with cardiovascular disease, especially those who have recently had a heart attack, to take this drug or other antidepressants in the same pharmacological class. In rare cases in which patients with cardiovascular disease must receive amoxapine, they should be monitored closely for cardiac rhythm disturbances and signs of cardiac stress or damage.

Side effects

Amoxapine shares side effects common to all tricyclic antidepressants. The most frequent of these are dry mouth, constipation, urinary retention, increased heart rate, sedation, irritability, dizziness, and decreased coordination. As with most side effects associated with tricyclic antidepressants, the intensity is highest at the beginning of therapy and tends to decrease with continued use.

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Patients may also chew sugarless gum or suck on sugarless candy in order to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement who take amoxapine may be especially likely to have problems with urinary retention. Symptoms include having difficulty starting a urine flow and more difficulty than usual passing urine. In most cases, urinary retention is managed with dose reduction or by switching to another type of antidepressant. In extreme cases, patients may require treatment with bethane-

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Benign prostate hypertrophy—Enlargement of the prostate gland.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

chol, a drug that reverses this particular side effect. People who think they may be experiencing any side effects from this or any other medication should tell their physicians.

Interactions

Dangerously high blood pressure has resulted from the combination of tricyclic antidepressants, such as amoxapine, and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Because of this, amoxapine should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting amoxapine or any other tricyclic antidepressant. The same holds true when discontinuing amoxapine and starting an MAO inhibitor.

Amoxapine may decrease the blood pressure–lowering effects of **clonidine**. Patients who take both drugs should be monitored for loss of blood-pressure control and the dose of clonidine may be increased as needed.

The sedative effects of amoxapine are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or medications used for other mental disorders such as **schizophrenia**. The anticholinergic effects of amoxapine are additive with other anticholinergic drugs such as **benztropine**, **biperiden**, **trihexyphenidyl**, and antihistamines.

See also Neurotransmitters

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Amphetamines

Definition

Amphetamines are a group of drugs that stimulate the central nervous system. Some of the brand names of amphetamines sold in the United States are Dexedrine, Biphetamine, Das, Dexampex, Ferndex, Oxydess II, Spancap No 1, Desoxyn, and Methampex. Some generic names of amphetamines include amphetamine, dextroamphetamine, and methamphetamine.

Purpose

Amphetamines stimulate the nervous system and are used in the treatment of depression, attention-deficit disorder, **obesity**, and **narcolepsy**, a disorder that causes individuals to fall asleep at inappropriate times during the day. Amphetamines produce considerable side effects and are especially toxic in large quantities. Amphetamines are commonly abused recreational drugs and are highly addictive.

Description

Amphetamines are usually given orally and their effects can last for hours. Amphetamines produce their effects by altering chemicals that transmit nerve messages in the body.

Recommended dosage

The typical dose for amphetamines in the treatment of narcolepsy in adults ranges from 5 mg to 60 mg per day. These daily doses are usually divided into at least two small doses taken during the day. Doses usually start on the low end of the range and are increased until the desired effects occur. Children over the age of 12 years with narcolepsy receive 10 mg per day initially. Children between the ages of six and 12 years start with 5 mg per day. The typical dose for adults with obesity ranges from 5 mg to 30 mg per day given in divided doses. The medication is usually given about one-half hour to one hour before meals.

The typical starting dose of amphetamines given to children with attention-deficit disorder over the age of six years is 5 mg per day. This is increased by 5 mg per day over a period of time until the desired effect is achieved. Children under the age of six years with this condition are usually started at 2.5 mg per day.

Precautions

People who are taking amphetamines should not stop taking these drugs suddenly. The dose should be lowered gradually and then discontinued. Amphetamines should only be used while under the supervision of a physician. People should generally take the drug early in the day so that it does not interfere with sleep at night. Hazardous activities should be avoided until the person's condition has been stabilized with medication. The effects of amphetamine can last up to 20 hours after the medication has last been taken. Amphetamine therapy given to women for medical reasons does not present a

significant risk to the developing fetus for congenital disorders. In such cases, there may be mild withdrawal in the newborn. However, illicit use of amphetamines for non-medical reasons presents a significant risk to the fetus and the newborn because of uncontrolled doses.

Amphetamines are highly addictive and should be used only if alternative approaches have failed. They should be used with great caution in children under three years of age, anyone with a history of slightly elevated blood pressure, people with neurological tics, and in individuals with Tourette's syndrome. Amphetamines should not be taken by individuals with a history of an overactive thyroid, those with moderate-to-severe high blood pressure, those with the eye disease called glaucoma, those who have severe arteriosclerosis (hardening of the arteries), or anyone with psychotic symptoms (**hallucinations** and **delusions**). Individuals with a history of drug abuse, psychological agitation, or cardiovascular system disease should also not receive amphetamine therapy. In addition, patients who have taken MAO inhibitors, a type of antidepressant, within the last 14 days should not receive amphetamines. MAO inhibitors include **phenelzine** (Nardil), and **tranylcypromine** (Parnate).

Side effects

The most common side effects that are associated with amphetamines include the development of an irregular heartbeat, increased heart rate, increased blood pressure, dizziness, **insomnia**, restlessness, headache, shakiness, dry mouth, metallic taste, diarrhea, constipation, and weight loss. Other side effects can include changes in sexual drive, nausea, vomiting, allergic reactions, chills, depression, irritability, and other problems involving the digestive system. High doses, whether for medical purposes or illicit ones, can cause **addiction**, dependence, increased aggression, and, in some cases, psychotic episodes.

Interactions

Patients taking amphetamines should always tell their physicians and dentists that they are using this medication. Patients should consult their physician before taking any over-the-counter medication while taking amphetamines. The interaction between over-the-counter cold medications with amphetamine, for instance, is particularly dangerous because this combination can significantly increase blood pressure. Such cold medications should be avoided when using amphetamine unless a physician has carefully analyzed the combination.

KEY TERMS

Anticonvulsant drugs—Medications that relieve or prevent seizures.

Arteriosclerosis—A thickening, hardening, and loss of elasticity of the walls of the arteries.

Attention-deficit disorder—A condition that mostly affects children and involves the inability to concentrate on various tasks.

Congenital—Present at birth.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

MAO inhibitors—A group of antidepressant drugs that decreases the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Tic—A sudden involuntary behavior that is difficult or impossible for the person to suppress. Tics may be either motor (related to movement) or vocal, and may become more pronounced under stress.

Tourette's syndrome—Neurological disorder characterized by multiple involuntary movements and uncontrollable vocalizations called tics that come and go over years, usually beginning in childhood and becoming chronic. Sometimes the tics include inappropriate language.

Tricyclic antidepressants—Antidepressant medications that have the common characteristic of a three-ring nucleus in their chemical structure. Imipramine and amitriptyline are examples of tricyclic antidepressants.

The combination of amphetamines and antacids slows down the ability of the body to eliminate the amphetamine. Furazolidone (Furoxone) combined with amphetamine can significantly increase blood pressure. Sodium bicarbonate can reduce the amount of amphetamine eliminated from the body and dangerously increase amphetamine levels in the body. Certain medications taken to control high blood pressure, including guanadrel (Hylorel) and guanethidine (Ismelin), MAO inhibitors, and selegiline (Eldepryl) should not be used in conjunction with amphetamines. In addition, tricyclic antidepressants [including **desipramine** (Norpramin) and **imipramine** (Tofranil)],

antihistamines, and anticonvulsant drugs should not be combined with amphetamines.

See also Attention-deficit/hyperactivity disorder; Tic disorders

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Amphetamines and related disorders

Definition

Amphetamines are a group of powerful and highly addictive substances that dramatically affect the central nervous system. They induce a feeling of well-being and improve alertness, attention, and performance on various cognitive and motor tasks. Closely related are the so-called "designer amphetamines," the most well known of which is the "club drug" MDMA, best known as "ecstasy." Finally, some over-the-counter drugs used as **appetite suppressants** also have amphetamine-like action. Amphetamine-related disorders refer to the effects of abuse, dependence, and acute intoxication stemming from inappropriate amphetamine and amphetamine-related drug usage.

Description

Several amphetamines are currently available in the United States. These include dextroamphetamine (Dexedrine), methamphetamine (Desoxyn), and **methylphenidate** (Ritalin). These Schedule II stimulants, known to be highly addictive, require a triplicate prescription that cannot be refilled. Amphetamines are also known as sympathomimetics, stimulants, and psychostimulants. Methamphetamine, the most common illegally produced amphetamine, goes by the street name of "speed," "meth," and "chalk." When it is smoked, it is called "ice," "crystal," "crank," and "glass." Methamphetamine is a white, odorless, bitter-tasting crystalline powder that dissolves in water or alcohol.

The leaves of the East African bush *Catha edulis* can be chewed for their stimulant effects. This drug, cathinone or Khat, has an effect on most of the central nervous system, in addition providing the other properties of amphetamines. Illegal laboratories have begun making methcathinone, which has effects similar to cathinone. Methcathinone, also known as "crank," is easily synthesized from ephedrine or pseudoephedrine.

Amphetamines were initially produced for medical use, and were first used in nasal decongestants and bronchial inhalers. Early in the 1900s, they were also used to treat several medical and psychiatric conditions, including **narcolepsy** (a rare condition in which an individuals falls asleep at dangerous and inappropriate moments and cannot maintain normal alertness), attention-deficit disorders, **obesity**, and depression. They are still used to treat these disorders today.

Amphetamine-like substances called ephedrine and propranolamine are available over the counter in the United States and are used as nasal decongestants. Phenylpropanolamine is also used as an appetite suppressant, and is available over the counter as well. These are less potent than the classic amphetamines, but are still subject to abuse, partly because of their ready availability and low price.

In the 1970s, governmental agencies initiated restrictions increasing the difficulty of obtaining amphetamines legally through prescription. During this same time period, a drug chemically related to the amphetamines began to be produced. This so-called designer drug, best known as "ecstasy," but also as MDMA, XTC, and Adam, has behavioral effects that combine amphetamine-like and hallucinogen-like properties.

The structure of amphetamines differs significantly from that of cocaine, even though both are stimulants with similar behavioral and physiological effects. Like cocaine, amphetamine results in an accumulation of the neurotransmitter dopamine. It is this excessive dopamine concentration that appears to produce the stimulation and feelings of euphoria experienced by the user. Cocaine is much more quickly metabolized and removed from the body, whereas amphetamines have a much longer duration of action. A large percentage of the drug remains unchanged in the body, leading to prolonged stimulant effects.

The handbook that mental health professionals use to diagnose mental disorders is the ***Diagnostic and***

KEY TERMS

Amphetamine abuse—An amphetamine problem in which the user experiences negative consequences from the use, but has not reached the point of dependence.

Amphetamine dependence—The most serious type of amphetamine problem.

Amphetamine intoxication—The effects on the body that develop during or shortly after amphetamine use.

Amphetamine withdrawal—Symptoms that develop shortly after reducing or stopping heavy amphetamine use.

Amphetamines—A group of powerful and highly addictive substances that stimulate the central nervous system. May be prescribed for various medical conditions, but are often purchased illicitly and abused.

Catecholamine—A group of neurotransmitters synthesized from the amino acid tyrosine and released by the hypothalamic-pituitary-adrenal system in the brain in response to acute stress. The catecholamines include dopamine, serotomin, norepinephrine, and epinephrine.

Catha edulis—Leaves of an East African bush that can be chewed for their stimulant effect.

Designer amphetamines—Substances close in chemical structure to classic amphetamines that provide both stimulant and hallucinogenic effects.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions. Large amounts of dopamine are released following ingestion of amphetamines.

Ecstasy—Best known of the so-called designer amphetamines, also known as MDMA. It produces both stimulant and hallucinogenic effects.

Ephedrine—An amphetamine-like substance used as a nasal decongestant.

Formication—The sensation of bugs creeping on the skin.

Hyperthermia—Elevated body temperature resulting from ingestion of amphetamines.

Methamphetamine—The most common illegally produced amphetamine.

Propranolamine—An amphetamine-like substance used as a nasal decongestant and diet aid.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression. Large amounts of serotonin are released after ingestion of MDMA.

"Speed run"—The episodic bingeing on amphetamines.

Statistical Manual of Mental Disorders, also known as the *DSM*. The 2000 edition of this manual (the Fourth Edition Text Revision, also known as *DSM-IV-TR*) describes four separate amphetamine-related disorders. These are:

- Amphetamine dependence, which refers to chronic or episodic binges (known as "speed runs"), with brief drug-free periods of time in between use.
- Amphetamine abuse, which is less severe than dependence. Individuals diagnosed with amphetamine abuse have milder but nevertheless still substantial problems due to their drug usage.
- Amphetamine intoxication, which refers to serious maladaptive behavioral or psychological changes that develop during, or shortly after, use of an amphetamine or related substance.
- Amphetamine withdrawal, which refers to symptoms that develop within a few hours to several days after reducing or stopping heavy and prolonged amphetamine use. Withdrawal symptoms are, in general, opposite to those seen during intoxication and include **fatigue**, vivid and unpleasant dreams, **insomnia** or **hypersomnia** (too much sleep), increased appetite and agitation or slowing down.

Causes and symptoms

Causes

All amphetamines are rapidly absorbed when taken orally, and even more rapidly absorbed when smoked, snorted, or injected. Tolerance develops with both standard and designer amphetamines, leading to the need for increasing doses by the user.

The classic amphetamines, dextroamphetamine, methamphetamine, and methylphenidate, produce their primary effects by causing the release of catecholamines, especially dopamine, in the **brain**. These effects are particularly strong in areas of the brain associated with pleasure, specifically, the cerebral cortex and the limbic system, known as the "reward pathway." The effect on this pathway is probably responsible for the addicting quality of the amphetamines. Catecholamines are any of several compounds found naturally in the body and act as hormones or **neurotransmitters** in the sympathetic nervous system. Dopamine, an intermediate substance that emerges from the biosynthesis of ephinephrine and norepinephrine, is one of those compounds.

Designer amphetamines, most notably MDMA, causes the release of catecholamines, dopamine and norepinephrine; and in addition, releases serotonin. Serotonin, also a neurotransmitter, produces hallucinogenic effects. The clinical effects of designer amphetamines blend the effects of classic amphetamines with those of hallucinogenic drugs, such as LSD.

Symptoms

CLASSIC AMPHETAMINES. According to the *DSM-IV-TR*, symptoms of heavy, chronic, or episodic use of amphetamine, known as amphetamine dependence, can be very serious. Amphetamine dependence is characterized by compulsive drug-seeking and drug use leading to functional and molecular changes in the brain. Aggressive or violent behavior may occur, especially when high doses are ingested. The individual may develop anxiety or paranoid ideas, also with the possibility of experiencing terrifying psychotic episodes that resemble **schizophrenia**, with visual or auditory **hallucinations**, **delusions** such as the sensation of insects creeping on the skin, known as "formication." hyperactivity, hypersexuality, confusion, and incoherence. Amphetamine-induced **psychosis** differs from true psychosis in that despite other symptoms, the disorganized thinking that is a hallmark of schizophrenia tends to be absent. Amphetamine dependence consistently affects relationships at home, school and/or work.

Amphetamine abuse is less serious than dependence, but can cause milder versions of the symptoms described above, as well as problems with family, school, and work. Legal problems may stem from aggressive behavior while using, or from obtaining drugs illegally. Individuals may continue to use despite the awareness that usage negatively impacts all areas of their lives.

Acute amphetamine intoxication begins with a "high" feeling which may be followed by feelings of euphoria. The user experiences enhanced energy, becoming more outgoing and talkative, and more alert. Other symptoms include anxiety, tension, grandiosity, repetitive behavior, anger, fighting, and impaired judgment.

In both acute and chronic intoxication, the individual may experience dulled feelings, along with fatigue or sadness, and social withdrawal. These behavioral and psychological changes are accompanied by other signs and symptoms including increased or irregular heartbeat, dilation of the pupils, elevated or lowered blood pressure, heavy perspiring or chills, nausea and/or vomiting, motor agitation or retardation, muscle weakness, respiratory depression, chest pain, and eventually confusion, **seizures**, coma, or a variety of cardiovascular problems, including **stroke**. With amphetamine overdoses, death can result if treatment is not received immediately. Long-term abuse can lead to memory loss as well, and contributes to increased transmission of hepatitis and HIV/AIDs. Impaired social and work functioning is another hallmark of both acute and chronic intoxication.

Following amphetamine intoxication, a "crash" occurs with symptoms of anxiety, shakiness, depressed mood, lethargy, fatigue, nightmares, headache, perspiring, muscle cramps, stomach cramps, and increased appetite. Withdrawal symptoms usually peak in two to four days and are gone within one week. The most serious withdrawal symptom is depression, possibly very severe and leading to suicidal thoughts.

DESIGNER AMPHETAMINES. Use of so-called designer amphetamines, the best-known of which is MDMA, leads to symptoms of classic amphetamine use. Users report a sense of feeling unusual closeness with other people and enhanced personal comfort. They describe seeing an increased luminescence of objects in the environment, although these hallucinogenic effects are less than those caused by other hallucinogens, such as LSD. Some psychotherapists have suggested further research into the possible use of designer amphetamines in conjunction with **psychotherapy**. This idea is highly controversial, however.

Like classic amphetamines, use of MDMA produces cardiovascular effects of increased blood pressure, heart rate, and heart oxygen consumption. People with pre-existing heart disease are at increased risk of cardiovascular catastrophe resulting from MDMA use. MDMA is not processed and removed from the body quickly, and remains active for a long period of time. As a result, toxicity may rise dramatically when users take multiple doses over brief time periods, leading to harmful reactions such as dehydration, hyperthermia, and seizures.

MDMA tablets often contain other drugs, such as ephedrine, a stimulant, and dextromethorphan, a cough suppressant with PCP-like effects at high doses. These

additives increase the harmful effects of MDMA. It appears also to have toxic effects on the brain's serotonin system. In tests of learning and memory, MDMA users perform more poorly than nonusers. Research with primates show that MDMA can cause long-lasting brain damage. Exposure to MDMA during the period of pregnancy in which the fetal brain is developing is associated with learning deficits that last into adulthood.

Demographics

Classic amphetamines

Amphetamine dependence and abuse occur at all levels of society, most commonly among 18- to 30-year-olds. Intravenous use is more common among individuals from lower socioeconomic groups, and has a male-to-female ratio of three or four to one. Among non-intravenous users, males and females are relatively equally divided.

An annual study known as the *Monitoring the Future Study*, or *MTF*, examines drug use and attitudes related to drugs held by American teenagers. It focuses primarily on teens in the eighth, 10th, and 12th grades, but also on young adults across the country. Recent data on methamphetamine use showed that in 1997, 4.4% of 12th graders had tried crystal methamphetamine at least once in their lifetime. This represented an increase from 2.7% in 1990. Also in 1997, 2.3% of seniors reported having used crystal methamphetamine at least once during the past year. This represented an increase from 1.3% in 1990.

According to the 2000 *National Household Survey on Drug Abuse*, approximately 8.8 million Americans have tried methamphetamine at some time during their lives. Data from the 2000 *Drug Abuse Warning Network (DAWN)*, which collects information on drug usage problems from emergency room departments in 21 metropolitan areas found that methamphetamine-related problems increased from 10,400 in 1999 to 13,500 in 2000, an increase of 30%.

Treatment admissions reports by the National Institute of Drug Abuse (NIDA) *Community Epidemiology Work Group*, or *CEWG*, showed that as of June 2001, methamphetamine usage continued to be the leading drug of abuse among clients in treatment in the San Diego area and Hawaii. Methamphetamine is the most prevalent illegal drug in San Diego. Both San Francisco and Honolulu also reported substantial methamphetamine use problems during the late 1990s. Increased use was also reported in Denver, Los Angeles, Minneapolis/St.Paul, Phoenix, Seattle, and Tucson.

Amphetamines are a group of powerful and highly addictive substances that dramatically affect the central nervous system. Closely related are the so-called "designer amphetamines," the most well-known of which is the "club drug" MDMA, or "ecstasy" (pictured above). Use of MDMA produces increased blood pressure, heart rate, and heart oxygen consumption. MDMA is not processed and removed from the body quickly, and remains active for a long period of time. As a result, toxicity may rise dramatically when users take multiple doses over brief time periods, leading to harmful reactions such as dehydration, hyperthermia, and seizures. *(Andrew Brookes/ CORBIS. Photo reproduced by permission.)*

Designer amphetamines

According to the NIDA, at a time when abuse of most illicit drugs has leveled off or declined slightly among youth in the United States, one drug has greatly increased in popularity: MDMA. It is the only drug for which an increase in use was shown among American 10th and 12th graders between 1999-2000. That year, even younger adolescents at the eighth-grade level showed an increase in use. Other evidence from NIDA shows that MDMA use is also increasing among older Americans who attend dance clubs, or all-night parties called "raves." Increasingly, Americans of diverse ages, social classes, and sexual orientations are using this drug in diverse social settings around the country.

Evidence indicates that in 2001, the rate of increase in teen use of MDMA slowed down. At the time the 2001 survey was conducted, of teens in grade eight, 1.8% reported using MDMA in the last month. Teens in grade 10 reported a 2.6% use, and in grade 12, 2.8% use in the last month. Survey data from 2001 show that an increas-

ing number of high school seniors—nearly half— say they believe that MDMA poses a great health risk.

Diagnosis

Classic amphetamines

Four classic ampetamine-related diagnostic categories are listed in the *DSM-IV-TR*. These are:

- amphetamine dependence
- amphetamine abuse
- amphetamine intoxication
- amphetamine withdrawal

Amphetamine dependence refers to chronic or episodic use of amphetamine involving drug binges known as "speed runs." These episodes are punctuated by brief, drug-free periods. Aggressive or violent behavior is associated with amphetamine dependence, particularly when high doses are ingested. Intense but temporary anxiety may occur, as well as paranoid ideas and psychotic behavior resembling schizophrenia. Increased tolerance and withdrawal symptoms are part of the diagnostic picture. Conversely, some individuals with amphetamine dependence become sensitized to the drug, experiencing increasingly greater stimulant, and other negative mental or neurological effects, even from small doses.

Amphetamine abuse, while not as serious as amphetamine dependence, can also cause multiple problems. Legal difficulties are common, in addition to increased arguments with family and friends. If tolerance or withdrawal occur, amphetamine dependence is diagnosed.

Amphetamine intoxication refers to serious behavioral or psychological changes that develop during, or shortly after, use of amphetamine. Intoxication begins with a "high" feeling, followed by euphoria, enhanced energy, talkativeness, hyperactivity, restlessness, hypervigilance indicated by an individual's extreme sensitivity, and closely observant of everything in the environment). Other symptoms are anxiety, tension, repetitive behavior, anger, fighting, and impaired judgment. With chronic intoxication, there may be fatigue or sadness and withdrawal from others. Other signs and symptoms of intoxication are increased heartrate, dilation of the pupils, elevated or lowered blood pressure, perspiration or chills, nausea or vomiting, weight loss, cardiac irregularities and, eventually, confusion, seizures, coma, or death.

During amphetamine withdrawal, intense symptoms of depression are typical. Additional diagnostic symptoms are fatigue, vivid and unpleasant dreams, insomnia or sleeping too much, increased appetite, and agitation.

Treatments

According to the NIDA, the most effective treatments for amphetamine **addiction** are cognitive-behavioral interventions. These are psychotherapeutic approaches that help the individual learn to identify their problematic patterns of thoughts and beliefs, and to change them. As a result of changed thoughts and beliefs, feelings become more manageable and less painful. They also help individuals increase their skills for coping with life's stressors. Amphetamine recovery groups, and Narcotics Anonymous also appear to help, along with cognitive-behavioral interventions.

No specific medications are known to exist that are helpful for treating amphetamine dependence. On occasion, antidepressant medications can help combat the depressive symptoms frequently experienced by newly abstinent amphetamine users.

Overdoses of amphetamines are treated in established ways in emergency rooms. Because hyperthermia (elevated body temperature), and convulsions are common, emergency room treatment focuses on reducing body temperature and administering anticonvulsant medications.

Acute methamphetamine intoxication is often handled by observation in a safe, quiet environment. When extreme anxiety or panic is part of the reaction, treatment with anti-anxiety medications may be helpful. In cases of methamphetamine-induced psychoses, short-term use of antipsychotic medications is usually successful.

Prognosis

Classic amphetamines

According to the *DSM-IV-TR*, some individuals who develop abuse or dependence on amphetamines initiate use in an attempt to control their weight. Others become introduced through the illegal market. Dependence can occur very quickly when the substance is used intravenously, or is smoked. The few long-term data available show a tendency for people who have been dependent on amphetamines to decrease or stop using them after eight to 10 years. This may result from the development of adverse mental and physical effects that emerge with long-term dependence. Few data are available on the long-term course of abuse.

Designer amphetamines

The NIDA reports that studies provide direct evidence that chronic use of MDMA causes brain damage in humans. Using advanced brain imaging techniques, one study found that MDMA harms neurons that release serotonin. Serotonin plays an important role in regulating memory and other mental functions.

In a related study, researchers found that heavy MDMA users have memory problems that persist for at least two weeks after stopping use of the drug. Both studies strongly suggest that the extent of damage is directly related to the amount of MDMA used.

Prevention

In 1999, NIDA began a program known as the "Club Drug Initiative" in response to recent increases in abuse of MDMA and related drugs. This ongoing program seeks to increase awareness of the dangers of these drugs among teens, young adults, parents, and communities.

Research indicates a pervasive perception among users that MDMA is a "fun" drug with minimal risks. This myth might point to the main reason for the widespread increase in the drug's abuse. The Club Drug Initiative seeks to make the dangers of MDMA use far better known. Evidence of the program's initial success of this initiative might be seen in what is considered a growing perception by high school seniors that MDMA is a dangerous drug.

See also Addiction; Appetite suppressants; Cognitve-behavioral therapy; Disease concept of chemical dependency; Narcolepsy; Obesity; Relapse and relapse prevention; Self-help groups; Support groups

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National Institute on Drug Abuse (NIDA). U.S. Department of Health and Human Services, 5600 Fishers Ln., Rockville, MD 20857. (301) 443-6245. <http://www.nida.nih.gov>.

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Anafranil *see* **Clomipramine**

Anorexia nervosa

Definition

Anorexia nervosa (AN) is an eating disorder characterized by an intense fear of gaining weight and becoming fat. Because of this fear, the affected individual starves herself or himself, and the person's weight falls to about 85% (or less) of the normal weight for age and height.

Description

AN affects females more commonly than males—90% of those affected are female. Typically, the disorder begins when an adolescent or young woman of normal or slightly overweight stature decides to diet. As weight falls, the intensity and **obsession** with dieting increases. Affected individuals may also increase physical exertion or exercise as weight decreases to lose more pounds. An affected person develops peculiar rules concerning exercise and eating. Weight loss and avoidance of food is equated in these patients with a sense of accomplishment and success. Weight gain is viewed as a sign of weakness (succumbing to eat food) and as failure. Eventually, the affected person becomes increasingly focused on losing weight and devotes most efforts to dieting and exercise.

Anorexia nervosa is a complex eating disorder that has biological, psychological, and social consequences for those who suffer from it. When diagnosed early, the prognosis for AN is good.

Causes and symptoms

Causes

The exact causes of AN are not currently known, but the current thinking about AN is that it is caused by multiple factors. There are several models that can identify risk factors and psychological conditions that predispose people to develop AN. The predisposing risk factors include:

- female gender
- perfectionism
- personality factors, including being eager to please other people and high expectations for oneself
- family history of eating disorders
- living in an industrialized society
- difficulty communicating negative emotions such as anger or fear
- difficulty resolving problems or conflict
- low self-esteem

KEY TERMS

Amenorrhea—Absence of menstrual periods.

Anemia—Condition that results when there is a deficiency of oxygen in the blood. Can cause fatigue and impair mental functions.

False-positive—A test result that is positive for a specific condition or disorder, but this result is inaccurate.

Lanugo—Downy hair, usually associated with infants, that sometimes develops on the face and back of people affected by anorexia nervosa.

Specialists in **family therapy** have demonstrated that dysfunctional family relationships and impaired family interaction can contribute to the development of AN. Mothers of persons with AN tend to be intrusive, perfectionistic, overprotective, and have a fear of separation. Fathers of AN-affected individuals are often described as passive, withdrawn, moody, emotionally constricted, obsessional, and ineffective. Sociocultural factors include the messages given by society and the culture about women's roles and the thinness ideal for women's bodies. Developmental causes can include adolescent "acting out" or fear of adulthood transition. In addition, there appears to be a genetic correlation since AN occurs more commonly in biological relatives of persons who have this disorder.

Precipitating factors are often related to the developmental transitions common in adolescence. The onset of menarche (first menstrual cycle) may be threatening in that it represents maturation or growing up. During this time in development, females gain weight as part of the developmental process, and this gain may cause a decrease in self-esteem. Development of AN could be a way that the adolescent retreats back to childhood so as not to be burdened by maturity and physical concerns. Autonomy and independence struggles during adolescence may be acted out by developing AN. Some adolescents may develop AN because of their ambivalence about adulthood or because of loneliness, isolation, and abandonment they feel.

Symptoms

Most of the physical symptoms associated with AN are secondary to starvation. The **brain** is affected— there is evidence to suggest alterations in brain size, neurotransmitter balance, and hormonal secretion signals originating from the brain. **Neurotransmitters** are the chemicals in the brain that transmit messages from nerve cell to nerve cell. Hormonal secretion signals modulate sex organ activity. Thus, when these signals are not functioning properly, the sex organs are affected. Significant weight loss (and loss in body fat, in particular) inhibits the production of estrogen, which is necessary for menstruation. AN patients experience a loss of menstrual periods, known as amenorrhea. Additionally, other physiologic systems are affected by the starvation. AN patients often suffer from electrolyte (sodium and potassium ion) imbalance and blood cell abnormalities affecting both white and red blood cells. Heart function is also compromised and a person affected with AN may develop congestive heart failure (a chronic weakening of the heart due to work overload), slow heart rate (bradycardia), and abnormal rates and rhythms (arrhythmias). The gastrointestinal tract is also affected, and a person with AN usually exhibits diminished gastric motility (movement) and delayed gastric emptying. These abnormalities may cause symptoms of bloating and constipation. In addition, bone growth is affected by starvation, and over the long term, AN patients can develop osteoporosis, a bone loss disease.

Physically, people with AN can exhibit cold hands and feet, dry skin, hair loss, headaches, fainting, dizziness, and lethargy (loss of energy). Individuals with AN may also develop lanugo (a fine downy hair normally seen in infants) on the face or back. Psychologically, these people may have an inability to concentrate, due to the problems with cognitive functioning caused by starvation. Additionally, they may be irritable, depressed, and socially withdrawn, and they obsessively avoid food. People affected with AN may also suffer from lowered body temperature (hypothermia), and lowered blood pressure, heart rate, glucose and white blood cells (cells that help fight against infection). They may also have a loss of muscle mass.

In order to diagnose AN, a patient's symptoms must meet the symptom criteria established in the professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***, also called the *DSM*. These symptoms include:

- Refusal to maintain normal body weight, resulting in a weight that is less than 85% of the expected weight.
- Even though the affected person is underweight, he or she has an intense fear of gaining weight.
- Distorted body image, obsession with body weight as key factor in self-evaluation, or **denial** of the seriousness of the low body weight.
- Amenorrhea.

Demographics

AN is considered to be a rare illness. The prevalence even in high-risk groups and high-risk situations is approximately 0.5%–1%. Partial disorders (diagnosed when symptoms are present, but do not meet the full criteria as established in the *DSM*) are more commonly seen in psychological practice. The incidence (number of new cases) of AN has increased during the last 50 years due to increased societal concerns regarding body shape, weight, and appearance. Some occupations such as ballet dancing and fashion modeling may predispose persons to develop AN, due to preoccupation with physical appearance. This disorder usually affects women more than men in a ratio of between one to 20 and one to 10.

Diagnosis

Initial assessment usually includes a careful interview and history (clinical evaluation). A weight history, menstrual history, and description of daily food intake are important during initial evaluation. Risk factors and family history are also vital in suspected cases. Laboratory results can reveal anemia (low red blood cell count in the blood), lowered white blood cells, pulse, blood pressure, and body temperature. The decreased temperature in extremities may cause a slight red-purple discoloration in limbs (acrocynanosis). There are two psychological questionnaires that can be administered to aid in **diagnosis**, called the Eating Attitudes Test (EAT) and Eating Disorders Inventory (EDI). The disadvantage of these tests is that they may produce false-positive results, which means that a test result may indicate that the test taker has anorexia, when, actually, s/he does not.

During adolescence, females gain weight as part of the normal developmental process. Typically, anorexia nervosa begins when an adolescent or young woman decides to diet. As weight falls, the intensity and obsession with dieting increases. The incidence of this disorder has increased during the last 50 years due to increased societal concerns regarding body shape, weight, and appearance. *(Richard Hutchings. Photo Researchers, Inc. Reproduced by permission.)*

Treatments

People affected with AN are often in denial, in that they don't see themselves as thin or in need of professional help. Education is important, as is engagement on the part of the patient—a connection from the patient to her treatment, so that she agrees to be actively involved. Engagement is a necessary but difficult task in the treatment of AN. If the affected person's medical condition has deteriorated, **hospitalization** may be required. Initially, treatment objectives are focused on reversing behavioral abnormalities and nutritional deficiencies. Emotional support and reassurance that eating and caloric restoration will not make the person overweight, are essential components during initial treatment sessions. Psychosocial (both psychological and social) issues and family dysfunction are also addressed, which may reduce the risk of relapsing behaviors. (Relapsing behaviors occur when an individual goes back to the old patterns that he or she is trying to eliminate.) At present, there is no standardized psychotherapeutic treatment model to address the multifactorial problems associated with AN. **Cognitive-behavioral therapy** (CBT) may help to improve and modify irrational perceptions and overemphasis of weight gain. Current treatment usually begins with behavioral interventions and should include family therapy (if age appropriate). **Psychodynamic psychotherapy** (also called exploratory **psychotherapy**) is often helpful in the treatment of AN. There are no medications to treat AN. Treatment for this disorder is often long term.

Prognosis

If this disorder is not successfully diagnosed or treated, the affected person may die of malnutrition and multi-organ complications. However, early diagnosis and

appropriate treatment interventions are correlated with a favorable outcome.

Research results concerning outcome of specific AN treatments are inconsistent. Some results, however, have been validated. The prognosis appears to be more positive for persons who are young at onset of the disorder, and/or who have experienced a low number of disorder-related hospitalizations. The prognosis is not as positive for people with long duration illness, very low body weight, and persistent family dysfunction. Additionally, the clinical outcome can be complicated by comorbid, or co-occurring or concurrent, disorders (without any causal relationship to AN) such as depression, anxiety, and substance abuse.

Prevention

A nurturing and healthy family environment during developing years is particularly important. Recognition of the clinical signs with immediate treatment can possibly prevent disorder progression, and, as stated, early diagnosis and treatment are correlated with a favorable outcome.

See also Bibliotherapy

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Antabuse *see* **Disulfiram**

Anti-anxiety drugs and abuse

Definition

Anti-anxiety drugs, or "anxiolytics," are powerful central nervous system (CNS) depressants that can slow normal **brain** function. They are often prescribed to reduce feelings of tension and anxiety, and/or to bring about sleep. Anti-anxiety medications are among the most abused drugs in the United States, obtained both legally, via prescription, and illegally, through the black market. These drugs are also known as sedatives.

Description

The drugs associated with this class of substance-related disorders are the benzodiazepines [such as **diazepam** (Valium), **chlordiazepoxide** (Librium), **alprazolam** (Xanax), **triazolam** (Halcion), and **estazolam** (ProSom)], the **barbiturates** [such as Seconal and pentobarbital (Nembutal)], and barbiturate-like substances including Quaalude, Equanil, and Doriden. Any of these drugs is capable of producing wakeful relief from tension, or sleep, depending upon dosage. Some non-psychiatric uses of anti-anxiety medications include treatment and prevention of **seizures**, muscle relaxants, anesthetics, and drugs to make other anesthetics work more effectively (known as "adjuvants").

Although the types of central nervous system depressants work differently, they all produce a pleasant drowsy or calming effect. If used over a long period of time, tolerance develops, and larger doses are needed to achieve the initial effects. Continued use can lead both to physical dependence when use is reduced or stopped, and to withdrawal symptoms. When combined with each other or other CNS depressants, such as alcohol, the effects are additive.

In addition to the drugs available in the United States by prescription, there are three other drugs that are predominantly central nervous system depressants with significant potential for abuse. These are:

- gamma hydroxybutyrate (GHB)
- flunitrazepam (Rohypnol)
- Ketamine

GHB has been abused in the United States since about 1990, for its euphoric, sedative, and anabolic (bodybuilding) effects. It was widely available over the counter in health food stores until 1992. Bodybuilders used it to aid in reducing percentage of body fat, and to build muscle. Street names for GHB include "Liquid ecstasy," "soap," "Easy lay," and "Georgia home boy."

Rohypnol has been of particular concern during the last few years because of its abuse in date rape. When mixed with alcohol, Rohypnol can incapacitate its victims and prevent them from resisting sexual assault. It can also lead to anterograde **amnesia**, in which individuals cannot remember what they experienced while under the influence. Rohypnol can be lethal when mixed with alcohol and/or other depressants. Rohypnol is not available by prescription in the United States, and it is illegal to import it. Even so, illegal use of Rohypnol started appearing in the United States in the early 1990s, where it became known as "rophies," "roofies," "roach," and "rope."

Ketamine is an anesthetic used predominately by veterinarians to treat animals. It can be injected or snorted. Ketamine goes by the street names of "Special K," or "Vitamin K." At certain doses, ketamine can cause dream-like states and **hallucinations**. It has become particularly common in club and rave (large, all-night dance marathon) settings, and has been used as a date rape drug. At high doses, it can cause **delirium**, amnesia, impaired motor functioning, high blood pressure, and depression. It can also cause potentially fatal respiratory problems.

Causes and symptoms

Causes

Anti-anxiety drugs can be taken orally to bring about a general calming or drowsy effect, usually experienced as pleasant. Abuse of anti-anxiety medication can develop with prolonged use, as tolerance grows relatively quickly. Increasing amounts of the drug are then needed to produce the initial effect. It is possible to become addicted to anti-anxiety drugs even when they are medically prescribed.

A second cause of anti-anxiety drug abuse is the use of these drugs, especially when combined with other drugs, such as cocaine. It is not uncommon for an addict to pair the use of a stimulant, such as cocaine or methamphetamine, with a CNS depressant. This allows the user to feel alert for an extended period of time, and then be able to "come down" from the high, and even fall asleep.

Symptoms

Even when prescribed for medical reasons, an individual taking central nervous system depressants usually feels sleepy and uncoordinated during the first few days of treatment. As the body adjusts to the effects of the drug, these feelings begin to disappear. If the drug is used long term, the body develops tolerance, and increasing doses are needed to obtain the desired effect of general calming or drowsiness.

KEY TERMS

Abuse—Substance abuse is a milder form of addiction than substance dependence. Generally, people who have been diagnosed with substance abuse don't experience the tolerance or withdrawal symptoms— the signs of physiological dependence— that people dependent on a substance experience.

Anxiolytic—A preparation or substance given to relieve anxiety; a tranquilizer.

Barbiturates—A class of medications (including Seconal and Nembutal) that causes sedation and drowsiness. They may be prescribed legally, but may also be used as drugs of abuse.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Dependence—The adaptation of neurons and other physical processes to the use of a drug, followed by withdrawal symptoms when the drug is removed; physiological and/ or psychological addiction.

GHB—GHB, or gamma hydroxybutyrate, is a central nervous system depressant that has been abused in the United States for euphoric, sedative, bodybuilding, and date-rape purposes.

Intoxication—the presence of significant problem behaviors or psychological changes following ingestion of a substance.

Ketamine—An anesthetic used predominately by veterinarians to treat animals that can be used as a date-rape drug.

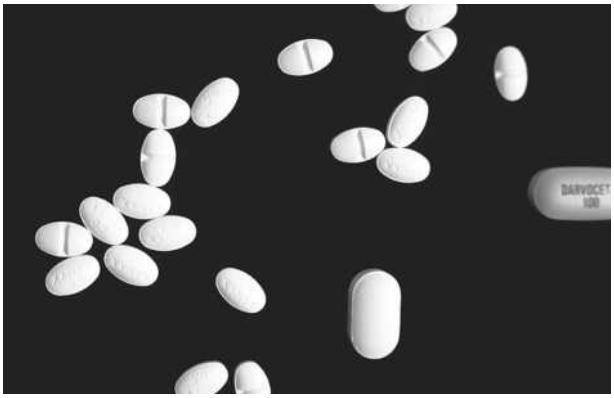
Rohypnol—Rohypnol, or flunitrazepam, is a central nervous system depressant that is not legal in the United States, but is used as a date-rape drug.

Sedative—A medication that induces relaxation and sleep.

Tranquilizer—A medication that induces a feeling of calm and relaxation.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

The use of anti-anxiety drugs can pose extreme danger when taken along with other medications that cause CNS depression, such as prescription pain medicines, some over-the-counter cold and allergy medications, or



Anti-anxiety drugs are powerful central nervous system depressants. They are often prescribed to reduce feelings of tension and anxiety, and/or to induce sleep. Anti-anxiety medications are among the most abused drugs in the United States. *(Thomas Craig/FPG International Corp. Reproduced by permission.)*

alcohol. Use of additional depressants can slow breathing and respiration, and can even lead to death.

Withdrawal from anti-anxiety medications can be dangerous if not done under medical supervision. The safest method of withdrawal involves a gradual reduction of dosage. Abrupt withdrawal from these medications can lead to seizures due to sudden increase in brain activity.

Demographics

Several studies conducted by the National Institute of Drug Abuse, or NIDA, suggest that prescription drug abuse is on the rise in the United States. According to the 1999 *National Household Survey on Drug Abuse*, an estimated 1.6 million Americans first tried prescription pain relievers for non-medical purposes in 1998. Between 1990 and 1998, the number of people who used tranquilizers increased by 132%, and the number of new sedative users increased by 90%. In 1999, an estimated four million people —almost 2% of the population aged 12 and older— by 2001 were using prescription drugs for non-medical purposes. Sedatives and tranquilizers were used by 1.3 million of these people.

In 1999, an estimated four million Americans, about 2% of the population age 12 and older, had used prescription drugs non-medically within the past month. Of these, 1.3 million misused sedatives and tranquilizers. Of particular concern is the growing abuse among older adults, adolescents, and women.

Misuse of prescribed medications may be the most common form of drug abuse among the elderly, according to the NIDA. Older people are given prescriptions approximately three times more often than the general population, and have poorer **compliance** with directions for use.

The *National Household Survey on Drug Abuse* indicates the steepest increase in new users of prescription drugs for non-medical purposes occur in 12- to 17- and 18- to 25-year-olds. Among 12- to 14-year-olds, psychoactive medications, including anti-anxiety drugs, were reportedly among the primary drugs used.

The 1999 *Monitoring the Future Survey*, a yearly survey of drug use and related attitudes conducted among eighth, 10th and 12th graders nationwide, found that for barbiturates, tranquilizers, and narcotics other than heroin, long-term declines in use during the 1980s leveled off in the early 1990s, with modest increases in use starting again in the mid-1990s.

Overall, men and women have approximately equal rates of non-medical use of prescription drugs, with the exception of 12- to 17-year-olds. In this age category, young women are more likely to use psychoactive drugs non-medically. Also, among women and men who use anti-anxiety drugs non-medically, women are almost twice as likely to become addicted.

GHB-related emergency room visits increased from 55 in 1994 to 2,973 in 1999, according to the NIDA. There were 13 reported Rohypnol-related emergency room visits in 1994, versus 634 in 1998. The number decreased to 540 in 1999. Ketamine-related emergency room visits rose from a reported 19 in 1994 to 396 in 1999. Recent use have been reported more frequently among white youth in many major metropolitan areas.

Diagnosis

The manual used by mental health professionals to diagnose mental illnesses, the ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM-IV-TR*, includes specific diagnostic criteria for four types of anti-anxiety medication abuse. These are:

- dependence
- abuse
- intoxication
- withdrawal

Dependence, the more severe form of **addiction**, refers to very significant levels of physiological dependence, with both tolerance and withdrawal symptoms. Abuse, the less severe form of addiction, may still result in risky behavior, such as driving while under the influence. An individual with an abuse disorder may miss work or school, or get into arguments with parents or spouse about substance use. The problem can easily escalate into full-blown dependence.

Intoxication refers to the presence of clinically significant problem behaviors or psychological changes, such as inappropriate sexual or aggressive behavior, mood swings, impaired judgment, or impaired social or work functioning that develop during or shortly after use of an anti-anxiety medication. As with other CNS depressants such as alcohol, these behaviors may be accompanied by slurred speech, unsteady gait, memory or attention problems, poor coordination, and eventually, stupor or coma. Memory impairment is relatively common, especially a kind known as anterograde amnesia that resembles alcoholic blackouts.

Withdrawal is a characteristic syndrome that develops when use of anti-anxiety medication is severely reduced or stopped abruptly. It is similar to abrupt cessation of heavy alcohol use. Symptoms may include increases in heart rate, respiratory rate, blood pressure or body temperature, sweating, hand tremor, **insomnia**, anxiety, nausea, and restlessness. Seizures may occur in perhaps as many as 20-30% of individuals undergoing untreated withdrawal. In the more severe forms of withdrawal, hallucinations and delirium can occur. Withdrawal symptoms are generally the opposite of the acute effects experience by first-time users of the drugs. Length of withdrawal varies depending upon the drug, and may last as few as 10 hours, or as long as three to four weeks. The longer the substance has been taken, and the higher the dosages used, the more likely that withdrawal will be severe.

Treatments

According to the NIDA, successful treatment for anti-anxiety medication addiction needs to incorporate several components. Counseling, particularly cognitive-behavior counseling, focuses on helping addicted individuals identify and change behaviors, attitudes, and beliefs that contributed to their drug usage. Combined with prescribed medications to make withdrawal safer and easier, counseling can help the addicted individual eventually make a full recovery. Often, it takes multiple courses of treatment before full recovery can be achieved. Various levels of care, from outpatient to residential care for up to 18 months, are available, depending upon need. Narcotics Anonymous also offers ongoing recovery support.

Prognosis

The most typical course, according to the *DSM-IV-TR* involves teens or young people in their early 20s who may escalate occasional use of anti-anxiety medications to the point at which they develop problems such as abuse or dependence. This is particularly likely for individuals who also abuse other substances. An initial pattern of use at parties can eventually lead to daily use and high degrees of tolerance.

A second course, observed somewhat less frequently, involves individuals who initially obtain medications by prescription, usually for treatment of anxiety or insomnia. Though the vast majority of people who use medications as prescribed do not go on to develop substance abuse problems, a small minority do. Again, tolerance develops and the need for higher dosages to reach the initial effects occurs. Individuals may justify their continued use on the basis of the original symptoms, but active substance-seeking becomes increasingly part of the picture. Others at higher risk are those with alcohol dependence who might be given prescription anti-anxiety medications to reduce their anxiety or insomnia.

Prevention

Health care professionals play a very important role in preventing and detecting abuse of prescription drugs. Primary care physicians, nurse practitioners and pharmacists can all play a role.

It is estimated by the NIDA that approximately 70% of all Americans visit a health care provider, at least once every two years. Thus, health care providers are in a unique position not only to prescribe medications as appropriate, but also to identify prescription drug abuse when it exists and recommend appropriate treatment for recovery. Screening for substance abuse should be incorporated into routine history taking, or if a patient presents with symptoms associated with problem drug use.

Over time, providers should be alert to any increases in the amount of medication being used, which may be a sign of tolerance. They should also be aware that individuals addicted to prescription medications may engage in "doctor shopping," that is, going from provider to provider in an effort to obtain multiple prescriptions of their abused drug.

Pharmacists can play a role in preventing prescription drug abuse as well. They should provide information and advice about the correct way to take prescribed medications, and be alert to drug interactions. They can also play a role in detecting prescription fraud by noticing suspicious-looking prescription forms.

See also Addiction; Anxiety and anxiety disorders; Anxiety-reduction techniques; Barbiturates; Buspirone; Chlordiazepoxide; Clonazepam; Clorazapine; Cognitive-behavioral therapy; Diazepam; Disease concept of chemical dependency; Estazolam; Flurazepam; Fluvoxamine; Insomnia; Lorazepam; Sedatives and related disorders;

Substance abuse and related disorders; Support groups; Triazolam; Zolpidem

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Barbara S. Sternberg, Ph.D.

Antisocial personality disorder

Definition

Also known as psychopathy, sociopathy or dyssocial personality disorder, antisocial personality disorder (APD) is a **diagnosis** applied to persons who routinely behave with little or no regard for the rights, safety or feelings of others. This pattern of behavior is seen in children or young adolescents and persists into adulthood.

The most recent edition of the *Diagnostic and Statistical Manual of Mental Disorders,* (the fourth edition, text revision or *DSM-IV-TR*) classifies APD as one of four "Cluster B Personality Disorders" along with borderline, histrionic, and narcissistic **personality disorders**.

Description

People diagnosed with APD in prison populations act as if they have no conscience. They move through society as predators, paying little attention to the consequences of their actions. They cannot understand feelings of guilt or remorse. Deceit and manipulation characterize their interpersonal relationships.

Men or women diagnosed with this personality disorder demonstrate few emotions beyond contempt for others. Their lack of empathy is often combined with an inflated sense of self-worth and a superficial charm that tends to mask an inner indifference to the needs or feelings of others. Some studies indicate people with APD can only mimic the emotions associated with committed love relationships and friendships that most people feel naturally.

People reared by parents with antisocial personality disorder or substance abuse disorders are more likely to develop APD than members of the general population. People with the disorder may be homeless, living in poverty, suffering from a concurrent substance abuse disorder, or piling up extensive criminal records, as antisocial personality disorder is associated with low socioeconomic status and urban backgrounds. Highly intelligent individuals with APD, however, may not come to the attention of the criminal justice or mental health care systems and may be underrepresented in diagnostic statistics.

Some legal experts and mental health professionals do not think that APD should be classified as a mental disorder, on the grounds that the classification appears to excuse unethical, illegal, or immoral behavior. Despite these concerns, juries in the United States have consistently demonstrated that they do not regard a diagnosis of APD as exempting a person from prosecution or punishment for crimes committed.

Furthermore, some experts disagree with the American Psychiatric Association's (APA's) categorization of antisocial personality disorder. The APA considers the term *psychopathy* as another, synonymous name for APD. However, some experts make a distinction between psychopathy and APD. Dr. Robert Hare, an authority on psychopathy and the originator of the **Hare Psychopathy Checklist**, claims that all psychopaths have APD but not all individuals diagnosed with APD are psychopaths.

Causes and symptoms

Causes

Studies of adopted children indicate that both genetic and environmental factors influence the development

of APD. Both biological and adopted children of people diagnosed with the disorder have an increased risk of developing it. Children born to parents diagnosed with APD but adopted into other families resemble their biological more than their adoptive parents. The environment of the adoptive home, however, may lower the child's risk of developing APD.

Researchers have linked antisocial personality disorder to childhood physical or sexual abuse; neurological disorders (which are often undiagnosed); and low IQ. But, as with other personality disorders, no one has identified any specific cause or causes of antisocial personality disorder. Persons diagnosed with APD also have an increased incidence of somatization and substance-related disorders.

DSM-IV-TR adds that persons who show signs of **conduct disorder** with accompanying **attention-deficit/hyperactivity disorder** before the age of ten have a greater chance of being diagnosed with APD as adults than do other children. The manual notes that abuse or **neglect** combined with erratic parenting or inconsistent discipline appears to increase the risk that a child diagnosed with conduct disorder will develop APD as an adult.

Symptoms

The central characteristic of antisocial personality disorder is an extreme disregard for the rights of other people. Individuals with APD lie and cheat to gain money or power. Their disregard for authority often leads to arrest and imprisonment. Because they have little regard for others and may act impulsively, they are frequently involved in fights. They show loyalty to few if any other people and are likely to seek power over others in order to satisfy sexual desires or economic needs.

People with APD often become effective "con artists." Those with well-developed verbal abilities can often charm and fool their victims, including unsuspecting or inexperienced therapists. People with APD have no respect for what others regard as societal norms or legal constraints. They may quit jobs on short notice, move to another city, or end relationships without warning and without what others would consider good reason. Criminal activities typically include theft, selling illegal drugs and check fraud. Because persons with antisocial personality disorder make "looking out for number one" their highest priority, they are quick to exploit others. They commonly rationalize these actions by dismissing their victims as weak, stupid or unwary.

KEY TERMS

Attention-deficit/hyperactivity disorder—A learning and behavioral disorder characterized by difficulty in sustaining attention, impulsive behavior, and excessive activity.

Conduct disorder—A behavioral and emotional disorder of childhood and adolescence in which children display physical aggression and infringe on or violate the rights of others. Youths diagnosed with conduct disorder may set fires, exhibit cruelty toward animals or other children, sexually assault others, or lie and steal for personal gain.

Psychopathy—A psychological syndrome that includes lack of a conscience or sense of guilt, lack of empathy, egocentricity, pathological lying, repeated violations of social norms, disregard of the law, shallow emotions and a history of victimizing others.

Somatization disorder—A type of mental disorder in which the patient suffers from physical complaints that serve as coping strategies for emotional distress.

Substance abuse disorder—Disorder that is characterized by: an individual's need for more of a drug or alcohol than intended, an inability to stop using by choice, and an ongoing difficulty in recovering from the effects of the substance.

Demographics

APD is estimated to affect 3% of males and 1% of females in the general United States population. Mental health professionals may diagnose 3%–30% of the population in clinical settings as having the disorder. The percentages may be even higher among prison inmates or persons in treatment for substance abuse. By some estimates, three-quarters of the prison population may meet the diagnostic criteria for APD.

Diagnosis

The diagnosis of antisocial personality disorder is usually based on a combination of a careful medical as well as psychiatric history and an interview with the patient. The doctor will look for recurrent or repetitive patterns of antisocial behavior. He or she may use a diagnostic questionnaire for APD, such as the Hare Psychopathy Checklist, if the patient's history suggests the diagnosis. A person aged 18 years or older with a

childhood history of disregard for the rights of others can be diagnosed as having APD if he or she gives evidence of three of the following seven behaviors associated with disregard for others:

- Fails to conform to social norms, as indicated by frequently performing illegal acts or pursuing illegal occupations.
- Deceives and manipulates others for selfish reasons, often in order to obtain money, sex, drugs or power. This behavior may involve repeated lying, conning or the use of false names.
- Fails to plan ahead or displays impulsive behavior, as indicated by a long succession of short-term jobs or frequent changes of address.
- Engages in repeated fights or assaults as a consequence of irritability and aggressiveness.
- Exhibits reckless disregard for safety of self or others.
- Shows a consistent pattern of irresponsible behavior, including failure to find and keep a job for a sustained length of time and refusal to pay bills or honor debts.
- Shows no evidence of sadness, regret or remorse for actions that have hurt others.

In order to meet *DSM-IV-TR* criteria for APD, a person must also have had some symptoms of conduct disorder before age 15. An adult 18 years or older who does not meet all the criteria for APD may be given a diagnosis of conduct disorder.

Antisocial behavior may appear in other mental disorders as well as in APD. These conditions must be distinguished from true APD. For instance, it is not uncommon for a person with a substance abuse disorder to lie to others in order to obtain money for drugs or alcohol. But unless indications of antisocial behavior were present during the person's childhood, he or she would not be diagnosed with antisocial personality disorder. People who meet the criteria for a substance abuse disorder as well as APD would be given a **dual diagnosis**.

Treatments

Antisocial personality disorder is highly unresponsive to any form of treatment, in part because persons with APD rarely seek treatment voluntarily. If they do seek help, it is usually in an attempt to find relief from depression or other forms of emotional distress. Although there are medications that are effective in treating some of the symptoms of the disorder, noncompliance with medication regimens or abuse of the drugs prevents the widespread use of these medications. The most successful treatment programs for APD are long-term structured residential settings in which the patient systematically earns privileges as he or she modifies behavior. In other words, if a person diagnosed with APD is placed in an environment in which they cannot victimize others, their behavior may improve. It is unlikely, however, that they would maintain good behavior if they left the disciplined environment.

If some form of individual **psychotherapy** is provided along with **behavior modification** techniques, the therapist's primary task is to establish a relationship with the patient, who has usually had very few healthy relationships in his or her life and is unable to trust others. The patient should be given the opportunity to establish positive relationships with as many people as possible and be encouraged to join **self-help groups** or prosocial reform organizations.

Unfortunately, these approaches are rarely if ever effective. Many persons with APD use therapy sessions to learn how to turn "the system" to their advantage. Their pervasive pattern of manipulation and deceit extends to all aspects of their life, including therapy. Generally, their behavior must be controlled in a setting where they know they have no chance of getting around the rules.

Prognosis

APD usually follows a chronic and unremitting course from childhood or early adolescence into adult life. The impulsiveness that characterizes the disorder often leads to a jail sentence or an early death through accident, homicide or **suicide**. There is some evidence that the worst behaviors that define APD diminish by midlife; the more overtly aggressive symptoms of the disorder occur less frequently in older patients. This improvement is especially true of criminal behavior but may apply to other antisocial acts as well.

Prevention

Measures intended to prevent antisocial personality disorder must begin with interventions in early childhood, before youths are at risk for developing conduct disorder. Preventive strategies include education for parenthood and other programs intended to lower the incidence of child abuse; Big Brother/Big Sister and similar mentoring programs to provide children at risk with adult role models of responsible and prosocial behavior; and further research into the genetic factors involved in APD.

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Anxiety and anxiety disorders

Definition

Anxiety is an unpleasant emotion triggered by anticipation of future events, memories of past events, or ruminations about the self.

Description

Stimulated by real or imagined dangers, anxiety afflicts people of all ages and social backgrounds. When the anxiety results from irrational fears, it can disrupt or disable normal life. Some researchers believe anxiety is synonymous with fear, occurring in varying degrees and in situations in which people feel threatened by some danger. Others describe anxiety as an unpleasant emotion caused by unidentifiable dangers or dangers that, in reality, pose no threat. Unlike fear, which is caused by realistic, known dangers, anxiety can be more difficult to identify and to alleviate.

Rather than attempting to formulate a strict definition of anxiety, most psychologists simply make the distinction between normal anxiety and neurotic anxiety, or anxiety disorders. Normal (sometimes called objective) anxiety occurs when people react appropriately to the situation causing the anxiety. For example, most people feel anxious on the first day at a new job for any number of reasons. They are uncertain how they will be received by coworkers, they may be unfamiliar with their duties, or they may be unsure they made the correct decision in taking the job. Despite these feelings and any accompanying physiological responses, they carry on and eventually adapt. In contrast, anxiety that is characteristic of anxiety disorders is disproportionately intense. Anxious feelings interfere with a person's ability to carry out normal or desired activities. Many people experience stage fright—the fear of speaking in public in front of large groups of people. There is little, if any, real danger posed by either situation, yet each can stimulate intense feelings of anxiety that can affect or derail a person's desires or obligations. Sigmund Freud described neurotic anxiety as a danger signal. In his id-ego-superego scheme of human behavior, anxiety occurs when unconscious sexual or aggressive tendencies conflict with physical or moral limitations.

According to a standard manual for mental health clinicians, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revised (also known as the *DSM-IV-TR*), the following disorders are considered anxiety disorders:

- **Panic disorder** without agoraphobia—A person with this disorder suffers from recurrent panic attacks and worries about experiencing more attacks, but **agoraphobia** is not present. Panic attacks are sudden attacks of intense fear or apprehension during which the sufferer may experience shortness of breath, increased heart rate, choking, and/or a fear of losing control. Agoraphobia is anxiety about places or situations from which escape might be difficult, or in which help might not be available.
- Panic disorder with agoraphobia—A person with this disorder also experiences recurrent panic attacks but also has agoraphobia. The anxiety about certain places or situations may lead to avoidance of those places or situations.
- Agoraphobia without history of panic disorder—The person with this disorder suffers from agoraphobia and experiences panic-like symptoms but does not experience recurring panic attacks.
- **Specific phobias**—A person diagnosed with a specific phobia suffers from extreme anxiety when he or she is exposed to a particular object or situation. The feared stimuli may include: particular animals (dogs, spiders, snakes, etc.), situations (crossing bridges, driving through tunnels), storms, heights, and many others.
- Social phobia—A person with **social phobia** fears social situations or situations in which the individual is expected to perform. These situations may include eating in public or speaking in public, for example.

- **Obsessive-compulsive disorder**—A person with this disorder feels anxiety in the presence of a certain stimulus or situation, and feels compelled to perform an act (a **compulsion**) to neutralize the anxiety. For example, upon touching a doorknob, a person may feel compelled to wash his or her hands four times, or more.
- **Post-traumatic stress disorder**—This disorder may be diagnosed after a person has experienced a traumatic event, and long after the event, the person still mentally re-experiences the event along with the same feelings of anxiety that the original event produced.
- **Acute stress disorder**— Disorder with similar symptoms to post-traumatic stress disorder, but is experienced immediately after the traumatic event. If this disorder persists longer than one month, the **diagnosis** may be changed to post-traumatic stress disorder.
- **Generalized anxiety disorder**—A person who has experienced six months or more of persistent and excessive worry and anxiety may receive this diagnosis.
- Anxiety due to a general medical condition—Anxiety that the clinician deems is caused by a medical condition.
- Substance-induced anxiety disorder—Symptoms of anxiety that are caused by a drug, a medication, or a toxin.
- Anxiety disorder not otherwise specified—This diagnosis may be given when a patient's symptoms do not meet the exact criteria for each of the above disorders as specified by *DSM-IV-TR*.

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Anxiety disorder *see* **Generalized anxiety disorder**

Anxiety reduction techniques

Definition

Anxiety reduction techniques are skills that are taught by a therapist to help an individual overcome anxiety, **stress**, and tension. Anxiety can be experienced in a variety of ways including tension, worry, and nervousness, and can occur in thoughts or experienced as bodily senations. The techniques to reduce anxiety can include relaxation, visualization and imagery, diaphragmatic breathing, stress inoculation, and **meditation**.

Relaxation or progressive relaxation

This anxiety reduction technique is based on the premise that anxiety and stress are associated with muscle tension. When one achieves deep muscle relaxation, muscle tension is reduced, and this relaxed state is incompatible with anxiety.

Visualization and imagery

This anxiety reduction technique aids the person in making a mental image of what he or she wants to accomplish. For example, a person might wish to release worries or concerns, or create a relaxing image to escape momentarily from a stressful event.

Diaphragmatic breathing

This technique involves teaching a person to breathe sufficient amounts of air to help the person's blood be purified properly and filled with oxygen. In this technique, the individual breathes deeply from the diaphragm, which is located low in the chest, near the abdomen.

Stress inoculation

Self-talk, or the things that people tell themselves about stressful situations, can be habitual. For example, a person may take an ordinary event and automatically magnify its importance. Stress inoculation training is a type of therapy that trains clients to cope with anxiety and stressful situations by learning more functional patterns of self-talk.

Meditation

In this anxiety reduction technique, a person is trained to focus his or her attention on one thing at a time.

Purpose

The goal of learning and implementing anxiety reduction techniques is to help reduce the intensity of anxiety that a person feels. These techniques are also helpful in teaching people how to relax and manage stress. Many of the techniques are used in combination with each other. For example, a person may be taught diaphragmatic breathing while also engaging in relax-

ation techniques, a visualization and imagery exercise, and/or meditation.

Relaxation or progressive relaxation

Relaxation has been used to help women during childbirth and people with chronic pain. Relaxation has also been used to treat muscle tension, muscle spasms, neck and back pain, and to decrease perspiration and respiratory rates. Furthermore, relaxation can help with **fatigue**, depression, **insomnia**, irritable bowel syndrome, high blood pressure, mild phobias, and **stuttering**.

Visualization and imagery

Visualization and imagery techniques have been helpful in treating general or specific anxiety, headaches, muscle tension and spasms, reducing or eliminating pain, and in the recovery from illnesses and injuries. Visualization and imagery techniques have also been used by athletes to help them achieve peak performance.

Diaphragmatic breathing

Diaphragmatic breathing has been found to help people reduce anxiety, depression, irritability, muscle tension, circulation, and fatigue.

Stress inoculation

Stress inoculation has been helpful in reducing interpersonal and general anxiety. For example, these techniques may be used when a person has an upcoming job interview, speech, or test. Stress inoculation has also been used to treat phobias, fear of heights, and chronic anger problems.

Meditation

Meditation has been used to treat and prevent high blood pressure, heart disease, strokes, migraine headaches, immunization diseases, obsessive thinking, attention problems, anxiety, depression, and anger difficulties.

Description

These various techniques are often practiced and demonstrated in therapy sessions with a trained professional. In addition, the person learning the techniques would need to continue to practice them on a regular basis, outside of the therapy sessions.

Relaxation or progressive relaxation

In progressive relaxation, a person is instructed to tighten and then relax various muscles. A person either lies down or sits in a chair with his or her head supported. Each muscle group (such as face muscles, arm muscles, leg muscles, etc.) is tensed for five to seven seconds and then relaxed for 20 to 30 seconds. This helps the person recognize the feeling of tense and relaxed muscles. This entire procedure is repeated one to five times, and usually starts with the face muscles and moves downward to the foot muscles. When relaxation is used with chronic pain and childbirth, the techniques focus the person's attention on breathing and relaxing muscles as a distraction from the pain. For mastery, relaxation techniques are typically practiced every day for one to two weeks. A person may engage in these techniques anywhere from 15 minutes to an hour per session. Sometimes, a person will record and replay instructions on tightening and relaxing various muscle groups until the person becomes familiar with the muscle groups and establishes a routine in which he or she is comfortable.

Visualization and imagery

The basic premise behind visualization and imagery is that one's thoughts become reality. For example, if one thinks anxious thoughts, then he or she will become tense. The principles behind visualization and imagery maintain that a person can use his or her imagination to be persuaded to feel a certain way or do anything that is physically possible to do. There are three basic types of visualization: programmed, receptive, and guided visualization.

In programmed visualization, the person creates a vivid image including sight, taste, sound, and smell. The person then imagines a goal he or she wants to attain or some type of healing that is desired. In the visualization, the goal is achieved, or the healing occurs.

An idea underlying both receptive visualization and guided visualization is that the person is seeking an answer to a life question or resolution to an issue, and the answer or resolution is within the person, but is buried or inaccessible to him or her because of fear, doubt, or anxiety. These techniques are similar to dream interpretation and free association techniques used in **psychoanalysis** or psychodynamic therapy. For example, an individual wonders whether he should remain in his current position. A proponent of these techniques would maintain that "deep down," below the level of conscious thought or subconsciously, the man knows what he really wants to do, but he is not allowing himself to listen to his desires or to act—he is blocking the message his subconsciousness is sending him. The goal of these techniques is to enable the person to relax and focus enough to receive that message, so that the person can do what needs to be done. In receptive visualization, the person creates a peaceful scene in his or her mind. For example, the person might imagine being on a beach. After the

image is formed, the person asks a question and waits for the answer. To continue the example above, the man imagines a beach, and he asks himself the question, "Should I leave my job?" He continues to relax and remain in the scene, and he may "hear" an answer blowing in the breeze or "see" a boat sailing away, which may be symbolic of his wish to leave his job.

In guided visualization, the person creates a very vivid image, as in programmed visualization, but omits some important elements. The person then waits for the subconscious to supply the missing pieces. For example, a computer programmer may wonder if she should stay in her present job or return to school for an advanced degree. In engaging in guided visualization, she may visualize her cubicle at work, the pictures on her desk, the feel of her desk chair, the sounds of people outside her cubicle typing and talking, but she will omit an element from the scene. In this case, she may omit her computer. She will then wait to see what her subconscious uses to replace her computer. This woman may find in her visualization that her computer has been replaced by books, which may represent her desire to return to school.

Visualization and imagery exercises work best when a person is relaxed. Visualization and imagery exercises are typically practiced two to three times a day for 10 to 20 minutes at a time. How quickly a person will see results can vary. Many times people report immediate symptom relief. However, the goals a person sets for him or herself, the power of a person's imagination, and the willingness to practice can all influence how rapidly benefits can be obtained. Some people find it helpful to tape record and replay detailed descriptions of what they want to visualize or imagine.

Diaphragmatic breathing

Diaphragmatic breathing can typically be learned in minutes; however, the benefits may not be recognized until after several months of persistent practice. When breathing from the diaphragm, clients are often told to lie down on a rug or blanket, with their legs slightly apart, arms to the sides, not touching the body, and eyes closed. Attention is brought to the client's breathing by placing one hand on the chest and the other hand on the abdomen area. The client is then instructed to breathe through the nose and exhale out the mouth. Each time the client breathes in, he or she should try to breathe deeper. This should be practiced for a minimum of five minutes once or twice a day. Over a few weeks of practice, the time period engaged in diaphragmatic breathing should be increased to 20 minutes and the activity can be performed while lying down, sitting, or standing.

Stress inoculation

As people go about their daily lives, they often have thoughts in which they are talking to themselves. Stress inoculation involves this self-talk in helping clients decrease their anxiety and stress. Stress inoculation therapy works on the basis of turning the client's own thought patterns into a "vaccine" against stress-induced anxiety. The first step is to develop a list of stressful situations and arrange them from least to most stressful. Once anxiety-producing situations are identified, the client is taught to curb the anxiety-provoking thoughts and replace them with more positive coping thoughts. Once these new thoughts are learned, they can be tried out in real situations. The time it takes to replace old habitual thoughts with new thoughts can vary depending on the amount of practice and commitment to make this change.

Meditation

There are various forms of meditation. Depending on the type used, the person focuses his or her attention in slightly different ways. For example, Zen meditation focuses on breathing, whereas in transcendental meditation, the person makes a sound or says a mantra selected to keep all other images and problems from intruding on his or her thoughts. With practice, a person can reach a meditative state and obtain its benefits within a few minutes.

Aftercare

After a person has learned and practiced anxiety reduction techniques, he or she may need additional instruction from a trained professional. Having a trained professional review these techniques with a person can help reinforce what the person has already learned and been practicing. Furthermore, the person may identify aspects of the techniques that he or she is doing incorrectly, areas that need more attention or focus, and alternative methods of engaging in the techniques.

Risks

There are minimal risks associated with these techniques, but some physical problems have occurred. For example, precautions should be taken when doing progressive relaxation and tensing the neck and back. Excessive tightening can create muscle or spinal damage. Additionally, tightening various muscle groups, such as the toes or feet, could result in muscle cramps. If physical problems occur, such as difficulty taking deep breaths, unusual muscle pain, or an increased level of anxiety, then the individual should seek assistance from a physician.

Normal results

In general, after engaging in these anxiety reduction techniques, many people report an increased sense of well-being and relaxation. People have a greater sense of control, and confidence in their coping abilities. This results in a decreased need to fear or avoid stressful situations.

Relaxation or progressive relaxation

Progressive relaxation can be useful in reducing muscle tension. Engaging in relaxation may help to improve a person's energy level, depression, and anxiety, as well as a person's ability to retrieve information from memory.

Visualization and imagery

By engaging in the positive thinking often associated with visualization and imagery, a person can create a clearer image of what he or she wants to accomplish. By repeating the image again and again, the person comes to expect what he or she wants will occur. As a result, the person will often begin to act in a way more consistent with accomplishing the goal.

Diaphragmatic breathing

Sufficient amounts of air reach the lungs, which purifies and oxygenates the blood. Waste products in the blood are removed, and organs and tissues become nourished.

Stress inoculation

A person will have more realistic views of stressful and anxiety-producing situations in his or her life. The person will be able to relax away tension by effectively thinking useful coping thoughts rather than negative interpretations of situations.

Meditation

As people learn to meditate, they often discover that they have some control over the thoughts that come to their minds, as opposed to feeling as though thoughts "pop" into their heads. Many people begin to recognize dysfunctional patterns of thought and perceptions that have influenced their lives. Additionally, many people report a greater ability to manage their emotions and gain a greater sense of stability. When a person meditates, he or she often suppresses the activity of the sympathetic nervous system, the part of the nervous system that activates the body for emergencies and activities. Meditation also lowers a person's metabolism, heart, and breathing rates. Additionally, meditation decreases the chemical in the body often associated with stress.

Abnormal results

Once a person begins to implement these anxiety reduction techniques effectively, he or she may discover old or hidden psychological pain. The person may feel angry, frightened, or depressed, and it may be beneficial for him or her to talk to a friend, mental health professional, or meditation teacher.

Some individuals have difficulty with various aspects of the different techniques. For example, people may feel restless when first learning how to meditate, or they may feel as though a thousand thoughts are running through their minds. However, with practice and assistance from a trained professional, these difficulties will subside. People who feel frustrated or discouraged may simply need to find ways to make the practice of these techniques more comfortable. As is the case with many other skills, effectively reducing anxiety with these techniques requires patience and practice. If an individual does not consistently practice these techniques, the benefits will probably not be obtained.

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American Psychological Association. 750 1st St. NE, Washington, D.C. 20002. (202) 336-5500. <http://www.apa.org>.

Anxiety Disorders Association of America, Inc. 11900 Parklawn Drive, Suite 100, Rockville, MD 20852. (301) 231-9350. <http://www.adaa.org>.

The National Institute of Mental Health, 5600 Fischers Lane, Room 15C05, Rockville, MD 20857. (301) 443-4513. <http://www.nimh.nih.gov/>.

The National Mental Health Association. 1201 Prince Street, Alexandria, VA 22314-2971.

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Apathy

Definition

Apathy can be defined as an absence or suppression of emotion, feeling, concern or passion. Further, apathy is an indifference to things generally found to be exciting or moving.

Description

A strong connection exists between apathy and mental disorders. Apathy is one of the hallmark symptoms of **schizophrenia**. Many people with schizophrenia express little interest in the events surrounding them. Apathy can also occur in **depression and depressive disorders**. For example, people who are depressed and have **major depressive disorder** or **dysthymic disorder** often feel numb to events occurring around them, and do not derive pleasure from experiences that they once found enjoyable.

The World Health Organization (WHO) defines health as an optimal state of being that maximizes one's potential for physical, mental, emotional and spiritual growth. It does not confine health to physical parameters or measures. Passion, interest and action are needed for optimal mental and emotional health. Persons who are apathetic would seem to fall short of the WHO definition of health.

All people may experience periods of apathy. Disappointment and dejection are elements of life, and apathy is a normal way for humans to cope with such stresses— to be able to "shrug off" disappointments enables people to move forward and try other activities and achieve new goals. When the stresses pass, the apparent apathy also disappears. A period of apathy can also be viewed as a normal and transient phase through which many adolescents pass.

It is important to note, however, that long-term apathy and detachment are not normal.

Treatment

Transient apathy can be overcome. Friends and professionals may be able to assist individuals to develop an interest in their surroundings. Attitude is important. Persons who desire to overcome apathy have much higher odds of succeeding than do persons lacking a positive attitude.

Other than support, no specific treatment is needed for apathy associated with adolescence, unless other, more troubling disorders are also present.

The treatment of more persistent apathy (in a depressive disorder, for example), or the apathy that is characteristic of schizophrenia, may respond to treatment for the primary disorder.

DEPRESSION. For depressive disorders, a number of antidepressants may be effective, including tricyclic antidepressants, monoamine oxidase inhibitors (MAOIs) and selective serotonin reuptake inhibitors (SSRIs). The tricyclic antidepressants include **amitriptyline** (Elavil), **imipramine** (Tofranil), and **nortriptyline** (Aventyl, Pamelor). MAOIs include **tranylcypromine** (Parnate) and **phenelzine** (Nardil). The most commonly prescribed SSRIs are **fluoxetine** (Prozac), **sertraline** (Zoloft), **paroxetine** (Paxil), **fluvoxamine** (Luvox), and **citalopram** (Celexa).

SCHIZOPHRENIA. For schizophrenia, the primary goal is to treat the more prominent symptoms (**positive symptoms**) of the disorder, such as the thought disorder and **hallucinations** that patients experience. Atypical antipsychotics are newer medications introduced in the 1990s that have been found to be effective for the treatment of schizophrenia. These medications include **clozapine** (Clozaril), **risperidone** (Risperdal), **quetiapine** (Seroquel), **ziprasidone** (Geodon), and **olanzapine** (Zyprexa). These newer drugs are more effective in treating the **negative symptoms** of schizophrenia (such as apathy) and have fewer side effects than the older antipsychotics. Most atypical antipsychotics, however, do have weight gain as a side effect; and patients taking clozapine must have their blood monitored periodically for signs of agranulocytosis, or a drop in the number of white blood cells.

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American Psychological Association. 750 First Street NW, Washington, DC, 20002-4242. Phone: (800) 374-2721 or (202) 336-5500, Web site: <http://www.apa.org/>.

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KEY TERMS

Appetite suppressants—Medications that assist in weight loss by reducing appetite or increasing the sensation of fullness

Dexfenfluramine (Redux)—A prescription appetite suppressant for weight loss that was withdrawn from the market due to unacceptable health risks.

Diethylpropion (Tenuate, Tenuate dospan)—A prescription appetite suppressant currently on the market for weight loss.

"Fen/phen"—The commonly used name for a combination of fenfluramine and phentermine that is no longer available due to the withdrawal of fenfluramine from the market.

Fenfluramine (Pondimin)—A prescription appetite suppressant for weight loss that was withdrawn from the market due to unacceptable health risks.

Insulin resistance—The body's inability to utilize blood sugar, at times leading to diabetes

Mazindol (Sanorex, Mazanor)—A prescription medication for weight loss currently on the market.

Orlistat (Xenical)—A prescription medication for weight loss currently on the market.

Phendimetrazine (Bontril, Plegine, Prelu-2, X-Trozine)—A prescription appetite suppressant for weight loss currently on the market.

Phentermine (Adipex-P, Fastin, Ionamin, Obytrim)—A prescription appetite suppressant currently on the market for weight loss.

Primary pulmonary hypertension (PPH)—A rare but potentially fatal disorder that affects the blood vessels in the lungs

Triglycerides—Fats in the blood

Appetite suppressants

Definition

Appetite-suppressant medications are drugs that promote weight loss by decreasing appetite or increasing the sensation of fullness.

Description

Obesity is a disease that affects millions of American adults, adolescents, and children, posing serious health risks. Medical professionals generally consider obesity to be a chronic illness requiring life-long treatment and management. It is often grouped with other chronic conditions, such as high blood pressure and diabetes, as a condition that can be controlled but not cured. One is considered obese if 20% over ideal body weight, according to standard height-weight charts, or if one's Body Mass Index, or BMI, (a ratio of height to weight, indicaating the amount of fat tissue in the body) exceeds 30%.

The most important strategies for managing obesity are not medications but rather, a healthy diet coupled with moderate exercise. As in other chronic conditions, the use of prescription medications may assist in managing the condition for some individuals but it is never the sole treatment for obesity, nor is it ever considered a cure.

The class of medications used most often for weight loss are commonly referred to as "appetite suppressants."

These medications promote weight loss by helping to diminish appetite, and/or by increasing the subjective feeling of fullness. They work by increasing serotonin or catecholamines, two **neurotransmitters** (chemicals) in the **brain** that affect both mood and appetite.

Several prescription medications are currently approved for treatment of obesity. In general, the effects of these medications are modest, leading to an average initial weight loss of between 5 and 22 pounds; though studies show that weight returns after cessation of the drugs. There is considerable individual difference in response to these medications; some people experience greater weight loss than others. The goal of prescribing weight loss medication is to help the medically at-risk obese patient "jump-start" their weight loss effort and lose 10% or more of their starting body weight. When this can be accomplished, it usually leads to a reduction in risk for obesity-related illnesses, such as high blood pressure, heart disease and diabetes. Weight loss tends to be greatest during the first few weeks or months of treatment, leveling off after about six months. Research suggests that if a patient does not lose at least four pounds during the first four weeks on a particular medication, that medication is unlikely to be effective over the long run. Few studies have addressed safety or effectiveness of medications taken for more than a few months at a time. Little data exists on the long-term effectiveness of the drugs.

All but two of the prescription appetite suppressants in the United States have been approved by the U.S. Food and Drug Administration (FDA), for short-term use only. Short-term use generally means a few weeks or months at the longest. One appetite suppressant medication was approved for longer-term use within the past decade, but that drug, dexfenfluramine (Redux) was withdrawn from the market because of unacceptable risks associated with its use.

Another medication was approved within the past few years for longer-term use, up to a year and possibly longer, in significantly obese patients. This drug, an appetite suppressant, is called sibutramine (Meridia). Individuals with a history of heart disease, irregular heartbeat, high blood pressure, or history of **stroke** should not take sibutramine. All patients taking this medication should have their blood pressure monitored regularly.

A relatively new drug, orlistat (Xenical), was approved in 1999 by the FDA for at least a year or longer, as well. Orlistat is not an appetite suppressant, but rather, a member of a new class of anti-obesity drugs known as "lipase inhibitors." These medications work by preventing enzymes in the gastrointestinal tract from breaking down dietary fats into smaller molecules that can be absorbed by the body. The result is that fat absorbed from food is decreased by about 30%. This effectively reduces the calories absorbed by the body by 30%, aiding in weight loss.

While the FDA regulates how a medication can be advertised or promoted by the manufacturer, these regulations do not constrain physicians from prescribing them as they believe appropriate. This practice of prescribing medications for conditions other than those for which they were approved, or at different dosages, or for different lengths of time, is known as "off-label" use. Many of the prescription medications available for weight management are used in an "off-label" manner.

Most of the side effects of prescription medications for weight loss are mild; but some very serious complications have been reported in recent years. They were so serious that two medications were voluntarily removed from the market by the manufacturers in 1997.These two medications, fenfluramine (Pondimin), and dexfenfluramine (Redux), were shown to be associated with a rare but very serious and potentially fatal disorder known as primary pulmonary hypertension (PPH), a disease of the lungs. Forty-five percent of patients with PPH die within four years of **diagnosis**.

Medications for weight loss

Prescription medications

Prescription medications currently prescribed for weight loss include:

- Generic name: Diethylpropion (Trade names: Tenuate, Tenuate dospan)
- Generic name: Mazindole (Trade name: Sanorex)
- Generic name: Orlistat (Trade name: Xenical)
- Generic name: Phendimetrazine (Trade names: Bontril, Plegine, Prelu-2, X-Troxine)
- Generic name: Phentermine (Trade name: Adipex-P, Fastin, Ionamin, Oby-trim)
- Generic name: Sibutramine (Trade name: Meridia)

Some antidepressant medications have been studied for use as possible appetite depressants, because they frequently depress appetite in the early weeks and months of use. Research indicates, however, that while individuals may lose weight initially during antidepressant treatment, a tendency to lose only modest amounts of weight arises after six months. Furthermore, most patients who lose weight early in antidepressant medication treatment tend to regain the weight while still using the medication.

Amphetamines and similar medications were frequently prescribed in the United States, during the 1960s

and 70s, as appetite suppressants. However, because of their addictive potential, they are not prescribed today for weight control, except by a remainder of "diet doctors" who defy political correctness and continue to distribute them.

SINGLE DRUG TREATMENT. The medications listed above are currently used to treat obesity. In general, these medications are modestly effective, especially when used in conjunction with a healthy diet and moderate exercise. Average weight losses between five and 22 pounds can be expected beyond those seen with non-drug obesity treatments, when only a low-calorie diet and exercise regimen are followed. There is considerable individual variation in response to weight-loss medications; some people experience more weight loss than others.

COMBINED DRUG TREATMENT. Combined drug treatment using fenfluramine and phentermine ("fen/phen") is no longer available due to the withdrawal of fenfluramine from the market. There is little information about the safety or effectiveness of other prescription drug combinations for weight loss. Until further research is conducted on safety or effectiveness, using combinations of medications for weight loss is not advised unless a patient is participating in a research study.

POTENTIAL BENEFITS OF APPETITE SUPPRESSANT TREATMENT. Short-term use of appetite suppressant medications has been shown to modestly reduce health risks for obese individuals. Studies have found that these medications can lower blood pressure, blood cholesterol, blood fats (triglycerides), and decrease insulin resistance (the body's ability to utilize blood sugar). Long-term studies need to be conducted to determine if weight loss assisted by appetite suppressant medications can improve health long-term.

POTENTIAL RISKS OF APPETITE SUPPRESSANT TREATMENT. All prescription medications used to treat obesity, with the exception of orlistat, are controlled substances. This means that doctors need to follow rigid guidelines when prescribing them. Although abuse and dependence are uncommon with non-amphetamine appetite suppressant medications, doctors need to exercise caution when prescribing them, especially for patients with a history of alcohol or drug abuse.

DEVELOPMENT OF TOLERANCE. Studies of appetite suppressant medications indicate that an individual's weight tends to level off after four to six months of treatment. While some patients and doctors may be concerned that this indicates growing tolerance to the medications, the leveling off may indicate that the medication has reached its limit of effectiveness. Current research is not clear regarding whether weight gained with continued medication is due to drug tolerance, or to reduced effectiveness of the medication over time.

Dexfenfluramine (Redux) was withdrawn from the market. The medication was shown to be associated with a rare but very serious and potentially fatal disorder known as primary pulmonary hypertension (PPH), a disease of the lungs. *(Miller/Custom Medical Stock Photo. Reproduced by permission.)*

SIDE EFFECTS. Because obesity is a condition affecting millions of Americans, many of whom are basically healthy, the side effects of using powerful medications such as appetite suppressants are of great concern. Most side effects of these medications are mild and diminish as treatment continues. Rarely, serious and even fatal outcomes have been reported. The FDA-approved appetite suppressant medications that affect serotonin (fenfluramine and dexfenfluramine) have been withdrawn from the market. Medications that affect catecholamine levels (such as phentermine, dietylpropion, and mazindol) may cause symptoms of sleeplessness, nervousness, and euphoria.

Primary pulmonary hypertension (PPH) is a rare but potentially fatal disease that affects the blood vessels in the lungs and causes death within four years in 45% of its victims. Patients who use the appetite suppressant medications that are prescribed for a use of three months are at increased risk of developing this condition if used longer. Estimates are that between 1 in 22,000 and 1 in 44,000 individuals will develop the disorder each year. While the risk of developing PPH is very small, doctors and patients should be aware of this potentially deadly complication when they consider the risks and benefits of using appetite suppressant medications for long-term treatment of obesity. Patients taking appetite suppressants should contact their doctors if they experience shortness of breath, chest pain, faintness, or swelling in the lower legs and ankles. The vast majority of cases of PPH related to appetite suppressant use have occurred in patients taking fenfluarmine or dexfenfluramine, either alone or in combination with each other or other drugs, such as phentermine. There have been only a few cases of PPH reported among patients taking phentermine alone, although the

possibility that phentermine alone may be associated with PPH cannot be ruled out at this time.

Animal research has suggested that appetite suppressant medications affecting the neurotransmitter serotonin, such as fenfluramine and dexfenfluramine, can damage the central nervous system. These findings have not been reported in humans. Some patients have reported depression or memory loss when using appetite suppressant medications alone or in combination, but it is not known if these problems are actually caused by the medications or by other factors.

Over-the-counter appetite suppressants

In addition to the numerous prescription medications for weight loss, a few over-the-counter agents are marketed for weight loss. The most common, phenylpropanalomine, is an appetite suppressant that is distantly related to the amphetamines. Like the amphetamines, this drug has the side effect of increased blood pressure and heart rate, and thus should not be used by anyone with hypertension or heart disease. Other over-the-counter medications contain fiber or bulking agents, and presumably work by increasing the sensation of fullness. Some preparations contain the anesthetic benzocaine. This agent numbs the mouth and may make eating less appealing temporarily. No evidence exists that any of these medications is effective in producing significant weight loss.

See also Amphetamines and related disorders; Diets; Anorexia nervosa; Bulimia nervosa; Obesity; Self-help groups; Support groups

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Overeaters Anonymous, 4025 Spencer Street, Suite 203, Torrance, CA 90503. (310) 618-8835. <http://www.overeatersanonymous.org/>.

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Aricept *see* **Donepezil**

Aromatherapy

Definition

Aromatherapy is a holistic treatment based on the external use of essential aromatic plant oils to maintain and promote physical, physiological, and spiritual well-being. The essential oils may be used in massage, added to a warm bath, used to moisten a compress that is applied to the affected part of the body, added to a vaporizer for inhalation, or diffused throughout a room.

The term aromatherapy (*aromatherapie* in the original French) was coined in 1928 by a French chemist, René Maurice Gattefossé, to describe the therapeutic use of aromatic substances (essential oils) in wound healing. Gattefossé discovered the healing properties of essential plant oils accidentally; after burning his hand in a laboratory accident, he found that **lavender** oil healed his burns in a very short time. He then experimented with plant oils in treating soldiers wounded in World War I, and found that there were several essential oils that speeded physical healing. As the practice of aromatherapy expanded, it came to incorporate a holistic emphasis on healing or invigorating all levels of a person's being. In the United States and Great Britain, the contemporary practice of aromatherapy is often associated with naturopathy and Western herbal medicine. In Great Britain, aromatherapy is one of the most frequently used forms of alternative medicine; in the United States, many hospital-affiliated centers for the study of complementary and alternative medicine (CAM) offer aromatherapy as well as other alternative approaches. Aromatherapy has also been added to holistic nursing board examinations in the United States within the last few years.

Purpose

One of the basic concepts of mind/body medicine is that a positive frame of mind helps to keep people in good health. Aromatherapists maintain that essential oils derived from plants help people to slow down, relax from **stress**, and enjoy the sensory experiences of massage, warm water, and pleasant smells. Aromatherapy is thought to improve a person's mental outlook and sense of well-being by affecting the limbic system via the olfactory nerve, or the sense of smell. The limbic system is the area of the **brain** that regulates emotions. Relaxing and pleasant smells stimulate emotional responses of pleasure and relaxation. From a holistic perspective, aromatherapy is a form of preventive health care. Most aromatherapists believe that aromatherapy should not be used as a substitute for mainstream medical or psychiatric care, but as an adjunct to it.

Aromatherapy is considered to be a useful complementary treatment for the relief of depression, anxiety, **insomnia**, **panic disorder**, stress-related physical disorders, menstrual cramps, and some gastrointestinal complaints. For example, peppermint oil calms gastrointestinal spasms when ingested, or taken by mouth. A recent Scottish study found that aromatherapy has a measurably calming effect on the symptoms of **dementia** in elderly people.

Aromatherapy can be used by itself, or combined with Swedish massage, shiatsu, acupressure, reflexology, or **light therapy** to reinforce the positive results of these treatments.

Although there are professional aromatherapists as well as practitioners of holistic medicine who offer aromatherapy among their other services, people can also use aromatherapy at home as part of self-care. There are many guides to the various techniques of aromatherapy and the proper use of essential plant oils available in inexpensive paperback editions.

Precautions

People who are interested in using essential oils at home should be careful to purchase them from reliable sources. The U. S. Food and Drug Administration (FDA) does not regulate the manufacture of essential plant oils. Consequently, instances of consumer fraud have been reported. In the case of essential oils, the most common form of fraud is substitution of synthetic compounds for natural essential oils, which are expensive to produce.

Another precaution is to avoid applying essential oils directly to the skin as a form of perfume. Some essential oils such as oil of orange or oil of peppermint are irritating to the skin if applied full-strength. When essential oils are used in massage, they are always diluted in a carrier oil.

A final precaution is to avoid taking essential oils internally without a consultation with a physician or naturopathist. Possible exceptions may be peppermint oil and aloe vera.

KEY TERMS

Carrier—A vegetable oil such as safflower, olive, grapeseed, or wheatgerm oil used to dilute essential oils for massage.

Enfleurage—A technique for extracting essential oils from flower petals by placing them on a layer of purified fat.

Essential oil—The product of special ducts or cells in the tissues of aromatic plants (or the sap of certain trees) that gives the plant its characteristic aroma and therapeutic properties. Essential oils are sometimes called volatile oils because they evaporate readily at room temperature.

Limbic system—A group of structures in the brain that includes the amygdala, hippocampus, olfactory bulbs, and hypothalamus. The limbic system is associated with homeostasis and the regulation and arousal of emotions.

Maceration—A technique for extracting essential oils from plant leaves and stems by crushing the plant parts and soaking them in warm vegetable oil.

Olfactory nerve—The cranial nerve that regulates the sense of smell.

Description

Essential plant oils are prepared for use in aromatherapy in several different ways. Most are prepared by steam distillation, a process in which the flowers, leaves, or other plant parts are heated by steam from boiling water. The vapors that result then pass through a condenser that separates the scented water from the essential oil, which is siphoned off into a separate container. Other methods of extracting essential oils include expression, or squeezing, which is limited to citrus oils; *enfleurage*, in which flower petals are placed on a bed of purified fat that soaks up the essential oils; and maceration, in which the plant parts are crushed and covered with warm vegetable oil that absorbs the essential oils.

There are several different techniques for the use of essential oils in aromatherapy:

- Massage: This is the technique that most people associate with aromatherapy. For use in massage, essential oils are mixed with a vegetable carrier oil, usually wheatgerm, avocado, olive, safflower, grapeseed, or soya bean oil. A ratio that is commonly recommended is 2.5–5% essential oil to 95–97.5% carrier oil.
- Full-body baths: In this technique, the essential oil is added to a tubful of warm (but not hot) water as the water is running. The dosage of essential oil is usually 5–10 drops per bath.
- Hand or foot baths: These are often recommended to treat arthritis or skin disorders of the hands or feet as well as sore muscles. The hands or feet are soaked for 10–15 minutes in a basin of warm water to which 5–7 drops of essential oil have been added.
- Inhalations: This technique is used to treat sinus problems or such nasal allergies as hay fever. Two cups of water are brought to a boil and then allowed to cool for five to ten minutes. Two to five drops of essential oil are added to the steaming water, and the person leans over the container and inhales the fragrant vapors for five to ten minutes.
- Diffusion: This technique requires the use of a special nebulizer to disperse microscopic droplets of essential oil into the air, or a clay diffuser that allows the oil to evaporate into the air when it is warmed by a small votive candle or electric bulb. Diffusion is recommended for treating emotional upsets.
- Compresses: These are made by soaking four or five layers of cotton cloth in a solution of warm water and essential oil, wringing out the cloth so that it is moist but not dripping, and applying it to the affected part of the body. The compress is then covered with a layer of plastic wrap, followed by a pre-warmed towel, and kept in place for one or two hours. Aromatherapy compresses are used to treat wounds, sprains, bruises, sore muscles, menstrual cramps, and respiratory congestion.
- Aromatic salves: Salves are made by melting together 1 1/4 cup of vegetable oil and 1 oz of beeswax in a double boiler over medium heat, and adding the desired combination of essential oils.
- Internal use: Some essential oils such as oil of peppermint and cinnamon can be used to make teas or mouthwashes, or mixed with a glass of honey and water. The dose depends on the oil, but a physician, naturopathist, or other practitioner should be consulted.

Preparation

Aromatherapists recommend the use of fresh oils and oil mixtures in the techniques described above. Both essential oils and vegetable carrier oils deteriorate over time and should not be kept longer than one or two months; thus, it is best to mix only small quantities of massage oils or salves at any one time.

No special preparation for an aromatherapy treatment is required on the patient's part.

Aftercare

Aromatherapy does not require any particular form of aftercare, although many patients like to rest quietly for a few minutes after a bath or massage with essential oils.

Risks

There are no risks involved in external aromatherapy when essential oils are diluted as recommended. Not all essential oils, however, should be taken internally. Benzoin and other essential oils derived from tree resins should not be used internally.

A few cases have been reported of dissociative episodes triggered by fragrances associated with traumatic experiences. Patients in treatment for **post-traumatic stress disorder** (PTSD) or any of the dissociative disorders should consult their therapist before they use aromatherapy.

Normal results

Normal results from aromatherapy include a sense of relaxation, relief from tension, and improved well-being.

Abnormal results

Abnormal results include skin irritations or other allergic reactions to essential oils, and the development of traumatic memories associated with specific smells.

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ORGANIZATIONS

American Association of Naturopathic Physicians. 601 Valley Street, Suite 105, Seattle, WA 98109. (206) 298-0126. <www.naturopathic.org>.

International Aromatherapy and Herb Association. 3541 West Acapulco Lane. Phoenix, AZ 85053-4625. (602) 938-4439. <www.aztec.asu.edu./iaha/>.

National Association for Holistic Aromatherapy (NAHA). 4509 Interlake Avenue North, #233, Seattle, WA 98103-6773. (888) ASK-NAHA or (206) 547-2164. <www.naha.org>.

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Art therapy *see* **Creative therapies**

Artane *see* **Trihexyphenidyl**

Asendin *see* **Amoxapine**

Asperger's disorder

KEY TERMS

Autistic psychopathy—Hans Asperger's original name for the condition now known as Asperger's disorder. It is still used occasionally as a synonym for the disorder.

DSM—Abbreviation for the *Diagnostic and Statistical Manual of Mental Disorders,* a handbook for mental health professionals that includes lists of symptoms that indicate specific diagnoses. The text is periodically revised, and the latest version was published in 2000 and is called *DSM-IV-TR*, for Fourth Edition, Text Revised.

Gillberg's criteria—A six-item checklist for AS developed by Christopher Gillberg, a Swedish researcher. It is widely used in Europe as a diagnostic tool.

High-functioning autism (HFA)—A subcategory of autistic disorder consisting of children diagnosed with IQs of 70 or higher. Children with AS are often misdiagnosed as having HFA.

Nonverbal learning disability (NLD)—A learning disability syndrome identified in 1989 that may overlap with some of the symptoms of AS.

Pervasive developmental disorders (PDDs)—A category of childhood disorders that includes Asperger's syndrome and Rett's disorder. The PDDs are sometimes referred to collectively as autistic spectrum disorders.

Semantic-pragmatic disorder—A term that refers to the difficulty that children with AS and some forms of autism have with pragmatic language skills. Pragmatic language skills include knowing the proper tone of voice for a given context, using humor appropriately, making eye contact with a conversation partner, maintaining the appropriate volume of one's voice, etc.

Definition

Asperger's disorder, which is also called Asperger's syndrome (AS) or autistic psychopathy, belongs to a group of childhood disorders known as **pervasive developmental disorders** (PDDs) or autistic spectrum disorders. The essential features of Asperger's disorder are severe social interaction impairment and restricted, repetitive patterns of behavior and activities. It is similar to **autism**, but children with Asperger's do not have the same difficulties in acquiring language that children with autism have.

In the mental health professional's diagnostic handbook, the ***Diagnostic and Statistical Manual of Mental Disorders*** fourth edition text revised, or *DSM-IV-TR*, Asperger's disorder is classified as a developmental disorder of childhood.

Description

AS was first described by Hans Asperger, an Austrian **psychiatrist**, in 1944. Asperger's work was unavailable in English before the mid-1970s; as a result, AS was often unrecognized in English-speaking countries until the late 1980s. Before *DSM-IV* (published in 1994) there was no officially agreed-upon definition of AS. In the words of ICD-10, the European equivalent of the *DSM-IV,* Asperger's is "a disorder of uncertain nosological validity." (Nosological refers to the classification

of diseases.) There are three major reasons for this lack of clarity: differences between the diagnostic criteria used in Europe and those used in the United States; the fact that some of the diagnostic criteria depend on the observer's interpretation rather than objective measurements; and the fact that the clinical picture of Asperger's changes as the child grows older.

Asperger's disorder is one of the milder pervasive developmental disorders. Children with AS learn to talk at the usual age and often have above-average verbal skills. They have normal or above-normal intelligence and the ability to feed or dress themselves and take care of their other daily needs. The distinguishing features of AS are problems with social interaction, particularly reciprocating and empathizing with the feelings of others; difficulties with nonverbal communication (such as facial expressions); peculiar speech habits that include repeated words or phrases and a flat, emotionless vocal tone; an apparent lack of "common sense" a fascination with obscure or limited subjects (for example, the parts of a clock or small machine, railroad schedules, astronomical data, etc.) often to the exclusion of other interests; clumsy and awkward physical movements; and odd or eccentric behaviors (hand wringing or finger flapping; swaying or other repetitious whole-body movements; watching spinning objects for long periods of time).

Causes and symptoms

Causes

There is some indication that AS runs in families, particularly in families with histories of depression and **bipolar disorder**. Asperger noted that his initial group of patients had fathers with AS symptoms. Knowledge of the genetic profile of the disorder, however, is quite limited as of 2002.

In addition, about 50% of AS patients have a history of oxygen deprivation during the birth process, which has led to the hypothesis that the disorder is caused by damage to **brain** tissue before or during childbirth. Another cause that has been suggested is an organic defect in the functioning of the brain.

As of 2002, there is no known connection between Asperger's disorder and childhood trauma, **abuse** or **neglect**.

Symptoms

In young children, the symptoms of AS typically include problems picking up social cues and understanding the basics of interacting with other children. The child may want friendships but find him- or herself unable to make friends.

Most children with Asperger's are diagnosed during the elementary school years because the symptoms of the disorder become more apparent at this point. They include:

- Poor pragmatic language skills. This phrase means that the child does not use the right tone or volume of voice for a specific context, and does not understand that using humorous or slang expressions also depends on social context.
- Problems with hand-eye coordination and other visual skills.
- Problems making eye contact with others.
- Learning difficulties, which may range from mild to severe.
- Tendency to become absorbed in a particular topic and not know when others are bored with conversation about it. At this stage in their education, children with AS are likely to be labeled as "nerds."
- Repetitive behaviors. These include such behaviors as counting a group of coins or marbles over and over; reciting the same song or poem several times; buttoning and unbuttoning a jacket repeatedly; etc.

Adolescence is one of the most painful periods of life for young people with Asperger's, because social interactions are more complex in this age group and require more subtle social skills. Some boys with AS become frustrated trying to relate to their peers and may become aggressive. Both boys and girls with the disorder are often quite naive for their age and easily manipulated by "street-wise" classmates. They are also more vulnerable than most youngsters to peer pressure.

Little research has been done regarding adults with AS. Some have serious difficulties with social and occupational functioning, but others are able to finish their schooling, join the workforce, and marry and have families.

Demographics

Although the incidence of AS has been variously estimated between 0.024% and 0.36% of the general population in North America and northern Europe, further research is required to determine its true rate of occurrence—especially because the diagnostic criteria have been defined so recently. In addition, no research regarding the incidence of AS has been done on the populations of developing countries, and nothing is known about the incidence of the disorder in different racial or ethnic groups.

With regard to gender differences, AS appears to be much more common in boys. Dr. Asperger's first patients were all boys, but girls have been diagnosed with AS

since the 1980s. One Swedish study found the male/female ratio to be 4:1; however, the World Health Organization's ICD-10 classification gives the male to female ratio as 8 to 1.

Diagnosis

As of early 2002, there are no blood tests or brain scans that can be used to diagnose AS. Until *DSM-IV* (1994), there was no "official" list of symptoms for the disorder, which made its **diagnosis** both difficult and inexact. Although most children with AS are diagnosed between five and nine years of age, many are not diagnosed until adulthood. Misdiagnoses are common; AS has been confused with such other neurological disorders as Tourette's syndrome, or with attention-deficit disorder (ADD), **oppositional defiant disorder** (ODD), or **obsessive-compulsive disorder** (OCD). Some researchers think that AS may overlap with some types of learning disability, such as the nonverbal learning disability (NLD) syndrome identified in 1989.

The inclusion of AS as a separate diagnostic category in *DSM-IV* was justified on the basis of a large international field trial of over a thousand children and adolescents. Nevertheless, the diagnosis of AS is also complicated by confusion with such other diagnostic categories as "high-functioning (IQ higher than 70) autism" or HFA, and "schizoid personality disorder of childhood." Unlike **schizoid personality disorder** of childhood, AS is not an unchanging set of personality traits—AS has a developmental dimension. AS is distinguished from HFA by the following characteristics:

- Later onset of symptoms (usually around three years of age).
- Early development of grammatical speech; the AS child's verbal IQ (scores on verbal sections of standardized **intelligence tests**) is usually higher than performance IQ (how well the child performs in school). The reverse is usually true for autistic children.
- Less severe deficiencies in social and communication skills.
- Presence of intense interest in one or two topics.
- Physical clumsiness and lack of coordination.
- Family is more likely to have a history of the disorder.
- Lower frequency of neurological disorders.
- More positive outcome in later life.

DSM-IV-TR criteria for Asperger's disorder

The *DSM-IV-TR* specifies the following diagnostic criteria for AS:

- The child's social interactions are impaired in at least two of the following ways: markedly limited use of nonverbal communication (facial expressions, for example); lack of age-appropriate peer relationships; failure to share enjoyment, interests, or accomplishment with others; lack of reciprocity (turn-taking) in social interactions.
- The child's behavior, interests, and activities are characterized by repetitive or rigid patterns, such as an abnormal preoccupation with one or two topics, or with parts of objects; repetitive physical movements; or rigid insistence on certain routines and rituals.
- The patient's social, occupational, or educational functioning is significantly impaired.
- The child has normal age-appropriate language skills.
- The child has normal age-appropriate cognitive skills, self-help abilities, and curiosity about the environment.
- The child does not meet criteria for another specific PDD or schizophrenia.

To establish the diagnosis, the child psychiatrist or **psychologist** would observe the child, and would interview parents, possibly teachers, and the affected child (depending on the child's age), and would gather a comprehensive medical and social history.

Other diagnostic scales and checklists

Other instruments that have been used to identify children with AS include Gillberg's criteria, a six-item list compiled by a Swedish researcher that specifies problems in social interaction, a preoccupying narrow interest, forcing routines and interests on the self or others, speech and language problems, nonverbal communication problems, and physical clumsiness; and the Australian Scale for Asperger's Syndrome, a detailed multi-item questionnaire developed in 1996.

Brain imaging findings

As of 2002, only a few structural abnormalities of the brain have been linked to AS. Findings include abnormally large folds in the brain tissue in the left frontal region, abnormally small folds in the operculum (a lid-like structure composed of portions of three adjoining brain lobes), and damage to the left temporal lobe (a part of the brain containing a sensory area associated with hearing). The first single photon emission tomography (SPECT) study of an AS patient found a lower-than-normal supply of blood to the left parietal area of the brain, an area associated with bodily sensations. Brain **imaging studies** on a larger sample of AS patients is the next stage of research.

Yoga instructor and three teenagers in an integrated movement therapy session. The teens have various pervasive developmental disorders, including Asperger's disorder and autism. These therapy sessions combine social interaction and movement, both of which are beneficial for adolescents with autistic disorders. *(AP Photo/Cheryl Hatch. Photo reproduced by permission.)*

Treatments

As of 2002, there is no cure for AS and no prescribed treatment regimen for all AS patients. Specific treatments are based on the individual's symptom pattern.

Medications

Many children with AS do not require any medication. For those who do, the drugs that are recommended most often include psychostimulants (**methylphenidate**, **pemoline**), **clonidine**, or one of the tricyclic antidepressants (TCAs) for hyperactivity or inattention; **beta blockers**, neuroleptics (antipsychotic medications), or lithium (**lithium carbonate**) for anger or aggression; selective serotonin reuptake inhibitors (SSRIs) or TCAs for rituals (repetitive behaviors) and preoccupations; and SSRIs or TCAs for anxiety symptoms. One alternative herbal remedy that has been tried with AS patients is **St. John's wort**.

Psychotherapy

AS patients often benefit from individual **psychotherapy**, particularly during adolescence, in order to cope with depression and other painful feelings related to their social difficulties. Many children with AS are also helped by **group therapy**, which brings them together with others facing the same challenges. There are therapy groups for parents as well.

Therapists who are experienced in treating children with Asperger's disorder have found that the child should be allowed to proceed slowly in forming an emotional bond with the therapist. Too much emotional intensity at the beginning may be more than the child can handle. Behavioral approaches seem to work best with these children. **Play therapy** can be helpful in teaching the child to recognize social cues as well as lowering the level of emotional tension.

Adults with AS are most likely to benefit from individual therapy using a cognitive-behavioral approach, although many also attend group therapy. Some adults have been helped by working with speech therapists on their pragmatic language skills. A relatively new approach called behavioral coaching has been used to help adults with Asperger's learn to organize and set priorities for their daily activities.

Educational considerations

Most AS patients have normal or above-normal intelligence, and are able to complete their education up through the graduate or professional school level. Many are unusually skilled in music or good in subjects requiring rote memorization. On the other hand, the verbal skills of children with AS frequently cause difficulties with teachers, who may not understand why these "bright" children have social and communication problems. Some AS children are dyslexic; others have difficulty with writing or mathematics. In some cases, AS children have been mistakenly put in special programs either for children with much lower levels of functioning, or for children with conduct disorders. AS children do best in structured learning situations in which they learn problem-solving and social skills as well as academic subjects. They frequently need protection from the teasing and bullying of other children, and often become hypersensitive to criticism by their teenage years. One approach that has been found helpful at the high-school level is to pair the adolescent with AS with a slightly older teenager who can serve as a mentor. The mentor can "clue in" the younger adolescent about the slang, dress code, cliques, and other "facts of life" at the local high school.

Employment

Adults with AS are productively employed in a wide variety of fields, including the learned professions. They do best, however, in jobs with regular routines or occupations that allow them to work in isolation. In large companies, employers or supervisors and workplace colleagues may need some information about AS in order to understand the new employee's "eccentricities."

Prognosis

AS is a lifelong but stable condition. The prognosis for children with AS is generally good as far as intellectual development is concerned, although few school districts as of 2002 are equipped to meet the special social needs of this group of children. Adults with AS appear to be at greater risk of depression than the general population. In addition, some researchers believe that people with AS have an increased risk of a psychotic episode (a period of time during which the affected person loses touch with reality) in adolescence or adult life.

Prevention

Effective prevention of Asperger's disorder awaits further genetic mapping together with ongoing research in the structures and functioning of the brain. The only practical preventive strategy as of 2002 is better protection of the fetus against oxygen deprivation during childbirth.

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ORGANIZATIONS

Autism Research Institute. 4182 Adams Avenue, San Diego, CA 92116.

Families of Adults Afflicted with Asperger's Syndrome (FAAAS). P.O. Box 514, Centerville, MA 02632. <www.faaas.org>.

National Association of Rare Disorders (NORD). P.O. Box 8923, New Fairfield, CT 06812-8923. (800) 999-NORD or (203) 746-6518.

Yale-LDA Social Learning Disabilities Project. Yale Child Study Center, New Haven, CT. The Project is looking for study subjects with PDDs between the ages of 8 and 24, including AS patients. Contact person: Sanno Zack at (203) 785-3488 or Sanno.Zack@yale.edu. <www.info.med.Yale.edu/chldstdy/autism>.

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Assertiveness training

Definition

Assertiveness training is a form of behavior therapy designed to help people stand up for themselves—to empower themselves, in more contemporary terms. Assertiveness is a response that seeks to maintain an appropriate balance between passivity and aggression. Assertive responses promote fairness and equality in human interactions, based on a positive sense of respect for self and others.

Assertiveness training has a decades-long history in mental health and personal growth groups, going back to the women's movement of the 1970s. The approach was introduced to encourage women to stand up for themselves appropriately in their interactions with others, particularly as they moved into graduate education and the workplace in greater numbers. The original association of assertiveness training with the women's movement in the United States grew out of the discovery of many women in the movement that they were hampered by their inability to be assertive. Today, assertiveness training is used as part of communication training in settings as diverse as schools, corporate boardrooms, and psychiatric hospitals, for programs as varied as substance abuse treatment, **social skills training**, vocational programs, and responding to harassment.

Purpose

The purpose of assertiveness training is to teach persons appropriate strategies for identifying and acting on their desires, needs, and opinions while remaining respectful of others. This form of training is tailored to the needs of specific participants and the situations they find particularly challenging. Assertiveness training is a broad approach that can be applied to many different personal, academic, health care, and work situations.

Learning to communicate in a clear and honest fashion usually improves relationships within one's life. Women in particular have often been taught to hide their real feelings and preferences, and to try to get their way by manipulation or other indirect means. Specific areas of **intervention** and change in assertiveness training include conflict resolution, realistic goal-setting, and **stress** management. In addition to emotional and psychological benefits, taking a more active approach to self-determination has been shown to have positive outcomes in many personal choices related to health, including being assertive in risky sexual situations; abstaining from using drugs or alcohol; and assuming responsibility for self-care if one has a chronic illness like diabetes or cancer.

Precautions

There are a few precautions with assertiveness training. One potential caution would be to remain within assertive responses, rather than become aggressive in standing up for oneself. Some participants in assertiveness training programs who are just learning the techniques of appropriate assertiveness may "overdo" their new behaviors and come across as aggressive rather than assertive. Such overcompensation would most likely disappear with continued practice of the techniques.

One additional precaution about assertiveness training is that it should not be regarded as the equivalent of martial arts training or similar physical self-defense techniques. It is important to distinguish between contexts or situations in which verbal assertiveness is appropriate and useful, and those in which it is irrelevant. In some situations, a person's decision to leave the situation or seek help because they sense danger is preferable to an encounter with a criminal.

Description

Assertiveness training is often included within other programs, but "stand-alone" programs in self-assertion are often given in women's centers or college counseling centers. Corporate programs for new personnel sometimes offer assertiveness training as part of communication or teamwork groups, or as part of a program on sexual harassment.

Assertiveness training typically begins with an information-gathering exercise in which participants are asked to think about and list the areas in their life in which they have difficulty asserting themselves. Very often they will notice specific situations or patterns of behavior that they want to focus on during the course. The next stage in assertive training is usually role-plays designed to help participants practice clearer and more direct forms of communicating with others. The role-plays allow for practice and repetition of the new techniques, helping each person learn assertive responses by acting on them. Feedback is provided to improve the response, and the role-play is repeated. Eventually, each person is asked to practice assertive techniques in everyday life, outside the training setting. Role-plays usually incorporate specific problems for individual participants, such as difficulty speaking up to an overbearing boss; setting limits to intrusive friends; or stating a clear preference about dinner to one's spouse. Role-plays often include examples of aggressive and passive responses, in

addition to the assertive responses, to help participants distinguish between these extremes as they learn a new set of behaviors.

Assertiveness training promotes the use of "I" statements as a way to help individuals express their feelings and reactions to others. A commonly used model of an "I" statement is "when you _____, I feel _____", to help the participant describe what they see the other person as doing, and how they feel about that action. "I" statements are often contrasted with "you" statements, which are usually not received well by others. For example, "When you are two hours late getting home from work, I feel both anxious and angry," is a less accusing communication than "You are a selfish and inconsiderate jerk for not telling me you would be two hours late." Prompts are often used to help participants learn new communication styles. This approach helps participants learn new ways of expressing themselves as well as how it feels to be assertive.

Learning specific techniques and perspectives, such as self-observation skills, awareness of personal preferences and assuming personal responsibility are important components of the assertiveness training process. Role-play and practice help with self-observation, while making lists can be a helpful technique for exploring personal preferences for those who may not have a good sense of their own needs and desires. Participants may be asked to list anything from their ten favorite movies or pieces of music to their favorite foods, places they would like to visit, subjects that interest them, and so on.

Preparation

Preparation for assertiveness training varies from person to person. For some participants, no preparation is needed before practicing the techniques; for others, however, individual counseling or therapy may help prepare the individual for assertiveness training. For participants who may be more shy and feel uncomfortable saying "no" or speaking up for themselves, a brief course of individual therapy will help to prepare them psychologically and emotionally to use assertive techniques.

Aftercare

Aftercare can involve ongoing supportive therapy, again based on the individual's level of comfort in using the assertive techniques. For those who are comfortable using the techniques on their own, a supportive social network or occasional participation in a support group will be enough to help maintain the new behavioral patterns. The ultimate goal is for each participant to self-monitor effectively his or her use of assertive techniques on an ongoing basis.

KEY TERMS

Assertive—Confidently self-assured; able to express oneself constructively and directly.

Overcompensation—An attempt to overcome or correct a behavior by going too far in the opposite direction.

Role-playing—A technique used in assertiveness training and other forms of therapy in which participants act out roles relevant to real-life situations in order to change their attitudes and behaviors.

Risks

There are minimal risks associated with assertiveness training. Personal relationships may be affected if those around the participant have difficulty accepting the changes in their friend or family member. This risk, however, is no greater than that associated with any other life change.

Another potential risk is that of overcompensating in the early stages of training by being too aggressive. With appropriate feedback, participants can usually learn to modify and improve their responses.

People who are very shy or self-conscious, or who were harshly treated as children, may also experience anxiety during the training as they work toward speaking up and otherwise changing their behaviors. The anxiety may be uncomfortable, but should decrease as the person becomes more comfortable with the techniques and receives encouragement from others in the program.

Normal results

An enhanced sense of well-being and more positive self-esteem are typical results from assertiveness training. Many participants report that they feel better about themselves and more capable of handling the stresses of daily life. In addition, people who have participated in assertiveness training have a better sense of boundaries, and are able to set appropriate and healthy limits with others. Being able to set appropriate limits (such as saying "no") helps people to avoid feeling victimized by others.

A healthy sense of self-determination and respect for others is the ultimate outcome of assertiveness training. Such a balance helps each person work better with others, and make appropriate decisions for themselves.

Abnormal results

Unusual results may include becoming too aggressive in setting boundaries, as if the individual is overcompensating. With appropriate training, role-play, and feedback, this response can be re-learned. Alternatively, for very shy individuals, a heightened sense of anxiety may be experienced when using the techniques initially. The nervousness or anxiety is usually due to the individual's concern about others' reactions to their assertive responses. Over time, the anxiety will usually decrease.

See also Behavior modification; Gender issues in mental health

Resources

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Deanna Pledge, Ph.D.

Assessment and diagnosis

Definition

The psychological assessment is a structured interview that gathers information from and/or tests a person to evaluate a mental health complaint.

Purpose

The psychological assessment (also called the biopsychosocial or psychiatric assessment) gathers information to diagnose any mental disorder that the person may have; it is the first step in treating a diagnosed disorder. The process typically starts with a chief complaint or presenting problem—this is usually what prompts the person to seek help. A complete psychological assessment should include:

- biopsychosocial history
- neurological assessment
- psychological testing (if applicable)
- physical examination (if required by a **psychiatrist**)
- **brain** imaging (if necessary)

Once complete, the assessment will help establish either a tentative or definitive **diagnosis**. With this information, the clinician can inform the patient of the results, and treatment can begin.

Precautions

Accurate information gathering and objective notes are essential for psychological assessment. However, these can be difficult to obtain if the person is not willing to disclose all necessary information, either out of embarrassment or through **denial** that symptoms of a mental problem even exist.

Description

The psychological assessment, an extremely effective and accepted diagnostic tool, is a structured interview that has several parts:

- identifying information
- chief complaint (presenting problem)
- history of present illness
- past medical and psychological history
- personal history
- family history
- substance abuse history
- mental status examination (MSE)

Before beginning, the clinician should introduce himself or herself and attempt to make the person comfortable in a professional setting. A common fluency in language or competent translator is essential for information gathering and questioning.

Identifying information

These are general and emotionally neutral questions that usually include name, age, occupation, and marital status.

Chief complaint (presenting problem)

This consists of questions such as "Why are you seeking psychological help today?" that reveal past mental disorders and/or the symptoms that made the person seek **psychotherapy**. The patient's responses can also help the clinician ask pertinent questions during other parts of the interview, and can help clarify the presence of symptoms.

History of present illness

The patient describes the onset of signs and symptoms that comprise the current mental problem.

Past medical and psychological history

Because medical problems—including thyroid disease, Parkinson's disease, head trauma, and brain infections—can cause psychological symptoms, a thorough medical history must be taken. The interviewer also asks about previous psychological/psychiatric treatment, including **hospitalization**, outpatient or substance abuse treatment, and medication prescribed for mental disorders. The treatment's duration, effectiveness, and outcome is also noted.

Personal history

This portion of the assessment provides information on the patient's entire life, beginning with prenatal development, including maternal abortions, nutrition, and drug use during pregnancy; birth trauma; and birth order. The patient's life is then discussed in distinct phases:

EARLY CHILDHOOD (INFANCY–THREE YEARS). Questions include information about temperament, walking, talking, toilet training, nutrition and feeding, family relationships, behavioral problems, hospitalization, and separation from early childhood caregivers.

MIDDLE CHILDHOOD (THREE–11 YEARS). Pertinent information will be gathered concerning learning, relationship with peers and family, behavioral problems, and general personality development.

ADOLESCENCE (12–18 YEARS). Information typically includes school history, behavioral problems, and sexual development.

ADULTHOOD. This section details the patient's education, sexual history, relationships and/or marriages, peer relationships, occupation, and current circumstances.

Family history

Family history is crucially important since many mental disorders can be inherited genetically. Additionally, family interactions may affect the patient's symptoms and disorder.

KEY TERMS

Affect—The expression of emotion displayed to others through facial expressions, hand gestures, tone of voice, etc. Types of affect include: flat (inanimate, no expression), blunted (minimally responsive), inappropriate (incongruous expressions of emotion relative to the content of a conversation), and labile (sudden and abrupt changes in type and intensity of emotion).

Assessment—In the context of psychological assessment (a structured interview), assessment is information-gathering to diagnose a mental disorder.

Biopsychosocial history—A history of significant past and current experiences that influence client behaviors, including medical, educational, employment, and interpersonal experiences. Alcohol or drug use and involvement with the legal system are also assessed in a biopsychosocial history.

Delusion—A false belief that is resistant to reason or contrary to actual fact. A patient may be convinced, for example, that someone is trying to poison him or her, or that he or she has a fatal illness despite evidence to the contrary.

Dependence—The adaptation of neurons and other physical processes to the use of a drug, followed by withdrawal symptoms when the drug is removed; physiological and/ or psychological addiction.

Hallucinations—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Phobia—Irrational fear of places, things, or situations that lead to avoidance.

Psychotropic drug—Medication that has an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

The psychological assessment (also called the biopsychosocial or psychiatric assessment) gathers information to diagnose any mental disorder that the person may have. A complete psychological assessment should include: complete and extensive medical and psychological history, neurological assessment, and may also include further tests and imaging studies. Once complete, the assessment will help establish a diagnosis. *(Photo by John Henley, The Stock Market. Reproduced by permission.)*

Substance use history

This portion of the psychological assessment details information on the patient's use of both illicit drugs (opiates, cocaine, alcohol, marijuana, hallucinogens, and depressants) and legally prescribed medications, as well as nicotine and caffeine. Questions usually focus on age of first use, age of last use, period of heaviest use, usage within the past 30 days, frequency, quantity, and route of usage. Tolerance and dependence, if present, are noted, as are the patient's treatment history, any medical complications (AIDS, for example), and legal problems associated with usage (such as driving or operating a vehicle or machine while impaired).

Mental Status Examination (MSE)

This assesses the patient's mental state, and begins by evaluating:

- Appearance—hygiene, general appearance, grooming, and attire.
- Behavior—abnormal movements, hyperactivity and eye contact with the interviewer.
- Speech—fluency, rate, clarity, and tone, all of which may indicate the patient's mental state. A fast-talking person, for example, may be anxious. Speech can also reveal intoxication or impairment as well as problems in the mouth (i.e. dentures, cleft palate) or speech impairment.

The examiner then goes on to assess other aspects of the patient's mental state, such as mood, thought process, and cognition, beginning with a question such as that suggested in the *Merck Manual of Geriatrics.* "I would like to ask you some questions about your feelings, your thinking, and your memory as a routine part of the examination. Is that all right with you?"

Mood and affect

These outward manifestations of the patient's mental state are important indicators. The clinician can ask the patient to describe his or her current mood ("How do you feel? Are you happy? Sad? Angry?"). The patient's affect, or emotional state, however, is observed and interpreted by the clinician throughout the interview, and described in standardized terms, such as excitable, flat, inappropriate, or labile (rapidly shifting).

Thought process and content

Thought process (or form) indicates whether or not the interviewee is properly oriented to time and place. Thought content reveals how connected, coherent, and logical the patient's thoughts are. The interviewer may ask the patient to identify themselves and loved ones, to name the current date, and/or to describe the route taken to the examiner's location. The patient's responses to questions can indicate disturbances in thought, such as circumstantial thinking (circuitous, persistent storytelling), tangential thinking (response not pertinent to the question) black/white (extreme) thinking, and impoverished (minimally responsive) thinking. Disturbed thought content can also indicate **delusions**, **hallucinations**, phobias, and obsessions. In addition, the examiner may question the patient about suicidal and/or homicidal thoughts.

Cognition

Cognition refers to the patient's attention, awareness, memory (long-, intermediate-, and short-term), general knowledge, abstract thinking ability, insight, and judgment. The interviewer may ask the patient to spell a word forward and backward, identify the current president, read and/or write something, compare two objects, and explain the meaning of common sayings.

Preparation

An evaluation session appointment is made with a qualified mental health practitioner. A specialist (someone specializing in anxiety/depressive disorders, pain management, **hypnotherapy**, or chemical dependency, for example) may be sought or recommended. A private,

quiet, nonthreatening, environment is recommended to ensure comfort and confidentiality.

Aftercare

Aftercare depends on the results of the evaluation. Treatment may be initiated and/or further tests may be required to confirm the diagnosis.

Risks

There are no known risks involved. A person seeking a mental health evaluation does so for a reason and may learn of an existing or potential mental problem.

Normal results

The patient does not require psychological therapy or psychotropic drug (medications beneficial to treat certain mental disorders) treatment.

Abnormal results

The person suffers from a mental disorder that may require psychotherapy or a combination of psychotherapy and medications.

Resources

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Laith Farid Gulli, M.D.
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Robert Ramirez

Ativan *see* **Lorazepam**

Attachment disorder *see* **Reactive attachment disorder of infancy or early childhood**

Attention-deficit/ hyperactivity disorder

Definition

Attention-deficit/hyperactivity disorder (ADHD) is a developmental disorder characterized by distractibility, hyperactivity, impulsive behaviors, and the inability to remain focused on tasks or activities.

KEY TERMS

Antisocial personality disorder—Disorder characterized by behavior pattern of disregard for others' rights. People with this disorder often deceive and manipulate, or their behavior might include aggression to people or animals or property destruction, for example. This disorder has also been called sociopathy or psychopathy.

Conduct disorder—A behavioral and emotional disorder of childhood and adolescence in which children display physical aggression and infringe on or violate the rights of others. Youths diagnosed with conduct disorder may set fires, exhibit cruelty toward animals or other children, sexually assault others, or lie and steal for personal gain.

Nervous tic—A repetitive, involuntary action, such as the twitching of a muscle or repeated blinking.

Oppositional defiant disorder—An emotional and behavioral problem of children and adolescents characterized by defiant, hostile, or disobedient behavior that has lasted for longer than six months.

Description

ADHD, also known as hyperkinetic disorder (HKD) outside of the United States, is estimated to affect 3%-9% of children, and afflicts boys more often than girls. Although difficult to assess in infancy and toddlerhood, signs of ADHD may begin to appear as early as age two or three, but the symptom picture changes as adolescence approaches. Many symptoms, particularly hyperactivity, diminish in early adulthood, but impulsivity and problems focusing attention remain with up to 50% of individuals with ADHD throughout their adult life.

Children with ADHD have short attention spans, becoming easily bored and/or frustrated with tasks. Although they may be quite intelligent, their lack of focus frequently results in poor grades and difficulties in school. Children with ADHD act impulsively, taking action first and thinking later. They are constantly moving, running, climbing, squirming, and fidgeting, but often have trouble with gross and fine motor skills and, as a result, may be physically clumsy and awkward. In social settings, they are sometimes shunned due to their impulsive and intrusive behavior.

Causes and symptoms

Causes

The causes of ADHD are not known. However, it appears that heredity plays a major role in the development of ADHD. Children with an ADHD parent or sibling are more likely to develop the disorder themselves. Before birth, ADHD children may have been exposed to poor maternal nutrition, viral infections, or maternal substance abuse. In early childhood, exposure to lead or other toxins can cause ADHD-like symptoms. Traumatic **brain** injury or neurological disorders may also trigger ADHD symptoms. Although the exact cause of ADHD is not known, an imbalance of certain **neurotransmitters** (the chemicals in the brain that transmit messages between nerve cells) is believed to be the mechanism behind ADHD symptoms.

Symptoms

The **diagnosis** of ADHD requires the presence of at least six of the following symptoms of inattention, or six or more symptoms of hyperactivity and impulsivity combined:

Inattention:

- fails to pay close attention to detail or makes careless mistakes in schoolwork or other activities
- has difficulty sustaining attention in tasks or activities
- does not appear to listen when spoken to
- does not follow through on instructions and does not finish tasks
- has difficulty organizing tasks and activities
- avoids or dislikes tasks that require sustained mental effort (such as homework)
- is easily distracted
- is forgetful in daily activities

Hyperactivity:

- fidgets with hands or feet or squirms in seat
- does not remain seated when expected to
- runs or climbs excessively when inappropriate (in adolescents and adults, feelings of restlessness)
- has difficulty playing quietly
- is constantly on the move
- talks excessively

Impulsivity:

- blurts out answers before the question has been completed
- has difficulty waiting for his or her turn
- interrupts and/or intrudes on others

Further criteria to establish a diagnosis also require that some symptoms develop before age seven, and that they significantly impair functioning in two or more settings (home and school, for example) for a period of at least six months.

Many individuals with ADHD have symptoms from all three of the above categories. Some children, however, have behavior patterns in which inattention dominates, or hyperactivity and impulsivity dominate. For this reason, ADHD can be further categorized, or subdivided, into three subtypes. Children who have at least six symptoms from both of the inattention and hyperactivity-impulsivity categories above may be diagnosed with ADHD, combined type. Children who meet the symptom criteria for inattention, but not for hyperactivity/impulsivity are diagnosed with attention-deficit/hyperactivity disorder, predominantly inattentive type, commonly called ADD. Children who experience more symptoms from the hyperactivity and impulsivity categories, but fewer than six symptoms of inattention may be diagnosed with ADHD, predominantly hyperactive-impulsive type.

Diagnosis

The first step in determining if a child has ADHD is to consult with a pediatrician. The pediatrician can make an initial evaluation of the child's developmental maturity compared to other children in his or her age group. The physician should also perform a comprehensive physical examination to rule out any organic causes of ADHD symptoms, such as an overactive thyroid or vision or hearing problems.

If no organic problem can be found, a **psychologist**, **psychiatrist**, neurologist, neuropsychologist, or learning specialist is typically consulted to perform a comprehensive ADHD assessment. A complete medical, family, social, psychiatric, and educational history is compiled from existing medical and school records and from interviews with parents and teachers. Interviews may also be conducted with the child, depending on his or her age. Along with these interviews, several clinical questionnaires may also be used, such as the Conners Rating Scales (Teacher's Questionnaire and Parent's Questionnaire), Child Behavior Checklist (CBCL), and the Achenbach Child Behavior Rating Scales. These inventories provide valuable information on the child's behavior in different settings and situations. In addition, the Wender Utah Rating Scale has been adapted for use in diagnosing ADHD in adults.

It is important to note that mental disorders such as depression and anxiety disorder can cause symptoms similar to ADHD. (Depression can cause attention prob-

A special education teacher helps a student with attention-deficit/hyperactivity disorder with his math assignment. *(Photo Researchers, Inc. Reproduced by permission.)*

lems, and anxiety can cause symptoms similar to hyperactivity.) A complete and comprehensive psychological assessment is critical to differentiate ADHD from other possible mood and behavioral disorders. **Bipolar disorder**, for example, may be misdiagnosed as ADHD.

Public schools are required by federal law to offer free ADHD testing upon request. A pediatrician can also provide a referral to a psychologist or pediatric specialist for ADHD assessment. Parents should check with their insurance plans to see if these services are covered.

Treatment

Therapy that addresses both psychological and social issues (called psychosocial therapy), usually combined with medications, is the treatment approach of choice to alleviate ADHD symptoms.

Medications

Medications known as psychostimulants, such as dextroamphetamine (Dexedrine), **pemoline** (Cylert), and **methylphenidate** (Ritalin), are commonly prescribed to control hyperactive and impulsive behavior and increase attention span. These medications work by stimulating the production of certain neurotransmitters in the brain. These medications are usually well-tolerated and safe in most cases, but possible side effects of stimulants include nervous tics, irregular heartbeat, loss of appetite, and **insomnia**.

For children who do not respond well to stimulant therapy, and for children who clearly suffer from depression as well as ADHD, tricyclic antidepressants (a group of drugs used to treat depression) may be recommended. Examples of these antidepressants include **desipramine** (Norpramin, Pertofane) and **amitriptyline** (Elavil). Reported side effects of these drugs include persistent dry mouth, sedation, disorientation, and cardiac arrhythmia (an abnormal heart rate), particularly with desipramine. Other medications prescribed for ADHD therapy include buproprion (Wellbutrin), an antidepressant; **fluoxetine** (Prozac), an SSRI antidepressant (a group of medications used to treat depression by directing the flow of a neurotransmitter called serotonin); and **carbamazepine** (Tegretol, Atretol), an antiseizure drug. **Clonidine** (Catapres), a medication used to treat high blood pressure, has also been used to control aggression and hyperactivity in some ADHD children, although it should not be used with Ritalin. Because a child's

response to medication will change with age and maturation, ADHD symptoms should be monitored closely and prescriptions adjusted accordingly.

Psychosocial therapies

Behavior modification therapy uses a reward system to reinforce good behavior and task completion and can be implemented both in the classroom and at home. A tangible reward such as a sticker may be given to the child every time he completes a task or behaves in an acceptable manner. A chart may be used to display the stickers and visually illustrate the child's progress. When a certain number of stickers are collected, the child may trade them in for a bigger reward such as a trip to the zoo or a day at the beach. The reward system stays in place until the good behavior becomes ingrained.

A variation of this technique, **cognitive-behavioral therapy**, may work for some children to decrease impulsive behavior by getting the child to recognize the connection between thoughts and behavior, and to change behavior by changing negative thinking patterns.

Individual **psychotherapy** can help an ADHD child build self-esteem, provide a place to discuss worries and anxieties, and help him or her to gain insight into behavior and feelings.

Family therapy may also be beneficial in helping family members develop coping skills and in working through feelings of guilt or anger parents may be experiencing.

ADHD children perform better within a familiar, consistent, and structured routine with positive reinforcements for good behavior and real consequences for bad behavior. Family, friends, and caretakers should all be educated on the special needs and behaviors of the ADHD child so that they can act consistently. Communication between parents and teachers is especially critical to ensuring an ADHD child has an appropriate learning environment.

Alternative treatment

A number of alternative treatments exist for ADHD. Although there is a lack of controlled studies to prove their efficacy, proponents report that they are successful in controlling symptoms in some ADHD patients. Some of the more popular alternative treatments include:

- EEG (electroencephalograph) **biofeedback**. By measuring brainwave activity and teaching the ADHD patient which type of brainwave is associated with attention, EEG biofeedback attempts to train patients to generate the desired brainwave activity.
- Limited sugar intake. However, data indicate that this method does not actually reduce symptoms.
- Relaxation training.

Prognosis

Untreated, ADHD negatively affects a child's social and educational performance and can seriously damage his or her sense of self-esteem. ADHD children have impaired relationships with their peers, and may be looked upon as social outcasts. They may be perceived as slow learners or troublemakers in the classroom. Siblings and even parents may develop resentful feelings towards a child with ADHD.

Some ADHD children also develop a **conduct disorder** problem. For those adolescents who have both ADHD and a conduct disorder, up to 25% go on to develop **antisocial personality disorder** and the criminal behavior, substance abuse, and high rate of **suicide** attempts that can be symptomatic of that disorder. Children diagnosed with ADHD are also more likely to have a learning disorder, a mood disorder such as depression, or an anxiety disorder.

Approximately 70%-80% of ADHD patients treated with stimulant medication experience significant relief from symptoms, at least in the short term. Approximately half of ADHD children seem to "outgrow" the disorder in adolescence or early adulthood; the other half will retain some or all symptoms of ADHD as adults. With early identification and **intervention**, careful **compliance** with a treatment program, and a supportive and nurturing home and school environment, children with ADHD can flourish socially and academically.

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ORGANIZATIONS

American Academy of Child and Adolescent Psychiatry. (AACAP). 3615 Wisconsin Ave. NW, Washington, DC 20016. (202) 966-7300. <http://www.aacap.org>.

Attention Deficit Disorder Association (ADDA). 1788 Second Street, Suite 200, Highland Park, IL 60035. Telephone: (847) 432-ADDA. <http://www.add.org>.

Children and Adults with Attention Deficit Disorder (CH.A.D.D.). 8181 Professional Place, Suite 201, Landover, MD 20785. CHADD National Call Center (800) 233-4050. Web site: <http://chadd.org>.

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Autism

Definition

The term "autism" refers to a cluster of conditions appearing early in childhood. All involve severe impairments in social interaction, communication, imaginative abilities, and rigid, repetitive behaviors. To be considered an autistic disorder, some of these impairments must be manifest before the age of three.

The reference book used by mental health professionals to diagnose mental disorders is the ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM*. The 2000 edition of this reference book (the Fourth Edition Text Revision known as *DSM-IV-TR*) places autism in a category called **pervasive developmental disorders**. All of these disorders are characterized by ongoing problems with mutual social interaction and communication, or the presence of strange, repetitive behaviors, interests, and activities. People diagnosed with these disorders are affected in many ways for their entire lives.

KEY TERMS

Impulse control disorders—Group of disorders characterized by impulsive behavior, such as stealing.

Obsessive-compulsive disorder—Disorder in which the affected individual has an obsession (such as a fear of contamination, or thoughts he or she doesn't like to have and can't control) and feels compelled to perform a certain act to neutralize the obsession (such as repeated handwashing).

Phenylketonuria—(PKU) An inherited disease in which the body cannot metabolize the amino acid phenylalanine properly. If untreated, phenylketonuria can cause mental retardation.

Temporal lobe—Large lobe of each side of the brain that contains a sensory area associated with hearing.

Description

Each child diagnosed with an autistic disorder differs from every other, and so general descriptions of autistic behavior and characteristics do not apply equally to every child. Still, the common impairments in social interaction, communication and imagination, and rigid, repetitive behaviors make it possible to recognize children with these disorders, as they differ markedly from healthy children in many ways.

Many parents of autistic children sense that something is not quite right even when their children are infants. The infants may have feeding problems, dislike being changed or bathed, or fuss over any change in routine. They may hold their bodies rigid, making it difficult for parents to cuddle them. Or, they may fail to anticipate being lifted, lying passively while the parent reaches for them, rather than holding their arms up in return. Most parents of autistic children become aware of the strangeness of these and other behaviors only gradually.

Impairments in social interaction are usually among the earliest symptoms to develop. The most common social impairment is a kind of indifference to other people, or aloofness, even towards parents and close caregivers. The baby may fail to respond to his or her name being called and may show very little facial expression unless extremely angry, upset, or happy. Babies with autism may resist being touched, and appear to be lost in their own world, far from human interaction. Between

seven and 10 months of age, most infants often resist being separated from a parent or well-known caregiver, but these infants may show no disturbance when picked up by a stranger.

Other children with autism may be very passive, although less resistant to efforts by others to interact. However, they do not initiate social interaction themselves. Still others may attempt to engage with adults and peers, but in ways that strike others as inappropriate, or odd.

In adolescence and adulthood, some of the higher-functioning individuals with autistic disorders may appear overly formal and polite. They may react with little spontaneity, as if social interaction doesn't come naturally or easily to them, and so they are trying to follow a pre-determined set of rules.

Some individuals with autism have normal intelligence, and many have special talents in areas such as music or memory. However, individuals with autism may have other mental or emotional problems that co-exist with their autism. Some of these other disorders may include impulse control disorders, **obsessive-compulsive disorder**, mood and anxiety disorders, and **mental retardation**.

Causes and symptoms

Causes

PSYCHOLOGICAL AND FAMILY FACTORS. Although Henry Maudsley, in the late 1800s, was the first **psychiatrist** to focus on very young children with mental disorders, it was the psychiatrist Leo Kanner who coined the phrase "early infantile autism" in 1943. Kanner believed that the parents of children with autistic behaviors were emotionally cold and intellectually distant. He coined the term "refrigerator parents" to describe them. His belief that parental personality and behavior played a powerful role in the development of autistic behaviors left a devastating legacy of guilt and self-blame among parents of autistic children that continues to this day. Recent studies are unequivocal, however, in demonstrating that parents of autistic children are no different from parents of healthy children in their personalities or parenting behaviors. In fact, many families with an autistic child also have one or more perfectly healthy children.

Because autistic children can be extremely sensitive to change, any change within the family situation can be potentially traumatic to the autistic child. A move, divorce, birth of a sibling or other stressors that occur in the lives of most families may evoke a more extreme reaction from an autistic child.

NEUROLOGICAL AND BIOLOGICAL FACTORS. While there is no single neurological abnormality found in children with autistic disorders, some research using non-invasive **brain** imaging techniques such as **magnetic resonance imaging** (MRI) suggests that certain areas of the brain may be involved. Several of the brain areas being researched are known to control emotion and the expression of emotion. These areas include the temporal lobe (large lobe of each side of the brain that contains a sensory area associated with hearing), the limbic system, the cerebellum, the frontal lobe, the amygdala, and the brain stem, which regulates homeostasis (body temperature and heart rate). Recent research has focused particularly on the temporal lobe because of the finding that previously healthy people who sustain temporal lobe damage may develop autistic-like symptoms. In animal research, when the temporal lobe is damaged, social behavior declines, and restless, repetitive motor behaviors are common. When measured by MRI, total brain volume appears to be greater for those with autistic disorders.

Other neurological factors include lesions to the brain, congenital rubella, undiagnosed and untreated phenylketonuria (PKU), tuberous sclerosis, and **Rett's disorder** (a related condition in which the baby develops in an apparently normal manner through age five months, and then begins to lose communicative and social interaction skills). There is also evidence of a higher proportion of perinatal complications (complications arising around the time of giving birth) among children with autistic symptoms. These complications include maternal bleeding after the first trimester and meconium in the amniotic fluid. (Meconium is a substance that accumulates in the bowel of the developing fetus and is discharged shortly after birth.) Some evidence suggests that the use of medications during pregnancy may be related to the development of autistic symptoms. As newborns, children with autistic behaviors show a higher rate of respiratory illness and anemia than healthy children.

ALLERGIES, INFECTIONS, AND IMMUNIZATIONS. Some professionals believe that autistic disorders may be caused by allergies to particular fungi, viral infections, and various foods. No controlled studies have supported these beliefs, but some parents and professionals report improvement when allergens and/or certain foods are eliminated from the diet.

Viral infections of the mother, such as rubella, or of the young child, such as encephalitis, mumps, and measles, occasionally appear to cause autistic disorders. The common childhood immunization series known as MMR (measles, mumps, rubella) has recently come under scrutiny as a possible cause of some autistic conditions.

Teachers assist kindergarten students at a school for autistic children in Salt Lake City, Utah. *(AP/ Wide World Photos. Reproduced by permission.)*

Symptoms

DSM-IV-TR specifies three diagnostic categories, each with four components, that are used to make a **diagnosis** of autistic disorder. These diagnostic categories include impairments in social interaction, communication, and particular patterns of behavior. More information about the individual diagnostic categories and components follows.

SOCIAL INTERACTION. Qualitative impairment in social interaction, as demonstrated by at least two of the following:

- impairment in the use of nonverbal behaviors such as eye contact, facial expression, body posture, and gestures used for social interaction
- failure to develop age-appropriate peer relationships
- lack of attempts to share pleasure, activities, interests, or achievements with other people (by failing to bring items of interest to a parent, or pointing out animals or objects, for example)
- inability to respond to social situations or other people's emotions with empathy or a concerned attitude

COMMUNICATION. Qualitative impairments in communicating in at least one of the following four areas:

- lack of, or delay in development of spoken language, without attempts to communicate through alternative means such as gestures or mime
- in individuals who do speak, severe impairment in the ability to initiate or sustain a conversation with others
- repetitive and stereotyped use of language, or use of words in unusual, idiosyncratic ways
- failure to show imaginative play, such as make-believe or social imitative play appropriate to developmental level

BEHAVIOR. Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities, as demonstrated by at least one of the following:

- unusual and overly absorbing preoccupation with one or more interests or activities
- a need for rigid adherence to specific routines or rituals in daily life
- stereotyped and repetitive motor behaviors using parts of the body such as fingers or hands, or the whole body
- persistent preoccupation with parts of objects

Demographics

Autistic disorders strike families of all racial, ethnic, and social backgrounds. These disorders are estimated to affect approximately four children in 10,000. Other estimates place the number affected at between 1 in 500 and 1 in 2,500 Americans. Autistic disorder occurs four times more frequently in boys than girls. Several surveys have shown that between two and four percent of siblings of autistic children also have autistic disorder. This rate is 50 times greater than in the general population. Among pairs of identical twins in which one child has autism, in 36% of the pairs, the other twin has autism as well. Among fraternal twins, there is no similar correlation. Some studies indicate that even among family members who are not diagnosed as autistic, there tends to be a higher-than-average rate of language and other cognitive problems. As many as 25% of autistic children develop epileptic **seizures** later in life, usually during adolescence. This symptom appears mostly in those who are also mentally retarded.

Recently, professionals have reported observing increasing numbers of children with autistic disorders. While no studies confirm this observation, there are three possible reasons why it appears so. First, the definition of "autism" and "autistic disorders" has widened considerably since the first case reports by Leo Kanner in 1943. The *DSM-IV-TR* definition currently in use includes a far greater range of behaviors than earlier definitions of autism. Second, there has been an increasing awareness of the existence of autism and autistic disorders among the general public and among health professionals, making a child with symptoms of autism much more likely to be diagnosed than in years past. Finally, it is possible that there is an actual increase in the number of children born with one of these disorders.

Diagnosis

Because young infants are so limited in their range of behavior, autistic disorders are generally discovered gradually, and rarely diagnosed before the age of two or three. Parents may not realize that their baby's behavior is different from that of other infants until he or she reaches an age where a wide range of behaviors are typically displayed. Most doctors may attempt to reassure concerned parents of infants under two years that their children are "normal," or will "grow out of" a disturbing behavior, because many children do. At the time that speech and language usually develop, parents are more likely to observe that their autistic child is not at the same level as other children his age. Once the child is old enough to play with other children, it becomes more apparent that the autistic child either isn't interested in doing so, or does so in strange, unusual ways that differ from most children of the same age. Motor development may also appear unusual, with repetitive motions such as spinning, self-injurious behaviors such as headbanging, and rocking back and forth, giving the parents strong clues that their child behaves differently from others.

The child who continues to display unusual behaviors at about the age of two years would most likely receive a referral from the pediatrician to a child psychiatrist or to an early **intervention** program with a multidisciplinary staff including psychiatrists, psychologists, and **social workers**. These professionals would be the ones to diagnose autistic disorder, and, ideally, offer an early intervention program simultaneously. In order to reach the diagnosis, the professional(s) would observe the child both with and without parents present, interview the parents about the pregnancy, birth, siblings, family history, and early behaviors, and an assessment like the Bayley Scales of Infant Development might be administered.

Differential diagnosis

Differential diagnosis is the process of distinguishing one disorder from other similar disorders. Because there are currently no medical tests (such as a blood test) to detect autism, the diagnosis is often established by ruling out other disorders.

MENTAL RETARDATION. It is estimated that approximately 40% to 60% of children with autistic disorders show some degree of mental retardation ranging from mild to profound. It is possible for a child to have both conditions. What distinguishes children with mental retardation who do not have autistic symptoms from those who do is evenness of development. Children with mental retardation tend to exhibit a more even level of functioning in all areas, whereas autistic children tend to exhibit extreme variability within areas and between areas. Children with autistic disorders show uneven development in areas such as motor, language, and social skills. A child with autism may have high-level cognitive functioning in one area, but low-level cognitive functioning in another area, for example. Or a child with autism may exhibit delayed cognitive development, but normal motor skills development. For this reason, autism is often referred to as a "spectrum disorder" because of the large spectrum or range of variability in symptoms and functioning. Also, many children with mental retardation relate well to people and enjoy social connection, which is rare for autistic children.

LANGUAGE DISORDER. Children with autistic disorders may appear similar in some ways to children with language disorders. Unlike autistic children, however, children with language disorders have normal responses

to most people, situations, and objects. They make eye contact and show interest in peer and adult relationships.

CHILDHOOD SCHIZOPHRENIA. Schizophrenia is a disturbance of emotion and thought processes that rarely occurs in young children. When it does, it is characterized by **hallucinations** and **delusions**— seeing and hearing things that are not there, for example. These are not symptoms that appear among autistic children.

DEGENERATIVE ORGANIC BRAIN DISORDER. This is an extremely rare condition that may at first appear similar to autistic disorders. In degenerative organic brain disorder, the child begins to develop normally. But over time, speech, language, motor skills and other age-appropriate behaviors disintegrate and do not return. The disintegration is progressive. In children with autistic disorders, some children may begin to develop words and language and then lose them at around eighteen months. However, with appropriate education, these skills can be relearned and surpassed by the autistic child.

Treatments

Autistic disorders cannot be cured, but children who have these disorders can make considerable progress in all areas of life. Depending upon the level of intellectual function, it is possible for some children with autism to become functioning, semi-independent adults capable of working and enjoy some social relationships. Parenting a child with autism can be extremely challenging, however, and many families find **support groups** to be helpful. Both medication and psychosocial therapies (therapies that address both psychological and social issues) can help ameliorate troubling symptoms. Education is key for helping these children learn socially acceptable behaviors, decreasing odd mannerisms and behaviors, and increasing appropriate verbal and non-verbal language skills.

Education

Most educational programs for children with autistic disorders involve small, specialized classes with teachers specially trained to work with autistic children. Often, these children are educated in special schools that have extended school years rather than lengthy summer vacations. Research has shown that autistic children need regular, daily structure and routine, and they maintain their skills best when there are not frequent disruptions of their daily school program.

One method that has been used extensively both within the classroom and at home is a **behavior modification** method known as "Applied Behavior Analysis," or ABA. Specially trained teachers break down large goals into small steps that are taught and repeated until the child masters each one. Slowly, step by step, more appropriate patterns of behavior and communication are formed or "shaped" in this way. Positive **reinforcement** is used in many forms such as praise, for those children who are motivated by it, time permitted to engage in a favorite activity, or a small favored food item. For ABA to be most effective, parents need to be trained to use these same skills to continue the work at home.

Medications

Although no one drug is helpful to children with autistic disorders, several medications are currently used, along with education, to reduce severe temper tantrums and destructive aggression, self-injurious behaviors, hyperactivity, and strange, repetitive behaviors. Medications may also help the autistic child become more receptive to learning and relating to others. Some of the medications commonly used today include **risperidone** (Risperdal), and **haloperidol** (Haldol). Although there are side effects associated with these medications, careful dosing and use of other medications to counteract side effects often enable the autistic child to function more effectively.

Non-conventional treatments

One non-conventional and experimental treatment for autism is the use of secretin, a hormone produced in the small intestine that stimulates the pancreas to release sodium bicarbonate and other digestive enzymes. Some researchers think that children with autistic disorders do not produce enough of this hormone, and that the lack of sufficient secretin may be the reason why children with autistic disorders suffer so frequently from digestive problems. There are some reports of treating autistic children with secretin that indicate improvement not only in digestion, but in eye contact, alertness, and the ability to learn.

Another non-conventional, experimental treatment involves *Candida albicans*, the technical term for a common yeast that is found in the human body. Some scientists believe that an overgrowth of this yeast may cause or worsen autism. Some reports indicate that children treated with anti-yeast medications improve in eye contact, social abilities, language skills, concentration, and sleep, and that they show a reduction in aggressive and hyperactive behavior.

An additional non-conventional treatment being researched for autism is a nutritional supplement, Vitamin B6. Some experts believe that Vitamin B6 holds promise for reducing autistic symptoms and helping autistic children progress in all areas. It may be combined with magnesium and the combination appears to have no known side effects. Improvements attributed to these

supplements in some studies include enhanced language, eye-contact, and behaviors, as well as more normal brain activity and improved immune system functioning.

These treatments remain outside mainstream medicine, however, and research is ongoing as to their efficacy. Parents interested in these therapies may wish to discuss them with their child's health care team.

Prognosis

Autistic disorders follow a continuous course throughout life. Autistic individuals with higher levels of intelligence may become able to work and live independently or, more frequently, semi-independently. This is especially true for those with IQ scores of 70 or higher. One in six children with autism becomes a well-adjusted adult. Another one out of six achieves a fair degree of adjustment in adult life. Others may never be able to leave the structured environment of home or, later, special group home placement. During adolescence, sexual feelings emerge that cannot usually be handled appropriately by the autistic teen. Supervision throughout life is needed for the majority of individuals diagnosed with these disorders.

Prevention

At present, no specific means of preventing autistic disorders exist. Because of an elevated likelihood of giving birth to more than one autistic child exists, genetic counseling is recommended.

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Families Working Together. 12400 Cypress Avenue, Space 20, Chino, CA 91710. <http://www.ucddfam.com>.

F.E.A.T (Families for Early Autism Treatment). PO Box 255722, Sacramento, CA 95865-1536. <http://www.feat.org>.

Barbara S. Sternberg, Ph.D.

Aventyl *see* **Nortriptyline**

Aversion therapy

Definition

Aversion therapy is a form of behavior therapy in which an aversive (causing a strong feeling of dislike or disgust) stimulus is paired with an undesirable behavior in order to reduce or eliminate that behavior.

Purpose

As with other behavior therapies, aversion therapy is a treatment grounded in learning theory—one of its basic principles being that all behavior is learned and that undesirable behaviors can be unlearned under the right circumstances. Aversion therapy is an application of the branch of learning theory called classical conditioning. Within this model of learning, an undesirable behavior, such as a deviant sexual act, is matched with an unpleasant (aversive) stimulus. The unpleasant feelings or sensations become associated with that behavior, and the behavior will decrease in frequency or stop altogether. Aversion therapy differs from those types of behavior therapy based on principles of operant conditioning. In operant therapy, the aversive stimulus, usually called punishment, is presented after the behavior rather than together with it.

The goal of aversion therapy is to decrease or eliminate undesirable behaviors. Treatment focuses on changing a specific behavior itself, unlike insight-oriented approaches that focus on uncovering unconscious motives in order to produce change. The behaviors that have been treated with aversion therapy include such addictions as alcohol abuse, drug abuse, and smoking; pathological gambling; sexual deviations; and more benign habits— including writer's cramp. Both the type of behavior to be changed and the characteristics of the aversive stimulus influence the treatment—which may

be administered in either outpatient or inpatient settings as a self-sufficient **intervention** or as part of a multimodal program. Under some circumstances, aversion therapy may be self-administered.

Precautions

A variety of aversive stimuli have been used as part of this approach, including chemical and pharmacological stimulants as well as electric shock. Foul odors, nasty tastes, and loud noises have been employed as aversive stimuli somewhat less frequently. The chemicals and medications generate very unpleasant and often physically painful responses. This type of aversive stimulation may be risky for persons with heart or lung problems because of the possibility of making the medical conditions worse. Patients with these conditions should be cleared by their doctor first. Often, however, the more intrusive aversive stimuli are administered within inpatient settings under medical supervision. An uncomfortable but safe level of electric (sometimes called faradic) shock is often preferred to chemical and pharmacological aversants because of the risks that these substances involve.

In addition to the health precautions mentioned above, there are ethical concerns surrounding the use of aversive stimuli. There are additional problems with patient acceptance and negative public perception of procedures utilizing aversants. Aversion treatment that makes use of powerful substances customarily (and intentionally) causes extremely uncomfortable consequences, including nausea and vomiting. These effects may lead to poor **compliance** with treatment, high dropout rates, potentially hostile and aggressive patients, and public relations problems. Social critics and members of the general public alike often consider this type of treatment punitive and morally objectionable. Although the scenes were exaggerated, the disturbing parts of the Stanley Kubrick film *A Clockwork Orange* that depicted the use of aversion therapy to reform the criminal protagonist, provide a powerful example of society's perception of this treatment.

Parents and other advocates for the mentally retarded and developmentally disabled have been particularly vocal in their condemnation of behavior therapy that uses aversive procedures in general. Aversive procedures are used within a variety of **behavior modification** strategies and that term is sometimes confused with the more specific technique of aversion therapy. Aversive procedures are usually based on an operant conditioning model that involves punishment. Advocates for special patient populations believe that all aversive procedures are punitive, coercive, and use unnecessary amounts of control and manipulation to modify behavior. They call for therapists to stop using aversive stimuli, noting that positive, non-aversive, behavioral-change strategies are available. These strategies are at least as, if not more, effective than aversive procedures.

KEY TERMS

Aversion—A strong feeling of dislike or disgust. Aversion therapy makes use of this feeling to reduce or eliminate an undesirable behavior. Chemicals or medications used to produce unpleasant effects are called aversants.

Classical conditioning—In psychology, a process in which a previously neutral stimulus eventually produces a specific response by being paired repeatedly with another stimulus that produces that response. The best-known example of classical conditioning is Pavlov's dogs, who were conditioned to salivate when they heard a bell ring (the previously neutral stimulus) because the sound had been paired repeatedly with their feeding time.

Compliance—In medicine or psychiatry, cooperation with a treatment plan or schedule of medications.

Detoxification—A process in which the body is allowed to free itself of a drug while the symptoms of withdrawal are treated. It is the primary step in any treatment program for drug or alcohol abuse.

Emetic—A medication intended to cause vomiting. Emetics are sometimes used in aversion therapy in place of electric shock. Their most common use in mainstream medicine is in treating accidental poisoning.

Faradic—A type of discontinuous alternating electric current sometimes used in aversion therapy. It is named for Michael Faraday, an eminent British physicist.

Protocol—A plan for carrying out a scientific study or a patient's course of treatment.

Stimulus—Something that incites or moves a person to thought, emotion, or action. In mainstream psychotherapy, a stimulus can be anything from a certain picture or image to a smell, a sound, or a word or idea. In aversion therapy, the stimulus is typically a mild electric shock or a medication that produces unpleasant results.

Description

A patient who consults a behavior therapist for aversion therapy can expect a fairly standard set of procedures. The therapist begins by assessing the problem, most likely measuring its frequency, severity, and the environment in which the undesirable behavior occurs. Although the therapeutic relationship is not the focus of treatment for the behavior therapist, therapists in this tradition believe that good rapport will facilitate a successful outcome. A positive relationship is also necessary to establish the patient's confidence in the rationale for exposing him or her to an uncomfortable stimulus. The therapist will design a treatment protocol and explain it to the patient. The most important choice the therapist makes is the type of aversive stimulus to employ. Depending upon the behavior to be changed, the preferred aversive stimulus is often electric stimulation delivered to the forearm or leg. This aversive stimulus should not be confused with **electroconvulsive therapy** (ECT), which is delivered to the **brain** to treat depression. Mild but uncomfortable electric shocks have several advantages over chemical and pharmacological stimuli. A great many laboratory research studies using animal and human subjects have used electrical shock and its characteristics are well known. In addition, it has been widely used in clinical settings. Electric shock is easy to administer, and the level of intensity can be preselected by the patient. The stimulation can be precisely controlled and timed. The equipment is safe, battery-powered, suitable for outpatients, portable, easy to use, and can be self-administered by the patient when appropriate.

Case example #1: What would a treatment protocol look like for a relatively well-adjusted patient specifically requesting aversion therapy on an outpatient basis to reduce or eliminate problem gambling behavior? The therapist begins by asking the patient to keep a behavioral diary. The therapist uses this information both to understand the seriousness of the problem and as a baseline to measure whether or not change is occurring during the course of treatment. Because electric shock is easy to use and is acceptable to the patient, the therapist chooses it as the aversive stimulus. The patient has no medical problems that would preclude the use of this stimulus. He or she fully understands the procedure and consents to treatment. The treatment is conducted on an outpatient basis with the therapist administering the shocks on a daily basis for the first week in the office, gradually tapering to once a week over a month. Sessions last about an hour. A small, battery-powered electrical device is used. The electrodes are placed on the patient's wrist. The patient is asked to preselect a level of shock that is uncomfortable but not too painful. This shock is then briefly and repeatedly paired with stimuli (such as slides of the race track, betting sheets, written descriptions of gambling) that the patient has chosen for their association with his or her problem gambling. The timing, duration, and intensity of the shock are carefully planned by the therapist to assure that the patient experiences a discomfort level that is aversive and that the conditioning effect occurs.

After the first or second week of treatment, the patient is provided with a portable shocking device to use on a daily basis for practice at home to supplement office treatment. The therapist calls the patient at home to monitor compliance as well as progress between office sessions. The conditioning effect occurs, the discomfort from the electric shock becomes associated with the gambling behavior, the patient reports loss of desire and stops gambling. Booster sessions in the therapist's office are scheduled once a month for six months. A minor relapse is dealt with through an extra office visit. The patient is asked to administer his or her own booster sessions on an intermittent basis at home and to call in the future if needed.

Case example #2: What would the treatment protocol look like for an alcohol-dependent patient with an extensive treatment history including multiple prior life-threatening relapses? The patient who is motivated to change but has not experienced success in the past may be considered a candidate for aversion therapy as part of a comprehensive inpatient treatment program. The treating therapist assesses the extent of the patient's problem, including drinking history, prior treatments and response, physical health, and present drinking pattern. Patients who are physically addicted to alcohol and currently drinking may experience severe withdrawal symptoms and may have to undergo **detoxification** before treatment starts. When the detoxification is completed, the patient is assessed for aversion therapy. The therapist's first decision is what type of noxious stimulus to use, whether electrical stimulation or an emetic (a medication that causes vomiting). In this case, when the patient's problem is considered treatment-resistant and a medically-monitored inpatient setting is available, an emetic may be preferable to electric shock as the aversive stimulus. There is some research evidence that chemical aversants lead to at least short-term avoidance of alcohol in some patients. An emetic is "biologically appropriate" for the patient in that it affects him or her in the same organ systems that excessive alcohol use does. The procedure is fully explained to the patient, who gives **informed consent**.

During a ten-day **hospitalization**, the patient may receive aversion therapy sessions every other day as part of a comprehensive treatment program. During the treatment sessions, the patient is given an emetic intra-

venously under close medical supervision and with the help of staff assistants who understand and accept the theory. Within a few minutes following administration, the patient reports beginning to feel sick. To associate the emetic with the sight, smell and taste of alcohol, the patient is then asked to take a sip of the alcoholic beverage of his or her choice without swallowing. This process is repeated over a period of 30–60 minutes as nausea and vomiting occur. As the unpleasant effects of the emetic drug become associated with the alcoholic beverage, the patient begins to lose desire for drinking. Aversion therapy in an inpatient program is usually embedded within a comprehensive treatment curriculum that includes **group therapy** and such **support groups** as AA, couples/family counseling, **social skills training**, stress management, instruction in problem solving and conflict resolution, health education and other behavioral change and maintenance strategies. Discharge planning includes an intensive outpatient program that may include aversive booster sessions administered over a period of six to twelve months, or over the patient's lifetime.

Preparation

Depending upon his/her customary practice, a therapist administering aversion therapy may establish a behavioral contract defining the treatment, objectives, expected outcome, and what will be required of the patient. The patient may be asked to keep a behavioral diary to establish a baseline measure of the behavior targeted for change. The patient undergoing this type of treatment should have enough information beforehand to give full consent for the procedure. Patients with medical problems or who are otherwise vulnerable to potentially damaging physical side effects of the more intense aversive stimuli should consult their primary care doctor first.

Aftercare

Patients completing the initial phase of aversion therapy are often asked by the therapist to return periodically over the following six to twelve months or longer for booster sessions to prevent relapse.

Risks

Patients with cardiac, pulmonary, or gastrointestinal problems may experience a worsening of their symptoms, depending upon the characteristics and strength of the aversive stimuli. Some therapists have reported that patients undergoing aversion therapy, especially treatment that uses powerful chemical or pharmacological aversive stimuli, have become negative and aggressive.

Normal results

Depending upon the objectives established at the beginning of treatment, patients successfully completing a course of aversion therapy can expect to see a reduction or cessation of the undesirable behavior. If they practice relapse prevention techniques, they can expect to maintain the improvement.

Abnormal results

Some clinicians have reported that patients undergoing aversive treatment utilizing electric shocks have experienced increased anxiety and anxiety-related symptoms that may interfere with the conditioning process as well as lead to decreased acceptance of the treatment. As indicated above, a few clinicians have reported a worrisome increase in hostility among patients receiving aversion therapy, especially those undergoing treatment using chemical aversants. Although aversion therapy has some adherents, lack of rigorous outcome studies demonstrating its effectiveness, along with the ethical objections mentioned earlier, have generated numerous opponents among clinicians as well as the general public. These opponents point out that less intrusive alternative treatments, such as **covert sensitization**, are available.

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Avoidant personality disorder

Definition

Avoidant personality disorder is one of several **personality disorders** listed in the newest edition of the standard reference guide to mental disorders ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM-IV-TR*. It is characterized by marked avoidance of both social situations and close interpersonal relationships due to an excessive fear of rejection by others. Persons with this disorder exhibit feelings of inadequacy, low self-esteem, and mistrust toward others.

Description

People who are diagnosed with avoidant personality disorder desire to be in relationships with others but lack the skills and confidence that are necessary in social interactions. In order to protect themselves from anticipated criticism or ridicule, they withdraw from other people. This avoidance of interaction tends to isolate them from meaningful relationships, and serves to reinforce their nervousness and awkwardness in social situations.

The behavior of people with avoidant personality disorder is characterized by social withdrawal, shyness, distrustfulness, and emotional distance. These people tend to be very cautious when they speak, and they convey a general impression of awkwardness in their manner. Most are highly self-conscious and self-critical about their problems relating to others.

Causes and symptoms

Causes

The cause of avoidant personality disorder is not clearly defined, and may be influenced by a combination of social, genetic, and biological factors. Avoidant personality traits typically appear in childhood, with signs of excessive shyness and fear when the child confronts new people and situations. These characteristics are also developmentally appropriate emotions for children, however, and do not necessarily mean that a pattern of avoidant personality disorder will continue into adulthood. When shyness, unfounded fear of rejection, hypersensitivity to criticism, and a pattern of social avoidance persist and intensify through adolescence and young adulthood, a **diagnosis** of avoidant personality disorder is often indicated.

Many persons diagnosed with avoidant personality disorder have had painful early experiences of chronic parental criticism and rejection. The need to bond with the rejecting parents makes the avoidant person hungry for relationships but their longing gradually develops into a defensive shell of self-protection against repeated parental criticisms. Ridicule or rejection by peers further reinforces the young person's pattern of social withdrawal and contributes to their fear of social contact.

Symptoms

DSM-IV-TR specifies seven diagnostic criteria for avoidant personality disorder:

- The person avoids occupational activities that require significant interpersonal contact. Job interviews or promotions may be turned down because the person's own perceptions of his or her abilities do not match the job description.
- The person is reluctant to participate in social involvement without clear assurance that they will be accepted. People with this disorder assume other people are not safe to trust until proven otherwise. Others must offer repeated support and encouragement in order to persuade them to participate in a social event.
- The person fears being shamed or ridiculed in close relationships. As a result, people with this disorder become overly alert to behavioral cues that may indicate disapproval or rejection. They will flee a situation in which they believe that others might turn against them.
- The person is preoccupied with being criticized or rejected. Much mental and physical energy is spent brooding about and avoiding situations perceived as "dangerous."
- The person is inhibited in unfamiliar social situations due to feelings of inadequacy. Low self-esteem undermines their confidence in meeting and conversing with new acquaintances.
- The person regards him- or herself as socially inept. This self-disparagement is especially apparent when the person must make social contacts with strangers. People with avoidant personality disorder perceive themselves as unappealing or inferior to others.
- The person is reluctant to take social risks, in order to avoid possible humiliation. Avoidant people seek interactions that promise the greatest amount of acceptance while minimizing the likelihood of embarrassment or rejection. They might go to a school dance, for example, but remain in one corner chatting with close friends rather than going out on the dance floor with someone they do not know well.

Demographics

Avoidant personality disorder appears to be as frequent in males as in females. It affects between 0.5% and 1.0% of adults in the general North American population, but it has been diagnosed in approximately 10% of clinical outpatients.

Diagnosis

Many individuals exhibit some avoidant behaviors at one point or another in their lives. Occasional feelings of self-doubt and fear in new and unfamiliar social or personal relationships are not unusual, nor are they unhealthy, as these situations may trigger feelings of inadequacy and the wish to hide from social contact in even the most self-confident individuals. An example would be the anxious hesitancy of a new immigrant in a country with a different language and strange customs. Avoidant characteristics are regarded as meeting the diagnostic criteria for a personality disorder only when they: begin to have a long-term negative impact on the affected person; lead to functional impairment by significantly altering occupational choice or lifestyle, or otherwise impacting quality of life; and cause significant emotional distress.

Avoidant personality disorder can occur in conjunction with other social phobias, mood and anxiety disorders, and personality disorders. The diagnosis may be complicated by the fact that avoidant personality disorder may be either the cause or result of other mood and anxiety disorders. For example, individuals who suffer from **major depressive disorder** may begin to withdraw from social situations and experience feelings of worthlessness, symptoms that are also prominent features of avoidant personality disorder. On the other hand, the insecurity and isolation that are symptoms of avoidant personality disorder can trigger feelings of depression.

The characteristics of avoidant personality disorder may resemble those found in both schizoid and schizotypal personality disorders. Persons with any of these disorders are prone to social isolation. Those diagnosed with avoidant personality disorder, however, differ from those with schizoid or schizotypal disorder, because they want to have relationships with others but are prevented by their social inadequacies. Persons diagnosed with schizoid and schizotypal personality disorders, on the other hand, usually prefer social isolation.

Personality disorders are usually diagnosed following a complete medical history and an interview with the patient. Although there are no laboratory tests for personality disorders, the doctor may give the patient a physical examination to rule out the possibility that a general medical condition is affecting the patient's behavior. For example, people with disorders of the digestive tract may avoid social occasions for fear of a sudden attack of diarrhea or the need to vomit. If the interview with the patient suggests a diagnosis of avoidant personality disorder, the doctor may administer a diagnostic questionnaire or another type of assessment tool.

KEY TERMS

Cognitive restructuring—An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

Monoamine oxidase inhibitors (MAOIs)—A group of antidepressant drugs that decreases the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood. MAOIs are also used in the treatment of avoidant personality disorder.

Supportive—An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or educational approaches to treatment.

Assessment tools helpful in diagnosing avoidant personality disorder include:

- **Minnesota Multiphasic Personality Inventory** (MMPI-2)
- Millon Clinical Multiaxial Inventory (MCMI-II)
- Rorschach Psychodiagnostic Test
- **Thematic Apperception Test** (TAT)

Treatments

The general goal of treatment in avoidant personality disorder is improvement of self-esteem and confidence. As the patient's self-confidence and social skills improve, he or she will become more resilient to potential or real criticism by others.

Psychodynamically oriented therapies

These approaches are usually supportive; the therapist empathizes with the patient's strong sense of shame and inadequacy in order to create a relationship of trust. Therapy usually moves slowly at first because persons with avoidant personality disorder are mistrustful of others; treatment that probes into their emotional state too quickly may result in a more protective withdrawal by

the patient. As trust is established and the patient feels safer discussing details of his or her situations, he or she may be able to draw important connections between their deeply felt sense of shame and their behavior in social situations.

Cognitive-behavioral therapy

Cognitive-behavioral therapy (CBT) may be helpful in treating individuals with avoidant personality disorder. This approach assumes that faulty thinking patterns underlie the personality disorder, and therefore focuses on changing distorted cognitive patterns by examining the validity of the assumptions behind them. If a patient feels he is inferior to his peers, unlikable, and socially unacceptable, a cognitive therapist would test the reality of these assumptions by asking the patient to name friends and family who enjoy his company, or to describe past social encounters that were fulfilling to him. By showing the patient that others value his company and that social situations can be enjoyable, the irrationality of his social fears and insecurities are exposed. This process is known as "cognitive restructuring."

Group therapy

Group therapy may provide patients with avoidant personality disorder with social experiences that expose them to feedback from others in a safe, controlled environment. They may, however, be reluctant to enter group therapy due to their fear of social rejection. An empathetic environment in the group setting can help each member overcome his or her social anxieties. **Social skills training** can also be incorporated into group therapy to enhance social awareness and feedback.

Family and marital therapy

Family or couple therapy can be helpful for a patient who wants to break out of a family pattern that reinforces the avoidant behavior. The focus of marital therapy would include attempting to break the cycle of rejection, criticism or ridicule that typically characterizes most avoidant marriages. Other strategies include helping the couple to develop constructive ways of relating to one another without shame.

Medications

The use of monoamine oxidase inhibitors (MAOIs) has proven useful in helping patients with avoidant personality disorder to control symptoms of social unease and experience initial success. The major drawback of these medications is limitations on the patient's diet. People taking MAOIs must avoid foods containing a substance known as tyramine, which is found in most cheeses, liver, red wines, sherry, vermouth, beans with broad pods, soy sauce, sauerkraut, and meat extracts.

Prognosis

Higher-functioning persons with avoidant personality disorder can generally be expected to improve their social awareness and improve their social skills to some degree. But because of the significant social fear and deep-seated feelings of inferiority, these patterns usually do not change dramatically. Lower-functioning persons are likely to drop out of treatment if they become too anxious.

Prevention

Since avoidant personality disorder usually originates in the patient's family of origin, the only known preventive measure is a nurturing, emotionally stimulating and expressive family environment.

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American Psychiatric Association. 1400 K Street NW, Washington D.C. 20005. <http://www.psych.org>.

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B

Barbiturates

Definition

Barbiturates are a large class of drugs, consisting of many different brand name products with generic equivalents, that are used primarily for mild sedation, general anesthesia, and as a treatment for some types of epilepsy. One barbiturate, butalbital, exists only as a component of several headache preparations. The most common members of the barbiturate family are phenobarbital (Luminal)), pentobarbital (Nembutal), amobarbital (Amytal), secobarbital (Seconal), thiopental (Pentothal), methohexital (Brevital), and butalbital (component of Fiorinal and Fioricet). They exist in numerous formulations and strengths.

Purpose

Barbiturates are used to sedate patients prior to surgery as well as to produce general anesthesia, to treat some forms of epilepsy, and to treat simple and migraine headache. These drugs are highly addictive and are often abused as recreational drugs. Although still commercially available, barbiturates such as secobarbital, pentobarbital, and amobarbital are no longer routinely recommended for the treatment of **insomnia** because of their ability to cause dependence, tolerance, and withdrawal. These drugs also have significant side effects when taken in large doses and can cause respiratory failure and death.

Description

The therapeutic effects of barbiturates as a class of drugs are all related to their ability to sedate and, at high enough doses and with certain preparations, to induce sleep. All barbiturates also have anticonvulsant properties although phenobarbital is the preferred barbiturate to treat epilepsy because it can produce anticonvulsant effects at levels low enough not to cause extreme sedation or sleep.

Recommended dosage

The typical dose of phenobarbital for use as an anticonvulsant in adults is 50–100 mg given two to three times per day. When a series of serious **seizures** known as status epilepticus occurs, adults are usually first given 300–800 mg intravenously (directly into the vein) followed by 120–240 mg every 20 minutes up to a maximum of 1000–2000 mg. For sedation, adults are given 30–120 mg per day divided into two or three doses. For sedation before surgery, 100–200 mg are given in an intramuscular injection (a shot) about one hour before the surgery.

The typical dose for an anticonvulsant effect in newborns is 2 mg to 4 mg of phenobarbital per kilogram of body weight per day. In infants, this dose is 5 mg to 8 mg per kilogram of body weight per day. In children one to five years of age, the dose is 6 mg to 8 mg per kilogram of body weight per day. In children aged five to 12 years, the dose is 4 mg to 6 mg per kilogram of body weight per day. All of these doses are given in one to two divided doses per day.

In newborns with status epilepticus, phenobarbital 15 mg to 20 mg per kilogram of body weight is given in a single or divided dose. Infants and children are given 10 mg to 20 mg per kilogram of body weight in a single or divided dose. They may also receive 5 mg per kilogram of body weight every 15 to 30 minutes up to a maximum of 40 mg per kilogram body weight. For anesthesia before surgery, 1 mg to 3 mg per kilogram of body weight is given about one hour before the surgery.

The typical dose of butalbital, as a component of headache preparations such as Fiorinal or Fioricet, is 50-100 mg administered every four to six hours as needed.

Precautions

Children who are hyperactive should not receive phenobarbital or other barbiturates. Some children paradoxically become stimulated and hyperactive after receiving barbiturates.

KEY TERMS

Addiction—A compulsive need for, and use of, a habit-forming substance or behavior.

Anticonvulsant—A medication used to control abnormal electrical activity in the brain that causes seizures.

Corticosteroids—Any one of a number of hormonal steroid compounds that are derived from the adrenal gland.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness.

Dependence—The adaptation of neurons and other physical processes to the use of a drug, followed by withdrawal symptoms when the drug is removed; physiological and/ or psychological addiction.

Hyperactive—Behavior disturbances, usually in children and adolescents, that involves impulsiveness, low levels of concentration, and distractibility.

Intramuscular—An injection that is given into a muscle.

Monoamine oxidase inhibitors (MAOIs)—A group of antidepressant drugs that decreases the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Status epilepticus—Series of grand mal epileptic seizures that may occur when the patient is asleep or awake and involves diminished consciousness.

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

The use of barbiturates in the elderly (over age 65) should be watched closely. Elderly patients must be carefully monitored for confusion, agitation, **delirium**, and excitement if they take barbiturates. Barbiturates should be avoided in elderly patients who are receiving drugs for other mental disorders such as **schizophrenia** or depression.

Women should not use barbiturates during pregnancy unless they are necessary to control seizures. In these cases, they should take the minimum amount to control the seizures. Barbiturate use by pregnant women has been associated with increased risk of fetal damage and bleeding during childbirth. Women who are breast-feeding should not take barbiturates because these drugs enter the breast milk and may cause serious side effects in the nursing baby.

Long-term barbiturate use should be avoided unless there is a strong medical need, as in the case of epilepsy, because of the potential for **addiction**, dependence, tolerance, and withdrawal. People should not drive, operate heavy equipment, or perform other hazardous activities requiring mental alertness while taking barbiturates.

Side effects

The most common side effect of barbiturate use is drowsiness. Less common side effects include agitation, confusion, breathing difficulties, abnormally low blood pressure, nausea, vomiting, constipation, lower body temperature, decreased heart rate, movement difficulty, nightmares, anxiety, nervousness, mental depression, and dizziness. Rare but reported side effects include fever, headache, anemia, allergic reactions, and liver damage.

Interactions

Patients should always tell their doctor and dentist when they are taking barbiturates. Barbiturates should generally not be taken with other drugs used to treat mental disorders.

There are a number of drugs that barbiturates should not be combined with because the barbiturates may increase the metabolism of these drugs and thus, reduce the amount of these drugs available to be of benefit. These drugs include oral corticosteroids such as prednisolone, methylprednisolone, prednisone, or dexamethasone, estrogen and oral contraceptives, blood-thinning medications such as warfarin (Coumadin), the antibiotic doxycycline (Vibramycin), and anticonvulsants such as phenytoin (Dilantin).

Barbiturates should not be combined with alcohol because the combination produces additive depressant effects in the central nervous system.

Barbiturates may lower the amount of absorption of the vitamins D and K.

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Mark Mitchell, M.D.

Beck Depression Inventory

Definition

The Beck Depression Inventory (BDI) is a series of questions developed to measure the intensity, severity, and depth of depression in patients with psychiatric diagnoses. Its long form is composed of 21 questions, each designed to assess a specific symptom common among people with depression. A shorter form is composed of seven questions and is designed for administration by primary care providers. Aaron T. Beck, a pioneer in cognitive therapy, first designed the BDI.

Purpose

The BDI was originally developed to detect, assess, and monitor changes in depressive symptoms among people in a mental health care setting. It is also used to detect depressive symptoms in a primary care setting. The BDI usually takes between five and ten minutes to complete as part of a psychological or medical examination.

Precautions

The BDI is designed for use by trained professionals. While it should be administered by a knowledgeable mental health professional who is trained in its use and interpretation, it is often self-administered.

Description

The BDI was developed in 1961, adapted in 1969, and copyrighted in 1979. A second version of the inventory (BDI-II) was developed to reflect revisions in the Fourth Edition Text Revision of the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*, a handbook that mental health professionals use to diagnose mental disorders).

KEY TERMS

Reliability—The ability of a test to yield consistent, repeatable results.

Validity—The ability of a test to measure accurately what it claims to measure.

The long form of the BDI is composed of 21 questions or items, each with four possible responses. Each response is assigned a score ranging from zero to three, indicating the severity of the symptom. A version designed for use by primary care providers (BDI-PC) is composed of seven self-reported items, each correlating to a symptom of **major depressive disorder** experienced over the preceding two weeks.

Individual questions of the BDI assess mood, pessimism, sense of failure, self-dissatisfaction, guilt, punishment, self-dislike, self-accusation, suicidal ideas, crying, irritability, social withdrawal, body image, work difficulties, **insomnia**, **fatigue**, appetite, weight loss, bodily preoccupation, and loss of libido. Items 1 to 13 assess symptoms that are psychological in nature, while items 14 to 21 assess more physical symptoms.

Results

The sum of all BDI item scores indicates the severity of depression. The test is scored differently for the general population and for individuals who have been clinically diagnosed with depression. For the general population, a score of 21 or over represents depression. For people who have been clinically diagnosed, scores from 0 to 9 represent minimal depressive symptoms, scores of 10 to 16 indicate mild depression, scores of 17 to 29 indicate moderate depression, and scores of 30 to 63 indicate severe depression. The BDI can distinguish between different subtypes of depressive disorders, such as major depression and dysthymia (a less severe form of depression).

The BDI has been extensively tested for content validity, concurrent validity, and construct validity. The BDI has content validity (the extent to which items of a test are representative of that which is to be measured) because it was constructed from a consensus among clinicians about depressive symptoms displayed by psychiatric patients. Concurrent validity is a measure of the extent to which a test concurs with already existing standards; at least 35 studies have shown concurrent validity between the BDI and such measures of depression as the **Hamilton Depression Scale** and the **Minnesota**

Multiphasic Personality Inventory-D. Following a range of biological factors, attitudes, and behaviors, tests for construct validity (the degree to which a test measures an internal construct or variable) have shown the BDI to be related to medical symptoms, anxiety, **stress**, loneliness, sleep patterns, alcoholism, suicidal behaviors, and adjustment among youth.

Factor analysis, a statistical method used to determine underlying relationships between variables, has also supported the validity of the BDI. The BDI can be interpreted as one syndrome (depression) composed of three factors: negative attitudes toward self, performance impairment, and somatic (bodily) disturbance.

The BDI has also been extensively tested for reliability, following established standards for psychological tests published in 1985. Internal consistency has been successfully estimated by over 25 studies in many populations. The BDI has been shown to be valid and reliable, with results corresponding to clinician ratings of depression in more than 90% of all cases.

Higher BDI scores have been shown in a few studies to be inversely related to educational attainment; the BDI, however, does not consistently correlate with sex, race, or age.

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National Alliance for the Mentally Ill (NAMI). Colonial Place 3, 2107 Wilson Blvd, Suite 300, Arlington VA, 22201-3042. (703) 524-7600 or (800) 950-6264. <http://www.nami.org>.

National Depressive and Manic Depressive Association (NDMDA). 730 N. Franklin St, Suite 501, Chicago IL 60601-3526. (314) 642-0049 or (800) 826-3632. <http://www.ndmda.org>.

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Bed-wetting *see* **Enuresis**

Behavior modification

Definition

Behavior modification is a treatment approach, based on the principles of operant conditioning, that replaces undesirable behaviors with more desirable ones through positive or negative **reinforcement**.

Purpose

Behavior modification is used to treat a variety of problems in both adults and children. Behavior modification has been successfully used to treat **obsessive-compulsive disorder** (OCD), **attention-deficit/hyperactivity disorder** (ADHD), phobias, **enuresis** (bed-wetting), **generalized anxiety disorder**, and **separation anxiety disorder**, among others.

Description

Behavior modification is based on the principles of operant conditioning, which were developed by American behaviorist B. F. Skinner (1904-1990). Skinner formulated the concept of operant conditioning, through which behavior could be shaped by reinforcement or lack of it. Skinner considered his concept applicable to a wide range of both human and animal behaviors and introduced operant conditioning to the general public in his 1938 book, *The Behavior of Organisms*.

One behavior modification technique that is widely used is positive reinforcement, which encourages certain behaviors through a system of rewards. In behavior therapy, it is common for the therapist to draw up a contract with the client establishing the terms of the reward system.

In behavior modification, extinction eliminates the incentive for unwanted behavior by withholding the expected response. A widespread parenting technique based on extinction is the time-out, in which a child is separated from the group when he or she misbehaves. This technique removes the expected reward of parental attention. *(Cindy Roesinger. Photo Researchers, Inc. Reproduced by permission.)*

Another behavior modification technique is negative reinforcement. Negative reinforcement is a method of training that uses a negative reinforcer. A negative reinforcer is an event or behavior whose reinforcing properties are associated with its removal. For example, terminating an existing electric shock after a rat presses a bar is a negative reinforcer.

In addition to rewarding desirable behavior, behavior modification can also discourage unwanted behavior, through punishment. Punishment is the application of an aversive or unpleasant stimulus in reaction to a particular behavior. For children, this could be the removal of television privileges when they disobey their parents or teacher. The removal of reinforcement altogether is called extinction. Extinction eliminates the incentive for unwanted behavior by withholding the expected response. A widespread parenting technique based on extinction is the time-out, in which a child is separated from the group when he or she misbehaves. This technique removes the expected reward of parental attention.

Results

Normal results are that undesirable behaviors are replaced with more desirable ones.

See also Aversion therapy; Cognitive-behavioral therapy; Token economy system

Resources

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Association for the Advancement of Behavior Therapy. 15 W. 36th St. New York, NY, 10018. (212) 279-7970.

Behavior therapy *see* **Cognitive-behavioral therapy**

Behavioral self-control training *see* **Self-control strategies**

Benadryl *see* **Diphenhydramine**

Bender Gestalt Test

Definition

The Bender Gestalt Test, or the Bender Visual Motor Gestalt Test, is a psychological assessment instrument used to evaluate visual-motor functioning and visual perception skills in both children and adults. Scores on the test are used to identify possible organic **brain** damage and the degree maturation of the nervous system. The Bender Gestalt was developed by **psychiatrist** Lauretta Bender in the late nineteenth century.

Purpose

The Bender Gestalt Test is used to evaluate visual maturity, visual motor integration skills, style of responding, reaction to frustration, ability to correct mistakes, planning and organizational skills, and motivation. Copying figures requires fine motor skills, the ability to discriminate between visual stimuli, the capacity to integrate visual skills with motor skills, and the ability to shift attention from the original design to what is being drawn.

Precautions

The Bender Gestalt Test should not be administered to an individual with severe visual impairment unless his or her vision has been adequately corrected with eyeglass-

KEY TERMS

Psychometric testing—Pertaining to testing and measurement of mental or psychological abilities. Psychometric tests convert an individual's psychological traits and attributes into a numerical estimation or evaluation.

es. Additionally, the test should not be given to an examinee with a severe motor impairment, as the impairment would affect his or her ability to draw the geometric figures correctly. The test scores might thereby be distorted.

The Bender Gestalt Test has been criticized for being used to assess problems with organic factors in the brain. This criticism stems from the lack of specific signs on the Bender Gestalt Test that are definitively associated with brain injury, **mental retardation**, and other physiological disorders. Therefore, when making a **diagnosis** of brain injury, the Bender Gestalt Test should never be used in isolation. When making a diagnosis, results from the Bender Gestalt Test should be used in conjunction with other medical, developmental, educational, psychological, and neuropsychological information.

Finally, psychometric testing requires administration and evaluation by a clinically trained examiner. If a scoring system is used, the examiner should carefully evaluate its reliability and validity, as well as the normative sample being used. A normative sample is a group within a population who takes a test and represents the larger population. This group's scores on a test are then be used to create "norms" with which the scores of test takers are compared.

Description

The Bender Gestalt Test is an individually administered pencil and paper test used to make a diagnosis of brain injury. There are nine geometric figures drawn in black. These figures are presented to the examinee one at a time; then, the examinee is asked to copy the figure on a blank sheet of paper. Examinees are allowed to erase, but cannot use any mechanical aids (such as rulers). The popularity of this test among clinicians is most likely the short amount of time it takes to administer and score. The average amount of time to complete the test is five to ten minutes.

The Bender Gestalt Test lends itself to several variations in administration. One method requires that the examinee view each card for five seconds, after which the card is removed. The examinee draws the figure from memory. Another variation involves having the examinee draw the figures by following the standard procedure. The examinee is then given a clean sheet of paper and asked to draw as many figures as he or she can recall. Last, the test is given to a group, rather than to an individual (i.e., standard administration). It should be noted that these variations were not part of the original test.

Results

A scoring system does not have to be used to interpret performance on the Bender Gestalt Test; however, there are several reliable and valid scoring systems available. Many of the available scoring systems focus on specific difficulties experienced by the test taker. These difficulties may indicate poor visual-motor abilities that include:

- Angular difficulty: This includes increasing, decreasing, distorting, or omitting an angle in a figure.
- Bizarre doodling: This involves adding peculiar components to the drawing that have no relationship to the original Bender Gestalt figure.
- Closure difficulty: This occurs when the examinee has difficulty closing open spaces on a figure, or connecting various parts of the figure. This results in a gap in the copied figure.
- Cohesion: This involves drawing a part of a figure larger or smaller than shown on the original figure and out of proportion with the rest of the figure. This error may also include drawing a figure or part of a figure significantly out of proportion with other figures that have been drawn.
- Collision: This involves crowding the designs or allowing the end of one design to overlap or touch a part of another design.
- Contamination: This occurs when a previous figure, or part of a figure, influences the examinee in adequate completion of the current figure. For example, an examinee may combine two different Bender Gestalt figures.
- Fragmentation: This involves destroying part of the figure by not completing or breaking up the figures in ways that entirely lose the original design.
- Impotence: This occurs when the examinee draws a figure inaccurately and seems to recognize the error, then, he or she makes several unsuccessful attempts to improve the drawing.
- Irregular line quality or lack of motor coordination: This involves drawing rough lines, particularly when the examinee shows a tremor motion, during the drawing of the figure.
- Line extension: This involves adding or extending a part of the copied figure that was not on the original figure.

- Omission: This involves failing to adequately connect the parts of a figure or reproducing only parts of a figure.
- Overlapping difficulty: This includes problems in drawing portions of the figures that overlap, simplifying the drawing at the point that it overlaps, sketching or redrawing the overlapping portions, or otherwise distorting the figure at the point at which it overlaps.
- Perseveration: This includes increasing, prolonging, or continuing the number of units in a figure. For example, an examinee may draw significantly more dots or circles than shown on the original figure.
- Retrogression: This involves substituting more primitive figures for the original design—for example, substituting solid lines or loops for circles, dashes for dots, dots for circles, circles for dots, or filling in circles. There must be evidence that the examinee is capable of drawing more mature figures.
- Rotation: This involves rotating a figure or part of a figure by 45° or more. This error is also scored when the examinee rotates the stimulus card that is being copied.
- Scribbling: This involves drawing primitive lines that have no relationship to the original Bender Gestalt figure.
- Simplification: This involves replacing a part of the figure with a more simplified figure. This error is not due to maturation. Drawings that are primitive in terms of maturation would be categorized under "Retrogression."
- Superimposition of design: This involves drawing one or more of the figures on top of each other.
- Workover: This involves reinforcing, increased pressure, or overworking a line or lines in a whole or part of a figure.

Additionally, observing the examinee's behavior while drawing the figures can provide the examiner with an informal evaluation and data that can supplement the formal evaluation of the examinee's visual and perceptual functioning. For example, if an examinee takes a large amount of time to complete the geometric figures, it may suggest a slow, methodical approach to tasks, compulsive tendencies, or depressive symptoms. If an examinee rapidly completes the test, this could indicate an impulsive style.

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Benztropine

Definition

Benztropine is classified as an antiparkinsonian agent. It is sold in the United States under the brand name Cogentin and is also available under its generic name.

Purpose

Benztropine is used to treat a group of side effects (called parkinsonian side effects) that include tremors, difficulty walking, and slack muscle tone. These side effects may occur in patients who are taking antipsychotic medications used to treat mental disorders such as **schizophrenia**.

Description

Some medicines, called antipsychotic drugs, that are used to treat schizophrenia and other mental disorders can cause side effects that are similar to the symptoms of Parkinson's disease. The patient does not have Parkinson's disease, but he or she may experience shaking in muscles while at rest, difficulty with voluntary movements, and poor muscle tone. These symptoms are similar to the symptoms of Parkinson's disease.

One way to eliminate these undesirable side effects is to stop taking the antipsychotic medicine. Unfortunately, the symptoms of the original mental disorder usually come back, so in most cases simply stopping the antipsychotic medication is not a reasonable option. Some drugs such as benztropine that control the symptoms of

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Catheterization—Placing a tube in the bladder so that it can be emptied of urine.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Parkinsonian—Related to symptoms associated with Parkinson's disease, a nervous system disorder characterized by abnormal muscle movement of the tongue, face, and neck, inability to walk or move quickly, walking in a shuffling manner, restlessness, and/or tremors.

Parkinson's disease also control the parkinsonian side effects of antipsychotic medicines.

Benztropine works by restoring the chemical balance between dopamine and acetylcholine, two neurotransmitter chemicals in the **brain**. Taking benztropine along with the antipsychotic medicine helps to control symptoms of the mental disorder, while reducing parkinsonian side effects. Benztropine is in the same family of drugs (commonly known as anticholinergic drugs) as **biperiden** and **trihexyphenidyl**.

Recommended dosage

Benztropine is available in 0.5-, 1.0-, and 2.0-mg tablets and in an injectable form containing 2 mg of drug in each 2 mL glass container. For the treatment of tremor, poor muscle tone, and similar side effects, benztropine should be started at a dose of 1 to 2 mg orally. In cases of severe side effects, benztropine can be given as an intramuscular injection two to three times daily or as needed. Parkinson-like side effects caused by antipsychotic drugs may come and go, so benztropine may not be needed on a regular basis. Benztropine may also be prescribed to prevent these side effects before they actually occur. This is called as prophylactic (preventative) therapy.

Precautions

Benztropine should never be used in children under age three. It should be used cautiously and with close physician supervision in older children and in the elderly. Benztropine, like all anticholinergic drugs, decreases sweating and the body's ability to cool itself. People who are unaccustomed to being outside in hot weather should take care to stay as cool as possible and drink extra fluids. People who are chronically ill, have a central nervous system disease, or who work outside during hot weather may need to avoid taking benztropine.

People who have the following medical problems may experience increased negative side effects when taking benztropine. Anyone with these problems should discuss their condition with their physician before starting the drug:

- glaucoma, especially closed-angle glaucoma
- intestinal obstruction
- prostate enlargement
- urinary bladder obstruction

Although rare, some patients experience euphoria while taking benztropine and may abuse it for this reason. Euphoria can occur at doses only two to four times the normal daily dose. Patients with a history of drug abuse should be observed carefully for benztropine abuse.

Side effects

Although benztropine helps to control the side effects of antipsychotic drugs, it can produce side effects of its own. A person taking benztropine may have some of the following reactions, which may vary in intensity:

- dry mouth
- dry skin
- blurred vision
- nausea or vomiting
- constipation
- disorientation
- drowsiness
- irritability

- increased heart rate
- urinary retention

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by reducing or temporarily discontinuing benztropine. Chewing sugarless gum or sucking on sugarless candy may also help to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement may be especially prone to urinary retention. Symptoms of this problem include having difficulty starting a urine flow and more difficulty passing urine than usual. This side effect may be severe and require discontinuation of the drug. Urinary retention may require catheterization. People who think they may be experiencing any side effects from this or any other medication should tell their physician.

Patients who take an overdose of benztropine are treated with forced vomiting, removal of stomach contents and stomach washing, activated charcoal, and respiratory support if needed. They are also given physostigmine, an antidote for anticholinergic drug poisoning.

Interactions

When drugs such as benztropine are taken with antidepressants such as **amitriptyline**, **imipramine**, **trimipramine**, **desipramine**, **nortriptyline**, **protriptyline**, **amoxapine**, and **doxepin** or with many antihistamines that also have anticholinergic properties, the effects and side effects of benztropine are usually intensified.

Drugs such as benztropine decrease the speed with which food moves through the stomach and intestines. Because of this, the absorption of other drugs being taken may be enhanced by benztropine. Patients receiving benztropine should be alert to unusual responses to other drugs they might be taking and report any changes to their physician.

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Jack Raber, Pharm.D.

Bereavement *see* **Grief**

Beta blockers

Definition

Beta blockers, also known as beta antagonists, are a class of drugs that were first developed for the treatment of certain heart conditions and hypertension. Later, beta blockers were also found to be useful in glaucoma, migraine, and some psychiatric disorders such as performance anxiety, tremors secondary to lithium, and **movement disorders** that are caused by some drugs used in the treatment of **psychosis**. In the United States, the most commonly used beta blocker used in psychiatric practice is **propranolol** (Inderal). Nadolol (Corgard), metoprolol (Lopressor), and atenolol (Tenormin) are also used in psychiatric practice but to a lesser degree.

Purpose

Beta blockers are proven effective in the treatment of performance anxiety, lithium-induced tremor, and neuroleptic-induced akathisia (a physical condition caused by certain antipsychotic drugs). Beta blockers have sometimes been used with benzodiazepines in treating alcohol withdrawal.

Description

Beta blockers act on that part of the central nervous system that controls mental alertness, lung function, heart rate, and blood vessels. Although there is more than one mechanism by which beta blockers work in anxiety states, the most beneficial result probably arises from the fact that beta blockers slow the heart to a normal rate and rhythm. Therefore, persons with performance anxiety do not experience the usual chest tightness and rapid heart rate that is associated with such acts as public speaking or acting.

Certain antipsychotic medications known as neuroleptics can cause an unwanted effect called akathisia, which is the inability to sit, stand still, or remain inactive. Patients are restless, and in severe cases, may pace constantly and forcefully and repeatedly stomp their feet. Beta blockers can sometimes treat this condition with a lower incidence of side effects than any other drugs used to treat this condition.

Propranolol is available in 10- to 90-mg tablets. Nadolol is available in 20-, 40-, 80-, 120-, and 160-mg tablets. Atenolol is available in 50- and 100-mg tablets. Metoprolol is available in 50- and 100-mg tablets.

Recommended dosage

For the treatment of performance anxiety, sometimes called stage fright, a single dose of propranolol ranging

KEY TERMS

Akathisia—Agitated or restless movement, usually affecting the legs. Movement is accompanied by a sense of discomfort and an inability to sit, stand still, or remain inactive for periods of time. Akathisia is a common side effect of some neuroleptic (antipsychotic) medications.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Tremor—Involuntary shaking of the hands and arms.

from 10–40 mg is given 20–30 minutes before the event causing the unwanted reactions.

For lithium-induced tremors that cannot be controlled by reducing caffeine intake or administering the dosage of lithium at bedtime, propranolol at a dose of 20–160 mg daily can be given in two or three evenly divided doses.

For akathisia caused by antipsychotic medications, propranolol can be administered at doses of 10–30 mg three times daily.

Precautions

Because of their ability to narrow airways, beta blockers, especially propranolol, should not be taken by people with asthma and chronic obstructive pulmonary disease (COPD). If there is an urgent need to use beta blockers in persons with respiratory problems, atenolol or metoprolol are the beta-blockers of choice because they are less likely to have this side effect, although even these drugs should also be used with caution. Patients with congestive heart failure or certain cardiac conduction abnormalities such as a heart block, should also receive these drugs with caution.

Beta blockers should be used with close physician monitoring in people with diabetes, since the symptoms of low blood sugar (increased heart rate, lightheadedness, and abnormal perspiration) may be not be recognized by patients.

Side effects

Beta blockers can cause undesired decreases in blood pressure and are typically not given if blood pressure is 90/60 mm Hg or less.

Beta blockers can also cause an undesired drop in heart rate. People whose resting heart rate is less than 55 beats per minute should not take beta blockers.

Occasionally, beta blockers can cause rash, weakness, nausea, vomiting, and stomach discomfort.

Interactions

Each medication in the class of beta blockers has the potential to interact with a multitude of other medications. Anyone starting beta blocker therapy should review the other medications they are taking with their physician and pharmacist for possible interactions. Patients should always inform all their health care providers, including dentists, that they are taking beta blockers.

Resources

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Ajna Hamidovic, Pharm.D.

Bibliotherapy

Definition

Bibliotherapy is an adjunct to psychological treatment that incorporates appropriate books or other written materials, usually intended to be read outside of **psychotherapy** sessions, into the treatment regimen.

Purpose

The goal of bibliotherapy is to broaden and deepen the client's understanding of the particular problem that requires treatment. The written materials may educate the client about the disorder itself or be used to increase the client's acceptance of a proposed treatment. Many people find that the opportunity to read about their problem outside the therapist's office facilitates active participation in their treatment and promotes a stronger sense of personal responsibility for recovery. In addition, many are relieved to find that others have had the same disorder or problem and have coped successfully with it or recovered from it. From the therapist's standpoint, providing a client with specific information or assignments to be completed outside regular in-office sessions speeds the progress of therapy.

KEY TERMS

Adjunct—A form of treatment that is not strictly necessary to a therapy regimen but is helpful.

Cognitive-behavioral therapy—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

Dyslexia—A type of reading disorder.

Regimen—A regulated course of treatment for a medical or mental disorder.

Bibliotherapy has been applied in a variety of settings to many kinds of psychological problems, including eating disorders, anxiety and mood disorders, and alcohol and substance abuse. Many people find that the opportunity to read about their problem outside the therapist's office facilitates active participation in their treatment. *(Joseph Nettis. Photo Researchers, Inc. Reproduced by permission.)*

Bibliotherapy has been applied in a variety of settings to many kinds of psychological problems. Practitioners have reported successful use of bibliotherapy in treating eating disorders, anxiety and mood disorders, **agoraphobia**, alcohol and substance abuse, and stress-related physical disorders.

Precautions

Bibliotherapy is not likely to be useful with clients suffering from thought disorders, psychoses, limited intellectual ability, dyslexia, or active resistance to treatment. In addition, some clients may use bibliotherapy as a form of do-it-yourself treatment rather than seeking professional help.

Description

In most settings, bibliotherapy is used as an adjunct to more traditional forms of psychotherapy. Practitioners of cognitive-behavioral therapies are among the most enthusiastic supporters of bibliotherapy, particularly in the development of individualized treatment protocols, including workbooks, for specific disorders. For example, clients with eating disorders, especially **bulimia nervosa**, often benefit from receiving educational information appropriate to their stage of recovery, such as books or articles about cultural biases regarding weight, attractiveness, and dieting. This information helps clients better understand the rationale for their treatment and to work on new skills or behavioral changes more effectively.

Aftercare

Unlike many standard forms of psychotherapy, bibliotherapeutic approaches often include specific examples of ways to deal with relapses or setbacks. As long as the client keeps these materials, he or she has easy access to resources for getting back on track.

Risks

People who use self-help manuals without professional guidance run the risk of misapplying techniques or misdiagnosing their problems.

Normal results

As with any form of treatment, bibliotherapy is effective only if it actively engages the client's desire for and belief in recovery. For many people, additional information or workbooks that can be used in private reinforce their commitment to getting better. People who lack the time or finances to attend regular psychotherapy sessions at a practitioner's office often find that bibliotherapy can bridge the gap between infrequent appointments. Likewise, the nature of the disorder itself may preclude in-office treatment for some people, such as persons suffering from agoraphobia. Current research indicates that a bibliotherapeutic approach can be highly effective in helping agoraphobics better understand and cope with their symptoms.

Resources

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Jane A. Fitzgerald, Ph.D.

Binge eating

Description

Binge eating is a form of overeating in which a person ingests a large amount of food during a discrete period of time (within one or two hours, for example) and experiences feelings of being out of control and unable to stop eating during the episode. In practice, the duration of a binge may vary greatly from one event to the next, making it difficult to define the number of binges occurring in a given day. Binge eating often occurs in the absence of hunger and is characterized by eating very rapidly; eating alone (due to embarrassment over the amount being eaten); and having strong negative feelings, such as guilt, shame and depression, following the binge. Typically, a binge episode ends only when all the desirable binge foods have been consumed or when the person feels too full to continue eating.

While binge eating is a symptom of **bulimia nervosa**, it differs from this disorder in that behaviors intended to get rid of the food (fasting, excessive exercise, or using laxatives or inducing vomiting to "purge" the food from the system) are present among those with bulimia, but are generally absent among binge eaters. Binge eating may also occur in **anorexia nervosa**.

The clinician's diagnostic handbook, the ***Diagnostic and Statistical Manual of Mental Disorders*** (fourth edition, text revised, published in 2000) subsumes binge eating under the **diagnosis** of eating disorders not otherwise specified. Binge eating disorder is, however, under consideration as a separate diagnostic category, pending further study.

Symptoms and treatments

Binge eating episodes may occur in response to strong negative emotions, such as depression or anxiety, or to less defined feelings of distress or tension. The act of bingeing seems to alleviate these uncomfortable feelings temporarily and binge eaters typically describe themselves as "numb" or "spaced out" while engaged in these behaviors. Some people report that binges are related to the ingestion of certain "trigger foods," usually carbohydrates, but regardless of the stimulus, the feeling of eating without being able to control one's intake is a frightening experience for most people. The aftermath of a binge often includes an overwhelming sense of self-disgust, depression and anxiety.

While people who binge eat are clearly at high risk for becoming overweight, there are important differences between simple **obesity** and binge eating. People who binge eat are far more likely to report significant mood problems, especially depression, and to report greater dissatisfaction with their weight and shape than are comparably obese persons. They are also more likely to describe themselves as experiencing personal problems and work difficulties and to be hypersensitive to the thoughts and opinions of others. Like people with bulimia nervosa, they also have an increased likelihood of being diagnosed with major depression, substance-related disorders, and **personality disorders**, yet the overall rates of recovery for binge eating disorders are actually more favorable than those obtained in bulimia.

Binge eating is not common among the general public, but it is prevalent among persons attending weight loss clinics, where as many as half of the participants may fit this description. Both males and females develop binge-eating problems, but the rate of occurrence is 1.5 times greater among women. Age of onset is usually adolescence through young adulthood and the course of the disorder is often marked by a long history of on-again, off-again dieting.

As is the case with other forms of eating disorders, identification of specific causes for binge eating has been difficult. Since many people report relief from painful or uncomfortable mental states while bingeing, the behavior offers short-term emotional relief, making it likely to be repeated. Some investigators have considered genetic influences and personality variables. Still others have suggested that the "culture of thinness" in western societies contributes to the tendency toward harsh self-evaluation characterizing binge-eaters who then turn to food for solace.

At present, the most effective treatment approach to reducing the incidence of binge eating appears to be **cognitive-behavioral therapy** (CBT). The goal of this therapy is the development of skills for effectively coping with emotional distress rather than seeking to numb or disguise troubling feelings. This therapy focuses on helping the affected individual to decrease the binge eating behavior by recognizing the connection between thoughts and behavior, and to change behavior by changing negative thinking patterns. Follow-up research has

been very encouraging, documenting both a decrease in depressive symptoms and a corresponding likelihood of healthy weight loss as the individual achieves better control of eating behaviors.

Resources

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Jane A. Fitzgerald, Ph.D.

KEY TERMS

Bruxism—Habitual, often unconscious, grinding of the teeth.

Epilepsy—A neurological disorder characterized by the onset of seizures. Seizures are caused by a disturbance in the electrical activity in the brain and can cause loss of consciousness, muscle spasms, rhythmic movements, abnormal sensory experiences, or altered mental states.

Incontinence—Inability to control the release of urine or feces.

Irritable bowel syndrome (IBS)—A condition affecting the small and large intestine, usually associated with emotional stress. There may be complaints of diarrhea and pain in the lower abdomen.

Raynaud's syndrome—A disorder of the circulatory or vascular system characterized by abnormally cold hands and feet because of constricted blood vessels in these areas.

Temporomandibular joint disorder (TMJ)—Inflammation, irritation, pain, limited range of motion, and clicking sounds in the jaw caused by improper opening and closing of the joint.

Biofeedback

Definition

Biofeedback is a technique that uses monitoring instruments to measure and feed back information about muscle tension, heart rate, sweat responses, skin temperature, or **brain** activity.

Terms associated with biofeedback include applied psychophysiology or behavioral physiology. It is also viewed as a mind-body therapy method used in complementary and alternative medicine. Biofeedback is an important part of understanding the relationship between physical state and thoughts, feelings, and behaviors.

Purpose

The purpose of biofeedback is to enhance an individual's awareness of physical reactions to physical, emotional, or psychological **stress**, and their ability to influence their own physiological responses. The overall purpose is to develop self-regulation skills that play a role in improving health and well-being.

Biofeedback has been used as a part of a comprehensive treatment approach with a number of conditions, including chronic pain, irritable bowel syndrome (IBS), temporomandibular joint disorder (TMJ), Raynaud's syndrome, epilepsy, **attention-deficit/hyperactivity disorder** (ADHD), anxiety, migraine headaches, depression, traumatic brain injury, and **sleep disorders**. There is some support for using biofeedback in the treatment of diabetes when self-monitoring of blood glucose levels is maintained and within the context of regular physician consultation and supervision.

Biofeedback has been a useful tool in helping individuals with urinary incontinence regain bladder control by controlling the muscles used in urination. Sensors are placed in the vaginal or anal canal to help individuals learn when the muscles are properly contracted. A recent study found that this type of biofeedback treatment was safe, effective, and well liked by women patients 55 years and older.

Conditions related to stress are also treated using biofeedback, such as certain types of headaches, high blood pressure, bruxism or teeth grinding, **post-traumatic stress disorder** (PTSD), eating disorders, substance abuse, and some anxiety disorders. In treatment of

stress-related conditions, biofeedback is often used in combination with relaxation training. Sometimes, biofeedback is used to help individuals learn how to experience deeper relaxation, such as in childbirth education programs or general stress management. This is referred to as biofeedback-assisted relaxation training. Even for individuals who can achieve relaxation through other strategies such as **meditation** or relaxation, biofeedback can be a valuable added technique. Biofeedback offers special advantages, such as allowing the clinician to track closely the places where an individual tenses up and helps the individual learn what thoughts and feelings are associated with the tension.

Precautions

Biofeedback depends on the motivation and active participation of an individual. Thus, it may not be suitable for individuals with low motivation who are not willing to take a highly active role in treatment, such as those suffering from depression. Also, since biofeedback focuses on initiating behavioral changes, individuals inclined to examine their past to alleviate problems and symptoms may benefit more from other treatment types, such as **psychotherapy**. Individuals with cognitive impairment may be unable to remain engaged in the treatment, depending on their level of functioning. Also, individuals with a pacemaker or other implanted electrical devices should inform their health care professional before entering biofeedback training, as certain types of biofeedback sensors may interfere with the devices. Patients with specific pain symptoms in which the cause is unknown should have a thorough medical examination to rule out any serious underlying disease before starting biofeedback training. Biofeedback can be used in combination with conventional therapies; however, while it can be used in combination with conventional medical treatment for illnesses such as cancer and diabetes, it should not replace those treatments.

Research on the success of biofeedback in treating certain conditions is inconclusive or needs to be validated. Some research studies use a small number of participants, which makes it difficult to generalize the results to a larger population. Also, many conditions have different subtypes with a variety of psychological, social, and physical causes. This fact, combined with research design concerns, makes it difficult to compare research studies. For example, while most studies have reported positive outcomes in the treatment of alcohol abuse and dependence, problems with methods and statistical analyses have called study results into question. Also, its effectiveness in treating opiate abuse or dependence has not been consistently shown, as with its use in treating menopausal hot flashes, and there are limitations in studies relating to its use in cancer treatment. Continued research is needed to further evaluate and improve different biofeedback techniques for various conditions.

Description

According to the Association for Applied Psychophysiology and Biofeedback, the technique was developed in the early 1970s by psychologists and physicians. These techniques continue to be used by psychologists, physicians, nurses, and other health care professionals such as physical therapists. Prior to beginning any biofeedback training, individuals may need a comprehensive psychological, educational, and/or medical assessment. Biofeedback can be used in conjunction with nonmedical treatments, such as psychotherapy, **cognitive-behavioral therapy**, and behavioral treatment strategies.

How biofeedback works

Biofeedback utilizes electronic sensors, or electrodes, attached to various parts of the body to detect changes in physical responses. Signals then inform the individual of these changes by means of visual or auditory signals such as a light display or a series of beeps. While the individual views or listens to feedback, he or she begins to recognize thoughts, feelings, and mental images that influence his or her physical reactions. By monitoring this mind-body connection, the individual can use the same thoughts, feelings, and mental images as cues or reminders to become more relaxed, or to change heartbeat, brain wave patterns, body temperature, and other body functions. The individual uses trial-and-error to change the signals change in the desired direction. For example, individuals trying to control their blood pressure levels may see a light flash whenever the pressure drops below a certain level. They may then try to remember what their thoughts and feelings were at the moment and deliberately maintain them to keep the blood pressure level low.

Through training, the individual learns to control the targeted physical response and, over time, is able to recognize what is required to reduce problematic symptoms. Eventually, the external biofeedback becomes unnecessary as the individual learns to perceive internal physical responses and make the desired changes. The individual then has a powerful, portable, and self-administered treatment tool to deal with problematic symptoms.

Three stages of biofeedback training

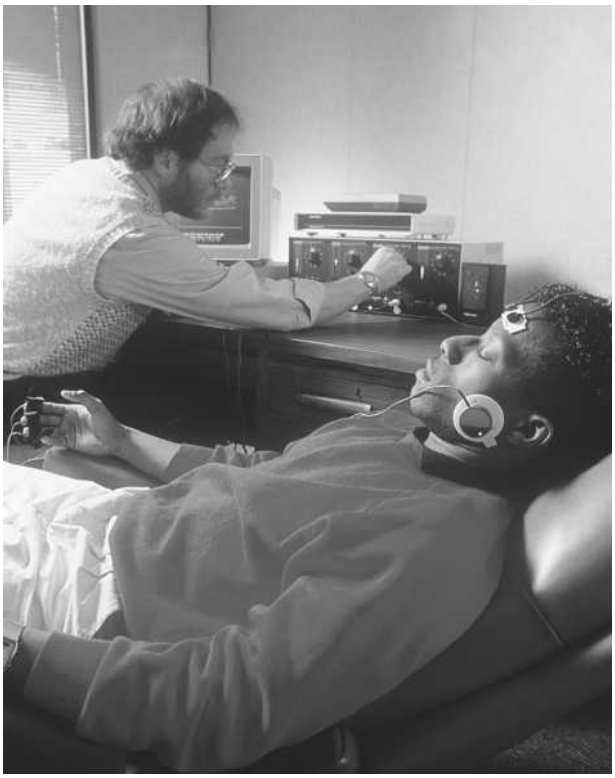
- Awareness of the problematic physical response: Individuals may complete a psychophysiological stress

profile (PSP) to identify how their bodies respond to a variety of stressors and determine their ability to overcome undesired physical reactions. This involves a period of rest, stress, and recovery. For example, various sensors are attached to various parts of the body, and a baseline measurement lasting from two to four minutes records physical responses. The individual then goes through a standard set of stressors (such as rapid math calculations or running in place) each lasting from two to four minutes. This is followed by another relaxation period to determine the length of the recovery period.

- Using signals from the biofeedback equipment to control physical responses: The individual is assisted in reaching certain goals related to managing a specific physical response.
- Transferring control from biofeedback equipment or the health care professional: Individuals learn to identify triggers that alert them to implement their new-found self-regulation skills.

Types of biofeedback equipment

- Electromyograph (EMG): Sensors (or electrodes) placed on the skin on pertinent parts of the body monitor electrical activity in muscles, specifically tension. This is the most frequently used biofeedback method in the treatment of various neurologic disorders such as **stroke**, cerebral palsy, traumatic brain injury, and multiple sclerosis. In children and adolescents, EMG may be used to treat tension headaches, **enuresis**, and **encopresis**. In treating TMJ or bruxism, EMG sensors are placed on jaw muscles. Chronic pain is treated by monitoring muscle tension in various places on the body.
- Galvanic skin response (GSR): Sensors on the fingers monitor perspiration or sweating. This is also referred to as obtaining a skin conductance level (SCL). GSR may be used in the treatment of anxiety, fears or phobias, stress, and sleep problems.
- Temperature or thermal sensors: Sensors monitor body temperature and changes in blood flow. Changes in hand temperature, for example, can indicate relaxation when there is increased blood flow to the skin. Temperature biofeedback may be useful for treating migraine headache, Raynaud's disorder, and anxiety disorders.
- Heart rate sensors: A pulse monitor placed on the fingertip monitors pulse rate. Increases in heart rate are associated with emotional arousal, such as being angry or fearful. Decreases in heart rate are associated with relaxation.
- Capnometry (CAP): Respiratory sensors monitor oxygen intake and carbon dioxide output. This differentiates correct breathing from problematic breathing practices. Breath control training may be used to treat panic attacks, asthma, and a variety of stress-related conditions.
- Electroencephalographs (EEG) or neurofeedback: Sensors attached to the scalp monitor brain wave activity in different parts of the brain. It may be used to treat conditions with proven or suspected impact on brain wave patterns such as seizure disorders or epilepsy, ADHD, learning disabilities, migraine headaches, traumatic brain injury, and sleep disorders.



A patient undergoes biofeedback monitoring for stress. *(Photo by Will and Deni McIntyre. Photo Researchers, Inc. Reproduced by permission.)*

Biofeedback is geared toward whatever a person finds most appealing and understandable and provided in several formats such as auditory, visual, or multimedia. Audio feedback, that may take the form of changes in tone and pitch, is useful because visual attention is not necessary. Visual feedback can be provided in various forms such as bar or line graphs on a computer screen. Initially, it was thought that—over time—computer signals could become boring or visually unappealing. In response to this, Barry Bittman developed Mindscope in 1992 that displays video scenes with realistic sounds on a high-definition television set connected to a computer. Physical responses detected by the biofeedback equipment control an engaging audiovisual environment of beautiful and realistic scenes. Clarity, perspective,

motion, and sounds improve as the individual deepens their relaxation. For children and adolescents, this may be described as a "video game for the body." Visual displays for EMG biofeedback may include sports such as basketball, baseball, and golf, where the individual plays against the computer.

The setting in which biofeedback training takes place can vary. Sometimes the clinician, client, and equipment are in the same room. Sometimes the client may sit in comfortable seating in a semi-dark, quiet room while the clinician is in another room with the equipment. In this arrangement, the clinician and client may communicate using an intercom.

In some cases, children and adolescents may reach the desired level of control in three to five sessions. Depending on the condition, biofeedback training may require a series of sessions for several days or weeks. In general, it may take 10 or 15 sessions at the lower end to 40 or 50 sessions at the higher end.

Preparation

Biofeedback is most successful when individuals are motivated to learn. It is useful for people who have difficulty relaxing, even when they make efforts to do so. A receptive and open attitude is important for attaining desired responses rather than actively focusing on attaining them. It is important that individuals are willing to practice regularly at home to apply the skill to everyday life. Establishing a foundation of trust and confidence in the health care professional is an important component of biofeedback training.

Before beginning biofeedback training, an initial consultation will be conducted to record medical history, treatment background, and biofeedback goals. The procedure will be explained to provide a clear understanding of how and why the training will be helpful. The individual may be shown the equipment and told where they will be placed and how they work.

Before electrodes are placed on the body, the skin surface must be adequately prepared by using alcohol preparation pads to remove oils, makeup, and dead skin cells that may interfere with the biofeedback signal. An electrode paste is then applied to the sensor, or a small adhesive pad is used to adhere the sensor to the skin. Heart rate, temperature, and GSR monitors may be placed on the fingertip with a Velcro or elastic band. With CAP, the tip of a small, flexible, plastic tube is positioned in the nostril using tape. An individual may be taught several forms of biofeedback initially, then the training may be tailored to the individual's preference.

The biofeedback trainer must have technical skill, an understanding of basic anatomy and physiology, knowledge of various conditions, and familiarity with computer hardware and software. The American Psychological Association views biofeedback as a proficiency area, master's and doctoral level training programs are available through a variety of sources, and certification is available through the Biofeedback Certification Institute of America.

Aftercare

One or two follow-up sessions may be arranged two to four months after the initial set of appointments. In this way, long-term progress can be assessed, support can be provided, and adjustments can be made, if needed.

Risks

There are no known side effects with properly administered biofeedback. Problems may occur if biofeedback is used to treat certain conditions where the use of biofeedback is not advised.

Normal results

A normal result may be indicated by achieving the desired changes in muscle tension, heart rate, sweat activity, respiration rate, temperate change, and brainwave activity. Health care professionals may use various criteria or normal values that have been developed for some biofeedback equipment. These values indicate levels that can be expected from normal physiological functioning or relaxation. Importantly, an individual learns to control their physical reactions, which may lead to feelings of empowerment and confidence.

Abnormal results

Unusual results may arise from a number of factors, including poor sensor or electrode contact with the skin and interference from other electrical signals or "noise." Some equipment may react to room temperature conditions, especially when the room is very hot or very cold. Although inexpensive monitoring equipment is available, such as watches that monitor heartbeat and handheld GSR devices, their results may not be accurate.

See also Anxiety and anxiety disorders; Substance abuse and related disorders

Resources

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ORGANIZATIONS

Association for Applied Psychotherapy and Biofeedback. 10200 W. 44th Avenue, Suite 304, Wheat Ridge, CO 80033-2840. (303) 422-8436. <http://www.aapb.org>.

Biofeedback Certification Institute of America. 1022 W. 44th Avenue, Suite 310, Wheat Ridge, CO 80033. (303) 420-2902. <http://www.bcia.org>.

Joneis Thomas, Ph.D.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Catheterization—Placing a tube in the bladder so that it can be emptied of urine.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Parkinsonian—Related to symptoms associated with Parkinson's disease, a nervous system disorder characterized by abnormal muscle movement of the tongue, face, and neck, inability to walk or move quickly, walking in a shuffling manner, restlessness, and/or tremors.

Biperiden

Definition

Biperiden is classified as an antiparkinsonian agent. It is sold in the United States under the brand name of Akineton.

Purpose

Biperiden is used to treat a group of side effects (called parkinsonian side effects) that include tremors, difficulty walking, and slack muscle tone. These side effects may occur in patients who are taking antipsychotic medications used to treat mental disorders such as **schizophrenia**.

Description

Some medicines, called antipsychotic drugs, that are used to treat schizophrenia and other mental disorders can cause side effects that are similar to the symptoms of Parkinson's disease. The patient does not have Parkinson's disease, but he or she may experience shaking in muscles while at rest, difficulty with voluntary movements, and poor muscle tone. These symptoms are similar to the symptoms of Parkinson's disease.

One way to eliminate these undesirable side effects is to stop taking the antipsychotic medicine. Unfortunately, the symptoms of the original mental disorder usually come back, so in most cases simply stopping the antipsychotic medication is not a reasonable option. Some drugs such as biperiden that control the symptoms of Parkinson's disease

also control the parkinsonian side effects of antipsychotic medicines.

Biperiden works by restoring the chemical balance between dopamine and acetylcholine, two neurotransmitter chemicals in the **brain**. Taking biperiden along with the antipsychotic medicine helps to control symptoms of the mental disorder, while reducing parkinsonian side effects. Biperiden is in the same family of drugs (commonly known as anticholinergic drugs) as **benztropine**, **amantadine**, and **trihexyphenidyl**.

Recommended dosage

Biperiden is available in 2-mg tablets. For the treatment of tremor, poor muscle tone, and similar parkinsonian side effects, the dose of biperiden is 2 mg orally one to three times daily. Parkinson-like side effects caused by antipsychotic drugs may come and go, so biperiden may not be needed on a regular basis. Biperiden may also be prescribed to prevent these side effects before they actually occur. This is called as prophylactic (preventative) therapy.

Precautions

Biperiden should never be used in children under age three. It should be used cautiously and with close physician supervision in older children and in the elderly. Biperiden, like all anticholinergic drugs, decreases sweating and the body's ability to cool itself. People who are unaccustomed to being outside in hot weather should take care to stay as cool as possible and drink extra fluids. People who are chronically ill, have a central nervous system disease, or who work outside during hot weather may need to avoid taking biperiden.

People who have the following medical problems may experience increased negative side effects when taking biperiden. Anyone with these problems should discuss their condition with their physician before starting the drug:

- glaucoma, especially closed-angle glaucoma
- intestinal obstruction
- prostate enlargement
- urinary bladder obstruction

Although rare, some patients experience euphoria while taking biperiden and may abuse it for this reason. Euphoria can occur at doses only two to four times the normal daily dose. Patients with a history of drug abuse should be observed carefully for biperiden abuse.

Side effects

Although biperiden helps control the side effects of antipsychotic drugs, it can produce side effects of its own. A person taking biperiden may have some of the following side effects, which may vary in intensity:

- dry mouth
- dry skin
- blurred vision
- nausea or vomiting
- constipation
- disorientation
- drowsiness
- irritability
- increased heart rate
- urinary retention

Dry mouth, if severe to the point of causing difficulty in speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Chewing sugarless gum or sucking on sugarless candy may also help to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement may be especially prone to urinary retention. Symptoms of this problem include having difficulty starting a urine flow and more difficulty passing urine than usual. This side effect may be severe and require discontinuation of the drug. Urinary retention may require catheterization. People who think they may be experiencing any side effects from this or any other medication should tell their physicians.

Patients who take an overdose of biperiden are treated with forced vomiting, removal of stomach contents and stomach washing, activated charcoal, and respiratory support if needed. They are also given physostigmine, an antidote for anticholinergic drug poisoning.

Interactions

When drugs such as biperiden are taken with antidepressants such as **amitriptyline**, **imipramine**, **trimipramine**, **desipramine**, **nortriptyline**, **protriptyline**, **amoxapine**, and **doxepin**, as well as with many antihistamines that also have anticholinergic properties, the effects of biperiden are usually intensified.

Drugs such as biperiden decrease the speed with which food moves through the stomach and intestines. Because of this, it is possible that the absorption of some drugs may be enhanced by biperiden. Patients receiving biperiden should be observed for unusual responses to other drugs they might be taking.

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Jack Raber, Pharm.D.

Biphetamine *see* **Amphetamines**

Bipolar disorder

Definition

Bipolar, or manic-depressive, disorder is a mood disorder that causes radical emotional changes and mood swings, from manic highs to depressive lows. The majority of bipolar individuals experience alternating episodes of mania (an elevated or euphoric mood or irritable state) and depression.

Description

In the United States alone, bipolar disorder afflicts an estimated three million people. According to a report by the National Institutes of Mental Health, the disorder costs over $45 billion annually. The average age of onset of bipolar disorder is from adolescence through the early twenties. However, because of the complexity of the disorder, a correct **diagnosis** can be delayed for several years or more.

The Diagnostic and Statistical Manual of Mental Disorders, fourth edition text revised (*DSM-IV-TR*), the diagnostic standard for mental health professionals in the United States, defines four separate categories of bipolar disorder: bipolar I, bipolar II, cyclothymia, and bipolar not otherwise specified (NOS).

Bipolar I disorder is characterized by manic episodes, the "high" of the manic-depressive cycle. A bipolar patient experiencing mania often has feelings of self-importance, elation, talkativeness, increased sociability, and a desire to embark on goal-oriented activities, coupled with the characteristics of irritability, impatience, impulsiveness, hyperactivity, and a decreased need for sleep. Usually this manic period is followed by a period of depression, although a few bipolar I individuals may not experience a major depressive episode. Mixed states, where both manic or hypomanic symptoms and depressive symptoms occur at the same time, also occur frequently with bipolar I patients (for example, depression with the racing thoughts of mania). Also, dysphoric mania is common (mania characterized by anger and irritability).

Bipolar II disorder is characterized by major depressive episodes alternating with episodes of hypomania, a milder form of mania. Bipolar depression may be difficult to distinguish from unipolar depression (depression without mania, as found in **major depressive disorder**). Patients with bipolar depression tend to have extremely low energy, retarded mental and physical processes, and more profound **fatigue** (for example, hypersomnia—a sleep disorder marked by a need for excessive sleep or sleepiness when awake) than people with unipolar depression.

KEY TERMS

Anticonvulsant medication—A medication that prevents convulsions or seizures; often prescribed in the treatment of epilepsy. Several anticonvulsant medications have been found effective in the treatment of bipolar disorder.

Antipsychotic medication—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

ECT—Electroconvulsive therapy is sometimes used to treat depression or mania when pharmaceutical treatment fails.

Hypomania—A milder form of mania which is characteristic of bipolar II disorder.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Mixed mania/mixed state—A mental state in which symptoms of both depression and mania occur simultaneously.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells. Changes in the levels of certain neurotransmitters, such as serotonin, norepinephrine, and dopamine, are thought to be related to bipolar disorder.

Psychomotor retardation—Slowed mental and physical processes characteristic of a bipolar depressive episode.

Cyclothymia refers to the cycling of hypomanic episodes with depression that does not reach major depressive proportions. A third of patients with cyclothymia will develop bipolar I or II disorder later in life.

A phenomenon known as rapid cycling occurs in up to 20% of bipolar I and II patients. In rapid cycling, manic and depressive episodes must alternate frequently—at least four times in 12 months—to meet the diagnostic definition. In some cases of "ultra-rapid cycling," the patient may bounce between manic and depressive states several times within a 24-hour period. This condition is very hard to distinguish from mixed states.

Bipolar NOS is a category for bipolar states that do not clearly fit into the bipolar I, II, or cyclothymia diagnoses.

Causes and symptoms

The source of bipolar disorder has not been clearly defined. Because two-thirds of bipolar patients have a family history of emotional disorders, researchers have searched for a genetic link to the disorder. Several studies have uncovered a number of possible genetic connections to the predisposition for bipolar disorder. There is significant evidence that correlates bipolar II with genetic causes. Studies have shown that identical twins have an 80% concordance rate (presence of the same disorder). Additionally, studies have demonstrated that the disorder is transmitted to children by autosomal dominant inheritance. This means that either affected parent (father or mother) has a 50% chance of having a child (regardless if the child is male or female) with the disorder.

Further studies concerning the genetic correlations have revealed specific chromosomes (the structures that contain genes) that contain mutated genes. Susceptible genes are located in specific regions of chromosomes 13, 18, and 21. The building blocks of genes, called nucleotides, are normally arranged in a specific order and quantity. If these nucleotides are repeated, a genetic abnormality usually results. Recent evidence suggests that a special type of nucleotide repeat is observed in persons with bipolar II on chromosome 18. However, the presence of this sequence does not worsen the disorder or change the age of onset. It is currently thought that expression of bipolar II involves multiple mutated genes. Further research is ongoing to discover precise mechanisms and to develop genetic markers (gene tags) that would predict which individuals are at higher risk.

Another possible biological cause for bipolar disorder under investigation is the presence of an excessive calcium buildup in the cells. Also, dopamine and other neurochemical transmitters (the chemicals that transmit messages from nerve cell to nerve cell) appear to be implicated in bipolar disorder and these are under intense investigation.

Over half of patients diagnosed with bipolar disorder have a history of substance abuse. There is a high rate of association between cocaine abuse and bipolar disorder. Some studies have shown up to 30% of abusers meeting the criteria for bipolar disorder. The emotional and physical highs and lows of cocaine use correspond to the manic depression of the bipolar patient, making the disorder difficult to diagnose.

For some bipolar patients, manic and depressive episodes coincide with seasonal changes. Depressive episodes are typical during winter and fall, and manic episodes are more probable in the spring and summer months.

Symptoms of bipolar depressive episodes include low energy levels, feelings of despair, difficulty concentrating, extreme fatigue, and psychomotor retardation (slowed mental and physical capabilities). Manic episodes are characterized by feelings of euphoria, lack of inhibitions, racing thoughts, diminished need for sleep, talkativeness, risk taking, and irritability. In extreme cases, mania can induce **hallucinations** and other psychotic symptoms such as grandiose **delusions** (ideas that the person affected is extremely important or has some unrecognized talent or insight).

Demographics

Manic-depression is a common psychological disorder that is difficult to detect. As stated, it is estimated that about three million people in the United States are affected. The disorder is more common among women than men. Women have been observed at increased risk of developing subsequent episodes in the period immediately following childbirth.

Diagnosis

Bipolar disorder is usually diagnosed and treated by a **psychiatrist** and/or a **psychologist** with medical assistance. In addition to an interview, several clinical inventories or scales may be used to assess the patient's mental status and determine the presence of bipolar symptoms. These include the Millon Clinical Multiaxial Inventory III (MCMI-III), **Minnesota Multiphasic Personality Inventory** II (MMPI-2), the Internal State Scale (ISS), the Self-Report Manic Inventory (SRMI), and the Young Mania Rating Scale (YMRS). The tests are verbal and/or written and are administered in both hospital and outpatient settings.

Psychologists and psychiatrists typically use the criteria listed in the *DSM-IV-TR* as a guideline for diagnosis of

bipolar disorder and other mental illnesses. *DSM-IV-TR* describes a **manic episode** as an abnormally elevated or irritable mood lasting a period of at least one week that is distinguished by at least three of the mania symptoms: inflated self-esteem, decreased need for sleep, talkativeness, racing thoughts, distractibility, increase in goal-directed activity, or excessive involvement in pleasurable activities that have a high potential for painful consequences. If the mood of the patient is irritable and not elevated, four of the symptoms are required.

Although many clinicians find the criteria too rigid, a hypomanic diagnosis requires a duration of at least four days with at least three of the symptoms indicated for manic episodes (four if mood is irritable and not elevated). *DSM-IV-TR* notes that unlike manic episodes, hypomanic episodes do not cause a marked impairment in social or occupational functioning, do not require **hospitalization**, and do not have psychotic features (no delusions or hallucinations). In addition, because hypomanic episodes are characterized by high energy and goal-directed activities and often result in a positive outcome, or are perceived in a positive manner by the patient, bipolar II disorder can go undiagnosed.

Bipolar symptoms often appear differently in children and adolescents than they appear in adults. Manic episodes in these age groups are typically characterized by more psychotic features than in adults, which may lead to a misdiagnosis of **schizophrenia**. Children and adolescents also tend toward irritability and aggressiveness instead of elation. Further, symptoms tend to be chronic, or ongoing, rather than acute, or episodic. Bipolar children are easily distracted, impulsive, and hyperactive, which can lead to a misdiagnosis of **attention-deficit/hyperactivity disorder** (ADHD). Furthermore, their aggression often leads to violence, which may be misdiagnosed as a **conduct disorder**.

Substance abuse, thyroid disease, and use of prescription or over-the-counter medication can mask or mimic the presence of bipolar disorder. In cases of substance abuse, the patient must ordinarily undergo a period of **detoxification** and abstinence before a mood disorder is diagnosed and treatment begins.

Treatment

Bipolar disorder is usually treated with both medical and psychosocial interventions. Psychosocial therapies address both psychological and social issues.

Medical interventions

A combination of mood-stabilizing agents with antidepressants, antipsychotics, and anticonvulsants is used to regulate manic and depressive episodes.

MOOD-STABILIZING AGENTS. Mood-stabilizing agents such as lithium, **carbamazepine**, and **valproic acid** (valproate) are prescribed to regulate the manic highs and lows of bipolar disorder:

- Lithium (**lithium carbonate**, Cibalith-S, Eskalith, Lithane, Lithobid, Lithonate, Lithotabs) is one of the oldest and most frequently prescribed drugs available for the treatment of bipolar mania and depression. Because the drug takes four to ten days to reach a therapeutic level in the bloodstream, it is sometimes prescribed in conjunction with neuroleptics (other psychiatric drugs) and/or benzodiazepines (medications that ease tension by slowing down the central nervous system) to provide more immediate relief of a manic episode. Lithium has also been shown to be effective in regulating bipolar depression, but is not recommended for mixed mania. Lithium may not be an effective long-term treatment option for rapid cyclers, who typically develop a tolerance for it, or may not respond to it. Possible side effects of the drug include weight gain, thirst, nausea, and hand tremors. Prolonged lithium use may also cause hyperthyroidism (a disease of the thryoid marked by heart palpitations, nervousness, the presence of goiter, sweating, and a wide array of other symptoms.)
- Carbamazepine (Tegretol, Atretol) is an anticonvulsant drug (a drug to treat **seizures**) usually prescribed in conjunction with other mood-stabilizing agents. The drug is often used to treat bipolar patients who have not responded well to lithium therapy. Blurred vision and abnormal eye movement are two possible side effects of carbamazepine therapy.
- Valproic acid (**divalproex sodium**, or Depakote; valproate, or Depakene) is one of the few drugs available that has been proven effective in treating rapid cycling bipolar and mixed states patients. Valproate is prescribed alone or in combination with carbamazepine and/or lithium. Stomach cramps, indigestion, diarrhea, hair loss, appetite loss, nausea, and unusual weight loss or gain are some of the common side effects of valproate.

ANTIDEPRESSANTS. Because antidepressants may stimulate manic episodes in some bipolar patients, their use is typically short-term. Selective serotonin reuptake inhibitors (SSRIs) or, less often, monoamine oxidase inhibitors (MAO inhibitors) are prescribed for episodes of bipolar depression. Tricyclic antidepressants used to treat unipolar depression may trigger rapid cycling in bipolar patients and are, therefore, not a preferred treatment option for bipolar depression.

- SSRIs, such as **fluoxetine** (Prozac), **sertraline** (Zoloft), and **paroxetine** (Paxil), regulate depression by regulating levels of serotonin, a neurotransmitter. Anxiety, diarrhea, drowsiness, headache, sweating,

nausea, sexual problems, and **insomnia** are all possible side effects of SSRIs.

- MAOIs such as **tranylcypromine** (Parnate) and **phenelzine** (Nardil) block the action of monoamine oxidase (MAO), an enzyme in the central nervous system. Patients taking MAOIs must cut foods high in tyramine (found in aged cheeses and meats) out of their diet.
- **Bupropion** (Wellbutrin) is a heterocyclic antidepressant. The exact neurochemical mechanism of the drug is not known, but it has been effective in regulating bipolar depression in some patients. Side effects of bupropion include agitation, anxiety, confusion, tremor, dry mouth, fast or irregular heartbeat, headache, and insomnia.

ADJUNCT TREATMENTS. Adjunct treatments are used in conjunction with a long-term pharmaceutical treatment plan:

- Long-acting benzodiazepines (medications that ease tension by slowing the central nervous system) such as **clonazepam** (Klonapin) and **alprazolam** (Xanax) are used for rapid treatment of manic symptoms to calm and sedate patients until mania or hypomania have waned and mood-stabilizing agents can take effect. Sedation is a common effect, and clumsiness, lightheadedness, and slurred speech are other possible side effects of benzodiazepines.
- Neuroleptics (antipsychotic medications) such as **chlorpromazine** (Thorazine) and **haloperidol** (Haldol) are also used to control mania while a mood stabilizer such as lithium or valproate takes effect. Because neuroleptic side effects can be severe (difficulty in speaking or swallowing, paralysis of the eyes, loss of balance control, muscle spasms, severe restlessness, stiffness of arms and legs, tremors in fingers and hands, twisting movements of body, and weakness of arms and legs), benzodiazepines are generally preferred over neuroleptics.
- ECT, or **electroconvulsive therapy**, has a high success rate for treating both unipolar and bipolar depression, and mania. However, because of the convenience of drug treatment and the **stigma** sometimes attached to ECT therapy, ECT is usually employed after all pharmaceutical treatment options have been explored. ECT is given under anesthesia and patients are given a muscle relaxant medication to prevent convulsions. The treatment consists of a series of electrical pulses that move into the **brain** through electrodes on the patient's head. Although the exact mechanisms behind the success of ECT therapy are not known, it is believed that this electrical current alters the electrochemical processes of the brain, consequently relieving depression. Headaches, muscle soreness, nausea, and confusion are possible side effects immediately following an ECT procedure. Temporary memory loss has also been reported in ECT patients. In bipolar patients, ECT is often used in conjunction with drug therapy.

Calcium channel blockers (nimodipine, or Nimotop), typically used to treat angina and hypotension (low blood pressure), have been found effective, in a few small studies, for treating rapid cyclers. Calcium channel blockers stop the excess calcium buildup in cells that is thought to be a cause of bipolar disorder. They are usually used in conjunction with other drug therapies such as carbamazepine or lithium.

Clozapine (Clozaril) is an antipsychotic medication used to control manic episodes in patients who have not responded to typical mood-stabilizing agents. The drug has also been a useful prophylactic, or preventative treatment, in some bipolar patients. Common side effects of clozapine include tachycardia (rapid heart rate), hypotension, constipation, and weight gain. Agranulocytosis, a potentially serious but reversible condition in which the white blood cells that typically fight infection in the body are destroyed, is a possible side effect of clozapine. Patients treated with the drug should undergo weekly blood tests to monitor white blood cell counts.

Risperidone (Risperdal) is an antipsychotic medication that has been successful in controlling mania in several clinical trials when low doses were administered. The side effects of risperidone are mild compared to many other antipsychotics (constipation, coughing, diarrhea, dry mouth, headache, heartburn, increased length of sleep and dream activity, nausea, runny nose, sore throat, fatigue, and weight gain).

A new potential treatment for bipolar II disorder may be **gabapentin**, an anticonvulsant that may help treat mania. Recent reports indicate that gabapentin is effective for treating sudden onset bipolar II. Very recent evidence suggests, however, that gabapentin can potentially induce aggressive and disruptive behavior in children treated with this drug for seizures.

rTMS, or repeated transcranial magnetic stimulation is a new and still experimental treatment for the depressive phase of bipolar disorder. In rTMS, a large magnet is placed on the patient's head and magnetic fields of different frequency are generated to stimulate the left front cortex of the brain. Unlike ECT, rTMS requires no anesthesia and does not induce seizures.

Psychosocial interventions

Because bipolar disorder is thought to be biological in nature, psychological therapy is recommended as a companion to, but not a substitute for, pharmaceutical treatment of the disease. **Psychotherapy**, such as **cogni-**

tive-behavioral therapy, can be a useful tool in helping patients and their families adjust to the disorder, in encouraging **compliance** to a medication regimen, and in reducing the risk of **suicide**. Also, educative counseling is recommended for the patient and family.

In educative counseling, patients (and their families) learn of the high rates of social dysfunction and marital discord associated with this disorder. Patients also learn how their treatment will progress, which factors can affect treatment, and what kind of follow-up after treatment will be implemented. Genetic counseling should be a part of **family education** programs since this disorder is more prevalent among first-degree relatives of individuals with the disorder.

Social support for individuals with bipolar disorder is also important. Some people with the disorder, as well as their families, may find **support groups** helpful.

Alternative treatment

General recommendations include maintaining a calm environment, avoiding over-stimulation, getting plenty of rest, regular exercise, and proper diet. Some Chinese herbs may soften mood swings, but care must be taken (and good communication with the physician is essential) when combining herbal therapies with medications. **Biofeedback** is effective in helping some patients control symptoms such as irritability, poor self-control, racing thoughts, and sleep problems. A diet low in vanadium (a mineral found in meats and other foods) and high in vitamin C may be helpful in reducing depression.

Prognosis

While most patients will show some positive response to treatment, response varies widely, from full recovery to a complete lack of response to all drugs and/or ECT therapy. Drug therapies frequently need adjustment to achieve the maximum benefit for the patient. Bipolar disorder is a chronic recurrent illness in over 90% of those afflicted, and one that requires lifelong observation and treatment after diagnosis. Patients with untreated or inadequately treated bipolar disorder have a suicide rate of 15-25% and a nine-year decrease in life expectancy. With proper treatment, the life expectancy of the bipolar patient increases by nearly seven years and work productivity increases by ten years.

Prevention

The ongoing medical management of bipolar disorder is critical to preventing relapse (recurrence) of manic episodes. Even in carefully controlled treatment programs, bipolar patients may experience recurring episodes of the disorder. Patient education in the form of psychotherapy or **self-help groups** is crucial for training bipolar patients to recognize signs of mania and depression and to take an active part in their treatment program.

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ORGANIZATIONS

American Psychiatric Association. 1400 K Street NW, Washington DC 20005. (888) 357-7924. <http://www.psych.org>.

National Alliance for the Mentally Ill (NAMI). Colonial Place Three, 2107 Wilson Blvd., Ste. 300, Arlington, VA 22201-3042. (800) 950-6264. <http://www.nami.org>.

National Depressive and Manic-Depressive Association (NDMDA). 730 N. Franklin St., Suite 501, Chicago, IL 60610. (800) 826-3632. <http://www.ndmda.org>.

National Institute of Mental Health. Mental Health Public Inquiries, 5600 Fishers Lane, Room 15C-05, Rockville, MD 20857. (888) 826-9438. <http://www.nimh.nih.gov>.

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Bipolar disorders

Definition

Bipolar disorders is the name given to a group of mental disorders characterized by extreme fluctuations in

KEY TERMS

Bipolar disorder not otherwise specified—Disorder of mood involving mood swings that do not meet criteria for other disorders specified above.

Bipolar disorders—Disorders characterized by wide fluctuations in mood.

Bipolar I disorder—A major mood disorder characterized by full-blown manic episodes, often interspersed with episodes of major depression.

Bipolar II disorder—Disorder with major depressive episodes and mild manic episodes known as hypomania.

Cyclothymic disorder—A relatively mild mood disorder characterized by mood swings between mild depression to mild mania.

Depression—A mental state characterized by excessive sadness. Other symptoms include altered sleep patterns, thoughts of suicide, difficulty concentrating, agitation, lack of energy, and loss of enjoyment in activities that are usually pleasurable.

Hypomania—A milder form of mania which is characteristic of bipolar II disorder.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

mood. People diagnosed with bipolar disorders experience moods ranging from deepest depression to mania, often with periods of less extreme moods, or even emotional stability, in between.

Description

Individuals diagnosed with bipolar disorders experience fluctuations in mood over which they have no control. All of the bipolar disorders cause great emotional distress. Even the state of elevated mood, or "mania," might sound as if it would feel good; but it is, in fact, a painful, pressured feeling that is not at all pleasurable. People with mania find their thoughts running at an unstoppable pace; they cannot sleep, often for many nights at a time. Their speech may become rapid, and they may have grandiose ideas. Often people in manic states spend money they do not have, and make important but disastrous life decisions.

Individuals in the depressed mood state experience loss of interest in activities and people. They also experience loss of appetite, difficulty sleeping, lack of sexual desire, and an extreme loss of general energy. The ability to concentrate and think clearly is also compromised. Work, social, and family relationships are always impaired. Feelings of worthlessness and helplessness are common, as is the feeling that nothing will every improve. While depressed individuals may or may not report feeling "down" or "depressed," the feelings they do experience are very painful.

The handbook used by mental health professionals to diagnose mental disorders is the *Diagnostic and Statistical Manual of Mental Disorders,* 4th Edition, Text Revision, also known as the *DSM-IV-TR.* It includes four basic types of **bipolar disorder**: Bipolar I Disorder, Bipolar II Disorder, Cyclothymia, and Bipolar Disorder Not Otherwise Specified.

Bipolar I disorder is characterized by one or more manic episodes, or so-called "mixed" episodes, which involve both manic and depressive feelings alternating rapidly, often within the same day or week. Individuals with Bipolar I disorder may also experience one or more major depressive episodes. **Suicide** occurs in 10-15% of individuals with this disorder.

Bipolar II disorder is characterized by the occurrence of one or more major depressive episodes, interspersed with periods of mild manic episodes referred to as "hypomania." Hypomanic episodes are similar to manic ones, but are far less intense and less severe in their consequences. In fact, individuals may not see their hypomanic episodes as a problem, feeling, instead, that they have bursts of energy in which they can accomplish a great deal.

Cyclothymic disorder is a chronic, low-level disturbance of mood, punctuated by periods of depressive symptoms and periods of hypomanic symptoms. Cyclothymia often begins early in life, and people with the disorder may not know they have it; they may simply think of themselves as sadder and/or less energetic than other people, with occasional bursts of energy.

Bipolar disorder not otherwise specified is the term used in the *DSM-IV-TR* for individuals who do not meet the criteria for one of the other three diagnoses, but who nevertheless experience patterns of mood swings alternating between depression and mania.

See also Affect; Bipolar disorder; Cognitive-behavioral therapy; Cyclothymic disorder; Depression and depressive disorders; Dysthymic disorder; Lithium carbonate; Manic episodes

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Barbara Sternberg, Ph.D.

KEY TERMS

Body image—A term that refers to a person's inner picture of his or her outward appearance. It has two components: perceptions of the appearance of one's body, and emotional responses to those perceptions.

Comorbidity—Association or presence of two or more mental disorders in the same patient. A disorder that is said to have a high degree of comorbidity is likely to occur in patients diagnosed with other disorders that may share or reinforce some of its symptoms.

Delusion—A false belief that is resistant to reason or contrary to actual fact. Common delusions include delusions of persecution, delusions about one's importance (sometimes called delusions of grandeur), or delusions of being controlled by others. In BDD, the delusion is related to the patient's perception of his or her body.

Displacement—A psychological process in which feelings originating from one source are expressed outwardly in terms of concern or preoccupation with an issue or problem that the patient considers more acceptable. In some BDD patients, obsession about the body includes displaced feelings, often related to a history of childhood abuse.

Muscle dysmorphia—A subtype of BDD, described as excessive preoccupation with muscularity and bodybuilding to the point of interference with social, educational, or occupational functioning.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Somatoform disorders—A group of psychiatric disorders in the *DSM-IV-TR* classification that are characterized by external physical symptoms or complaints. BDD is classified as a somatoform disorder.

Body dysmorphic disorder

Definition

Body dysmorphic disorder (BDD) is defined by the *DSM-IV-TR* (a handbook for mental health professionals) as a condition marked by excessive preoccupation with an imaginary or minor defect in a facial feature or localized part of the body. The diagnostic criteria specify that the condition must be sufficiently severe to cause a decline in the patient's social, occupational, or educational functioning. The most common cause of this decline is the time lost in obsessing about the "defect." The *DSM-IV-TR* assigns BDD to the larger category of somatoform disorders, which are disorders characterized by physical complaints that appear to be medical in origin but that cannot be explained in terms of a physical disease, the results of substance abuse, or by another mental disorder.

Although cases of BDD have been reported in the psychiatric literature from a number of different countries for over a century, the disorder was first defined as a formal diagnostic category by the *DSM-III-R* in 1987. The word *dysmorphic* comes from two Greek words, *dys* that means "bad," or "ugly;" and, *morphos,* that means "shape," or "form." BDD was previously known as dysmorphophobia.

Description

BDD is characterized by an unusually exaggerated degree of worry or concern about a specific part of the face or body, rather than the general size or shape of the body. It is distinguished from **anorexia nervosa** and **bulimia nervosa**, to the extent that patients with these disorders are preoccupied with their overall weight and body shape. For example, an adolescent who thinks that her breasts are too large and wants to have plastic surgery to reduce their size but is otherwise unconcerned about her weight and is eating normally would be diagnosed with BDD, not anorexia or bulimia. As many as 50% of patients diagnosed with BDD undergo plastic surgery to correct their perceived physical defects.

Since the publication of *DSM-IV* in 1994, some psychiatrists have suggested that a subtype of BDD exists, which they term muscle dysmorphia. Muscle dysmorphia is marked by excessive concern with one's muscularity and/or fitness. Persons with muscle dysmorphia spend unusual amounts of time working out in gyms or exercising rather than dieting obsessively or looking into plastic surgery. Although gender stereotypes would suggest that women are more likely to develop BDD while men are more vulnerable to developing muscle dysmorphia, surveys indicate that both disorders have approximately equal gender ratios. *DSM-IV-TR* has additional references regarding body build and excessive weight lifting to *DSM-IV*'s description of BDD to accommodate muscle dysmorphia.

BDD and muscle dysmorphia can both be described as disorders resulting from the patient's distorted body image. Body image refers to the mental picture individuala have of their outward appearance, including size, shape, and form. It has two major components: how the people perceive their physical appearance, and how they feel about their body. Significant distortions in self-perception can lead to intense dissatisfaction with one's body and dysfunctional behaviors aimed at improving one's appearance. Some patients with BDD are aware that their concerns are excessive; others do not have this degree of insight. About 50% of patients diagnosed with BDD also meet the criteria for a **delusional disorder**, which is characterized by beliefs that are not based in reality.

The usual age of onset of BDD is late childhood or early adolescence; the average age of patients diagnosed with the disorder is 17. Ironically, even though BDD begins in childhood or adolescence, most research and treatment studies to date have been done on adults aged 35 and older.

BDD has a high rate of comorbidity, which means that people diagnosed with the disorder are highly likely to have been diagnosed with another psychiatric disorder— most commonly major depression, **social phobia**, or **obsessive-compulsive disorder** (OCD).

Causes and symptoms

Causes

The causes of BDD fall into two major categories, neurobiological and psychosocial.

NEUROBIOLOGICAL CAUSES. Research indicates that patients diagnosed with BDD have serotonin levels that are lower than normal. Serotonin is a neurotransmitter—a chemical produced by the **brain** that helps to transmit nerve impulses across the junctions between nerve cells. Low serotonin levels are associated with depression and other mood disorders.

PSYCHOSOCIAL CAUSES. Another important factor in the development of BDD is the influence of the mass media in developed countries, particularly the role of advertising in spreading images of physically "perfect" men and women. Impressionable children and adolescents absorb the message that anything short of physical perfection is unacceptable. They may then develop distorted perceptions of their own faces and bodies.

A young person's family of origin also has a powerful influence on his or her vulnerability to BDD. Children whose parents are themselves obsessed with appearance, dieting, and/or bodybuilding; or who are highly critical of their children's looks, are at greater risk of developing BDD.

An additional factor in some young people is a history of childhood trauma or abuse. Buried feelings about the abuse or traumatic incident emerge in the form of **obsession** about a part of the face or body. This "reassignment" of emotions from the unacknowledged true cause to another issue is called displacement. For example, an adolescent who frequently felt overwhelmed in childhood by physically abusive parents may develop a preoccupation at the high school level with muscular strength and power.

Symptoms

The central symptom of BDD is excessive concern with a specific facial feature or body part. Research done in the United Kingdom and the United States indicates that the features most likely to be the focus of the patient's attention are (in order of frequency) complexion flaws (acne, blemishes, scars, wrinkles); hair (on the head or the body, too much or too little); and facial features (size, shape, or lack of symmetry). The patient's concerns may, however, involve other body parts, and may shift over time from one feature to another.

Other symptoms of body dysmorphic disorder include:

- Ritualistic behavior. Ritualistic behavior refers to actions that the patient performs to manage anxiety and that take up excessive amounts of his or her time. Patients are typically upset if someone or something interferes with or interrupts their ritual. In the context of BDD, ritualistic behaviors may include exercise or makeup routines, assuming specific poses or postures in front of a mirror, etc.
- Camouflaging the "problem" feature or body part with makeup, hats, or clothing. Camouflaging appears to be

the single most common symptom among patients with BDD; it is reported by 94%.

- Abnormal behavior around mirrors, car bumpers, large windows, or similar reflecting surfaces. A majority of patients diagnosed with BDD frequently check their appearance in mirrors or spend long periods of time doing so. A minority, however, react in the opposite fashion and avoid mirrors whenever possible.
- Frequent requests for reassurance from others about their appearance.
- Frequently comparing one's appearance to others.
- Avoiding activities outside the home, including school and social events.

The loss of functioning resulting from BDD can have serious consequences for the patient's future. Adolescents with BDD often cut school and may be reluctant to participate in sports, join church- or civic-sponsored youth groups, or hold part-time or summer jobs. Adults with muscle dysmorphia have been known to turn down job promotions in order to have more time to work out in their gym or fitness center. Economic consequences of BDD also include overspending on cosmetics, clothing, or plastic surgery.

Demographics

As was mentioned earlier, BDD is primarily a disorder of young people. Its true incidence in the general population is unknown; however, it has been diagnosed in 1.9% of nonclinical patients and 12% of psychiatric outpatients. The *DSM-IV-TR* gives a range of 5%–40% for patients in clinical mental health settings diagnosed with anxiety or depressive disorders to be diagnosed with BDD. One community study published in 2001 found that 0.7% of women between the ages of 36 and 44 met the criteria for BDD. The disorder appears to be equally common in men and women.

As a result of gaps in research, little is known as of 2002 about the lifetime course of BDD or its prevalence in different ethnic or racial groups. The majority of patients in research studies to date have been Caucasians, but it is not clear whether this reflects racial patterns in the wider society or whether it represents referral bias, in that most study subjects are patients in private psychiatric hospitals. Anecdotal evidence, however, indicates that Asian Americans and African Americans with BDD are more likely to obsess about facial features or skin color that conflict with appearance ideals that dominate the mass media and have been derived from Caucasian people. Information through research done on the history of the American cosmetics industry reveals the startling statistic that African Americans spend three to five times as much money on personal care products as Caucasian Americans. In addition, successful African American and Asian American models, male as well as female, tend to resemble the Caucasian appearance ideal more than they deviate from it.

Diagnosis

The **diagnosis** of BDD in children and adolescents is often made by physicians in family practice because they are more likely to have developed long-term relationships of trust with the young people. With adults, it is often specialists in dermatology, cosmetic dentistry, or plastic surgery who may suspect that the patient suffers from BDD because of frequent requests for repeated or unnecessary procedures. Reported rates of BDD among dermatology and cosmetic surgery patients range between 6% and 15%. The diagnosis is made on the basis of the patient's history together with the physician's observations of the patient's overall mood and conversation patterns. People with BDD often come across to others as generally anxious and worried. In addition, the patient's dress or clothing styles may suggest a diagnosis of BDD.

As of 2002, there are no diagnostic questionnaires specifically for BDD, although a semi-structured interview called the BDD Data Form is sometimes used by researchers to collect information about the disorder from patients. The BDD Data Form includes demographic information, information about body areas of concern and the history and course of the illness, and the patient's history of **hospitalization** or **suicide** attempts, if any. The diagnostic questionnaire most frequently used to identify BDD patients is the Structured Clinical Interview for DSM-III-R Disorders, or SCID-II.

There are no brain **imaging studies** or laboratory tests as of 2002 that can be used to diagnose BDD.

Treatments

The standard treatment regimen for body dysmorphic disorder is a combination of medications and **psychotherapy**. Surgical, dental, or dermatologic treatments have been found to be ineffective.

Medications

The medications most frequently prescribed for patients with BDD are the selective serotonin reuptake inhibitors, most commonly **fluoxetine** (Prozac) or **sertraline** (Zoloft). Other SSRIs that have been used with this group of patients include **fluvoxamine** (Luvox) and **paroxetine** (Paxil). In fact, it is the relatively high rate of positive responses to SSRIs among BDD patients that led to the hypothesis that the disorder has a neurobiological

component related to serotonin levels in the body. An associated finding is that patients with BDD require higher dosages of SSRI medications to be effective than patients who are being treated for depression with these drugs.

Psychotherapy

The most effective approach to psychotherapy with BDD patients is **cognitive-behavioral therapy**, of which cognitive restructuring is one component. Since the disorder is related to **delusions** about one's appearance, cognitive-oriented therapy that challenges inaccurate self-perceptions is more effective than purely supportive approaches. Relaxation techniques also work well with BDD patients when they are combined wih cognitive restructuring.

Hospitalization

BDD patients have high rates of self-destructive behavior, including performing surgery on themselves at home (liposuction followed by skin stapling, sawing down teeth, and removing facial scars with sandpaper) and attempted or completed suicide. Many are unable to remain in school, form healthy relationships, or keep steady jobs. In one group of 100 patients diagnosed with BDD, 48% had been hospitalized for psychiatric reasons, and 30% had made at least one suicide attempt.

Alternative treatments

Although no alternative or complementary form of treatment has been recommended specifically for BDD, herbal remedies for depressed feelings, such as **St. John's wort**, have been reported as helping some BDD patients. **Aromatherapy** appears to be a useful aid to relaxation techniques as well as a pleasurable physical experience for BDD patients. **Yoga** has helped some persons with BDD acquire more realistic perceptions of their bodies and to replace obsessions about external appearance with new respect for the inner structure and functioning of their bodies.

Prognosis

As of 2002, researchers do not know enough about the lifetime course of body dysmorphic disorder to offer a detailed prognosis. The *DSM-IV-TR* notes that the disorder "has a fairly continuous course, with few symptom-free intervals, although the intensity of symptoms may wax and wane over time."

Prevention

Given the pervasive influence of the mass media in contemporary Western societies, the best preventive strategy involves challenging those afflicted with the disorder and who consequently have unrealistic images of attractive people. Parents, teachers, primary health care professionals, and other adults who work with young people can point out and discuss the pitfalls of trying to look "perfect." In addition, parents or other adults can educate themselves about BDD and its symptoms, and pay attention to any warning signs in their children's dress or behavior.

See also Aromatherapy; Yoga

Resources

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ORGANIZATIONS

American Academy of Child and Adolescent Psychiatry. 3615 Wisconsin Avenue, NW, Washington, DC 20016-3007. (202) 966-7300. Fax: (202) 966-2891. <www.aacap.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

Rebecca J. Frey, Ph.D.

Bodywork therapies

Definition

Bodywork therapies is a general term that refers to a group of body-based approaches to treatment that emphasize manipulation and realignment of the body's structure in order to improve its function as well as the client's mental outlook. These therapies typically combine a relatively passive phase, in which the client receives deep-tissue bodywork or postural correction from an experienced instructor or practitioner, and a more active period of movement education, in which the client practices sitting, standing, and moving about with better alignment of the body and greater ease of motion.

Bodywork should not be equated with massage simply speaking. Massage therapy is one form of bodywork, but in massage therapy, the practitioner uses oil or lotion to reduce the friction between his or her hands and the client's skin. In most forms of body work, little if any lubrication is used, as the goal of this type of hands-on treatment is to warm, relax and stretch the fascia (a band or sheath of connective tissue that covers, supports, or connects the muscles and the internal organs) and underlying layers of tissue.

Purpose

The purpose of bodywork therapy is the correction of problems in the client's overall posture, connective tissue, and/or musculature in order to bring about greater ease of movement, less discomfort, and a higher level of energy in daily activity. Some forms of bodywork have as a secondary purpose the healing or prevention of repetitive stress injuries, particularly for people whose occupations require intensive use of specific parts of the body (such as dancers, musicians, professional athletes, opera singers, etc.) Bodywork may also heal or prevent specific musculoskeletal problems, such as lower back pain or neck pain.

KEY TERMS

Bodywork—Any technique involving hands-on massage or manipulation of the body.

Endorphins—A group of peptide compounds released by the body in response to stress or traumatic injury. Endorphins react with opiate receptors in the brain to reduce or relieve pain sensations. Shiatsu is thought to work by stimulating the release of endorphins.

Fascia (plural, fasciae)—A band or sheath of connective tissue that covers, supports, or connects the muscles and the internal organs.

Ki—The Japanese spelling of qi, the traditional Chinese term for vital energy or the life force.

Meridians—In traditional Chinese medicine, a network of pathways or channels that convey qi (also sometimes spelled "ki"), or vital energy, through the body.

Movement education—A term that refers to the active phase of bodywork, in which clients learn to move with greater freedom and to maintain the proper alignment of their bodies.

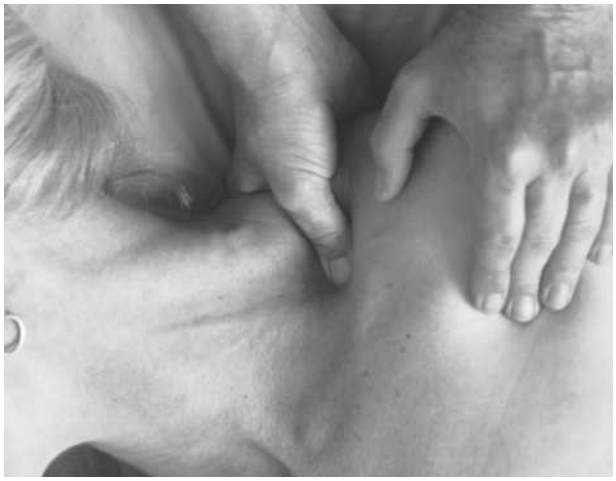
Repetitive stress injury (RSI)—A type of injury to the musculoskeletal and nervous systems associated with occupational strain or overuse of a specific part of the body. Bodywork therapies are often recommended to people suffering from RSIs.

Somatic education—A term used in both Hellerwork and the Feldenkrais method to describe the integration of bodywork with self-awareness, intelligence, and imagination.

Structural integration—The term used to describe the method and philosophy of life associated with Rolfing. Its fundamental concept is the vertical line.

Tsubo—In shiatsu, a center of high energy located along one of the body's meridians. Stimulation of the tsubos during a shiatsu treatment is thought to rebalance the flow of vital energy in the body.

Bodywork therapies are holistic in that they stress increased self-awareness and intelligent use of one's body as one of the goals of treatment. Some of these therapies use verbal discussion, visualization or guided imagery along with movement education to help clients break old patterns of moving and feeling. Although most bodywork therapists do not address mental disorders



Shiatsu, or acupressure, resembles acupuncture in its use of the basic concepts of ki, the vital energy that flows throughout the body, and the meridians, or 12 major pathways that channel ki to the various organs of the body. The shiatsu practitioner seeks out the meridians in the client's body through finger pressure, and stimulates points along the meridians, releasing energy that rebalances the body's energy level. *(Photo Researchers, Inc. Reproduced by permission.)*

directly in their work with clients, they are often knowledgeable about the applications of bodywork to such specific emotions as depession, anger, or fear.

Although some bodywork therapies, such as Rolfing or Hellerwork, offer programs structured around a specific number or sequence of lessons, all therapies of this type emphasize individualized treatment and respect for the uniqueness of each individual's body. Bodywork instructors or practitioners typically work with clients on a one-to-one basis, as distinct from a group or classroom approach.

Precautions

Persons who are seriously ill, acutely feverish, or suffering from a contagious infection should wait until they have recovered before beginning a course of bodywork. As a rule, types of bodywork that involve intensive manipulation or stretching of the deeper layers of body tissue are not suitable for persons who have undergone recent surgery or have recently suffered severe injury. In the case of Tragerwork, shiatsu, and trigger point therapy, clients should inform the therapist of any open wounds, bruises, or fractures so that the affected part of the body can be avoided during treatment. Craniosacral therapy, the Feldenkrais method, and the Alexander technique involve gentle touch and do not require any special precautions.

Persons who are recovering from **abuse** or receiving treatment for any post-traumatic syndrome or dissociative disorder should consult their therapist before undertaking bodywork. Although bodywork is frequently recommended as an adjunctive treatment for these disorders, it can also trigger flashbacks if the bodywork therapist touches a part of the patient's body associated with the abuse or trauma. Many bodywork therapists, however, are well informed about post-traumatic symptoms and disorders, and able to adjust their treatments accordingly.

Description

The following are brief descriptions of some of the more popular bodywork therapies.

Alexander technique

The Alexander technique was developed by an Australian actor named F. Matthias Alexander (1869-1955), who had voice problems that were not helped by any available medical treatments. Alexander decided to set up a number of mirrors so that he could watch himself during a performance from different angles. He found that he was holding his head and neck too far forward, and that these unconscious patterns were the source of the tension in his body that was harming his voice. He then developed a method for teaching others to observe the patterns of tension and stress in their posture and movement, and to correct these patterns with a combination of hands-on guidance and visualization exercises. As of 2002, the Alexander technique is included in the curricula of the Juilliard School of Music and many other drama and music schools around the world, because performing artists are particularly vulnerable to repetitive stress injuries if they hold or move their bodies incorrectly.

In an Alexander technique session, the client works one-on-one with an instructor who uses verbal explanations as well as guided movement. The sessions are usually referred to as "explorations" and last about 30 minutes. Although most clients see positive changes after only two or three sessions, teachers of the technique recommend a course of 20–30 sessions so that new movement skills can be learned and changes maintained.

Rolfing

Rolfing, which is also called Rolf therapy or structural integration, is a holistic system of bodywork that uses deep manipulation of the body's soft tissue to realign and balance the body's myofascial (muscular and connective tissue) structure. It was developed by Ida Rolf (1896-1979), a biochemist who became interested in the structure of the human body after an accident damaged her health. She studied with an osteopath as well as with practitioners of other forms of alternative medicine, and developed her own technique of body movement that she

called structural integration. Rolfing is an approach that seeks to counteract the effects of gravity, which tends to pull the body out of alignment over time and cause the connective tissues to stiffen and contract.

Rolfing treatment begins with the so-called "Basic Ten," a series of ten sessions each lasting 60–90 minutes, spaced a week or longer apart. After a period of integration, the client may undertake advanced treatment sessions. "Tune-up" sessions are recommended every six months. In Rolfing sessions, the practitioner uses his or her fingers, hands, knuckles, or elbows to rework the connective tissue over the client's entire body. The deep tissues are worked until they become pliable, which allows the muscles to lengthen and return to their proper alignment. Rolfing treatments are done on a massage table, with the client wearing only undergarments.

Hellerwork

Hellerwork is a bodywork therapy developed by Joseph Heller, a former NASA engineer who became a certified Rolfer in 1972 and started his own version of structural integration, called Hellerwork, in 1979. Heller describes his program as "a powerful system of somatic education and structural bodywork" based on a series of eleven sessions. Hellerwork is somewhat similar to Rolfing in that it begins with manipulation of the deep tissues of the body. Heller, however, decided that physical realignment of the body by itself is insufficient, so he extended his system to include movement education and "self-awareness facilitated through dialogue."

The bodywork aspect of Hellerwork is intended to release the tension that exists in the fascia, which is the sheath or layer of connective tissue that covers, supports, or connects the muscles and internal organs of the body. Fascia is flexible and moist in its normal state, but the effects of gravity and ongoing physical stresses lead to misalignments that cause the fascia to become stiff and rigid. The first hour of a Hellerwork session is devoted to deep connective tissue bodywork in which the Hellerwork practitioner uses his or her hands to release tension in the client's fascia. The bodywork is followed by movement education, which includes the use of video feedback to help clients learn movement patterns that will help to keep their bodies in proper alignment. The third component of Hellerwork is verbal dialogue, which is intended to help clients become more aware of the relationships between their emotions and attitudes and their body.

Tragerwork

Trager psychophysical integration, which is often called simply Tragerwork, was developed by Milton Trager (1908-1977), a man who was born with a spinal deformity and earned a medical degree in his middle age after working out an approach to healing chronic pain. Tragerwork is based on the theory that many illnesses are caused by tension patterns that are held in the unconscious mind as much as in the tissues of the body; clients are advised to think of Tragerwork sessions as "learning experiences" rather than "treatments." Tragerwork sessions are divided into bodywork, which is referred to as tablework, and an exercise period. Trager practitioners use their hands during tablework to perform a variety of gentle motions—rocking, shaking, vibrating, and gentle stretching—intended to help the client release their patterns of tension by experiencing how it feels to move freely and effortlessly on one's own. Following the tablework, clients are taught how to perform simple dance-like exercises called Mentastics, for practice at home. Tragerwork sessions take between 60–90 minutes, while clients are advised to spent 10–15 minutes three times a day doing the Mentastics exercises.

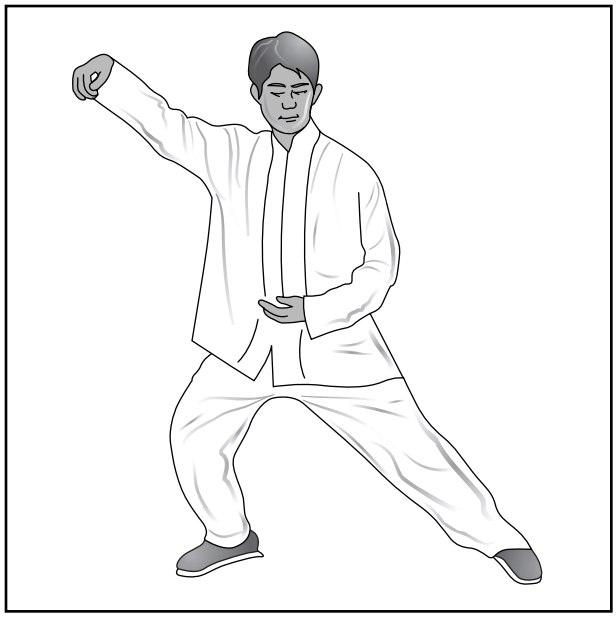
Feldenkrais method

The Feldenkrais method, like Hellerwork, refers to its approach as "somatic education." Developed by Moshe Feldenkrais (1904-1984), a scientist and engineer who was also a judo instructor, the Feldenkrais method consists of two major applications— Awareness Through Movement (ATM) lessons, a set of verbally directed exercise lessons intended to engage the client's intelligence as well as physical perception; and Functional Integration (FI), in which a Feldenkrais practitioner works with the client one-on-one, guiding him or her through a series of movements that alter habitual patterns and convey new learning directly to the neuromuscular system. Functional Integration is done with the client fully clothed, lying or sitting on a low padded table.

Perhaps the most distinctive feature of the Feldenkrais method is its emphasis on new patterns of thinking, attention, cognition, and imagination as byproducts of new patterns of physical movement. It is the most intellectually oriented of the various bodywork therapies, and has been described by one observer as "an unusual melding of motor development, biomechanics, psychology, and martial arts." The Feldenkrais method is the form of bodywork that has been most extensively studied by mainstream medical researchers.

Trigger point therapy

Trigger point therapy, which is sometimes called myotherapy, is a treatment for pain relief in the musculoskeletal system based on the application of pressure to trigger points in the client's body. Trigger points are defined as hypersensitive spots or areas in the muscles



T'ai chi is an ancient Chinese form of meditation with slow, smooth movements. The posture above is part of the single whip sequence of t'ai chi motions.

that cause pain when subjected to stress, whether the stress is an occupational injury, a disease, or emotional stress. Trigger points are not necessarily in the same location where the client feels pain.

Myotherapy is a two-step process. In the first step, the therapist locates the client's trigger points and applies pressure to them. This step relieves pain and also relaxes the muscles associated with it. In the second part of the therapy session, the client learns a series of exercises that progressively stretch the muscles that have been relaxed by the therapist's pressure. Most clients need fewer than 10 sessions to benefit from myotherapy. One distinctive feature of trigger point therapy is that clients are asked to bring a relative or trusted friend to learn the pressure technique and the client's personal trigger points. This "buddy system" helps the client to maintain the benefits of the therapy in the event of a relapse.

Shiatsu

Shiatsu is the oldest form of bodywork therapy, having been practiced for centuries in Japan as part of traditional medical practice. As of 2002, it is also the type of bodywork most commonly requested by clients in Western countries as well as in East Asia. The word *shiatsu* itself is a combination of two Japanese words that mean "pressure" and "finger." Shiatsu resembles **acupuncture** in its use of the basic concepts of ki, the vital energy that flows throughout the body, and the meridians, or 12 major pathways that channel ki to the various organs of the body. In Asian terms, shiatsu works by unblocking and rebalancing the distribution of ki in the body. In the categories of Western medicine, shiatsu may stimulate the release of endorphins, which are chemical compounds that block the receptors in the **brain** that perceive pain.

A shiatsu treatment begins with the practitioner's assessment of the client's basic state of health, including posture, vocal tone, complexion color, and condition of hair. This evaluation is used together with ongoing information about the client's energy level gained through the actual bodywork. The shiatsu practitioner works with the client lying fully clothed on a futon. The practitioner seeks out the meridians in the client's body through finger pressure, and stimulates points along the meridians known as tsubos. The tsubos are centers of high energy where the ki tends to collect. Pressure on the tsubos results in a release of energy that rebalances the energy level throughout the body.

Craniosacral therapy

Craniosacral therapy, or CST, is a form of treatment that originated with William Sutherland, an American osteopath of the 1930s who theorized that the manipulative techniques that osteopaths were taught could be applied to the skull. Sutherland knew from his medical training that the skull is not a single piece of bone, but consists of several bones that meet at seams; and that the cerebrospinal fluid that bathes the brain and spinal cord has a natural rise-and-fall rhythm. Sutherland experimented with gentle manipulation of the skull in order to correct imbalances in the distribution of the cerebrospinal fluid. Contemporary craniosacral therapists practice manipulation not only of the skull, but of the meningeal membranes that cover the brain and the spinal cord, and sometimes of the facial bones. Many practitioners of CST are also osteopaths, or DOs.

In CST, the patient lies on a massage table while the therapist gently palpates, or presses, the skull and spine. If the practitioner is also an osteopath, he or she will take a complete medical history as well. The therapist also "listens" to the cranial rhythmic impulse, or rhythmic pulsation of the cerebrospinal fluid, with his or her hands. Interruptions of the normal flow by abnormalities caused by tension or by injuries are diagnostic clues to the practitioner. Once he or she has identified the cause of the abnormal rhythm, the skull and spinal column are gently manipulated to restore the natural rhythm of the cranial impulse. Craniosacral therapy appears to be particularly useful in treating physical disorders of the head, including migraine headaches, ringing in the ears, sinus problems, and injuries of the head, neck, and spine. In addition, patients rarely require extended periods of CST treatments.

Preparation

Bodywork usually requires little preparation on the client's or patient's part, except for partial undressing for Rolfing, trigger point therapy, and Hellerwork.

Aftercare

Aftercare for shiatsu, trigger point therapy, and craniosacral therapy involves a brief period of rest after the treatment.

Some bodywork approaches involve various types of long-term aftercare. Rolfing clients return for advanced treatments or tune-ups after a period of integrating the changes in their bodies resulting frm the Basic Ten sessions. Tragerwork clients are taught Mentastics exercises to be done at home. The Alexander technique and the Feldenkrais approach assume that clients will continue to practice their movement and postural changes for the rest of their lives. Trigger point therapy clients are asked to involve friends or relatives who can help them maintain the benefits of the therapy after the treatment sessions are over.

Risks

The deep tissue massage and manipulation in Rolfing and Hellerwork are uncomfortable for many people, particularly the first few sessions. There are, however, no serious risks of physical injury from any form of bodywork that is administered by a trained practitioner of the specific treatment. As mentioned, however, bodywork therapies that involve intensive manipulation or stretching of the deeper layers of body tissue are not suitable for persons who have undergone recent surgery or have recently suffered severe injury.

Normal results

Normal results from bodywork include deep relaxation, improved posture, greater ease and spontaneity of movement, greater range of motion for certain joints, greater understanding of the structures and functions of the body and their relationship to emotions, and release of negative emotions.

Many persons also report healing or improvement of specific conditions, including migraine headaches, repetitive stress injuries, osteoarthritis, **insomnia**, sprains and bruises, sports injuries, stress-related illnesses, sciatica, postpregnancy problems, menstrual cramps, temporomandibular joint disorders, lower back pain, whiplash injuries, disorders of the immune system, asthma, depression, digestive problems, chronic **fatigue**, and painful scar tissue. The Alexander technique has been reported to ease the process of childbirth by improving the mother's postural alignment prior to delivery.

Some studies of the Feldenkrais method have found that its positive effects on subjects' self-esteem, mood, and anxiety sympoms are more significant than its effects on body function.

Abnormal results

Abnormal results from bodywork therapies would include serious physical injury or trauma-based psychological reactions.; Acupuncture; Energy therapies

Resources

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The Guild for Structural Integration. 209 Canyon Blvd. P.O. Box 1868. Boulder, CO 80306-1868. (303) 449-5903. (800) 530-8875. <www.rolfguild.org>.

Hellerwork. 406 Berry St. Mt. Shasta, CA 96067. (530) 926-2500. <www.hellerwork.com>.

International School of Shiatsu. 10 South Clinton Street, Doylestown, PA 18901. (215) 340-9918. Fax: (215) 340-9181. <www.shiatsubo.com>.

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OTHER

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NIH National Center for Complementary and Alternative Medicine (NCCAM) Clearinghouse. P. O. Box 8218, Silver Spring, MD 20907-8218. TTY/TDY: (888) 644-6226. Fax: (301) 495-4957. Web site: <http://www.nccam.nih.gov>.

Rebecca J. Frey, Ph.D.

Borderline personality disorder

Definition

Borderline personality disorder (BPD) is a mental disorder characterized by disturbed and unstable interpersonal relationships and self-image, along with impulsive, reckless, and often self-destructive behavior.

Description

Individuals with BPD have a history of unstable interpersonal relationships. They have difficulty interpreting reality and view significant people in their lives as either completely flawless or extremely unfair and uncaring (a phenomenon known as "splitting"). These alternating feelings of idealization and devaluation are the hallmark feature of borderline personality disorder. Because borderline patients set up such excessive and unrealistic expectations for others, they are inevitably disappointed when their expectations aren't realized.

The term "borderline" was originally used by **psychologist** Adolf Stern in the 1930s to describe patients whose condition bordered somewhere between **psychosis** and **neurosis**. It has also been used to describe the borderline states of consciousness these patients sometimes feel when they experience dissociative symptoms (a feeling of disconnection from oneself).

Causes and symptoms

Causes

Adults with borderline personalities often have a history of significant childhood traumas such as emotional, physical, and/or sexual **abuse** and parental **neglect** or loss. Feelings of inadequacy and self-loathing that arise from these situations may be key in developing the borderline personality. It has also been theorized that these patients try to compensate for the care they were denied in childhood through the idealized demands they now make on themselves and on others as adults. Some studies suggests that this disorder is associated with mood or impulse control problems, others implicate malfunctioning **neurotransmitters** (the chemicals that send messages to nerve cells). The disorder has a genetic correlation since it occurs more commonly among first-degree relatives.

Symptoms

The handbook used by mental health professionals to diagnose mental disorders is the *Diagnostic and Statistical Manual of Mental Disorders (DSM).* The 2000 edition of this manual (fourth edition, text revised) is known as the *DSM-IV-TR.* Published by the American Psychiatric Association, the *DSM* contains diagnostic criteria, research findings, and treatment information for mental disorders. It is the primary reference for mental health professionals in the United States. BPD was first listed as a disorder in the third edition *DSM-III,* which was published in 1980, and has been revised in subsequent editions.

The *DSM-IV-TR* requires that at least five of the following criteria (or symptoms) be present in an individual for a **diagnosis** of borderline disorder:

- frantic efforts to avoid real or perceived abandonment
- pattern of unstable and intense interpersonal relationships, characterized by alternating between idealization and devaluation ("love-hate" relationships)
- extreme, persistently unstable self-image and sense of self
- impulsive behavior in at least two areas (such as spending, sex, substance abuse, reckless driving, binge eating)

- recurrent suicidal behavior, gestures, or threats, or recurring acts of self-mutilation (such as cutting or burning oneself)
- unstable mood caused by brief but intense episodes of depression, irritability, or anxiety
- chronic feelings of emptiness
- inappropriate and intense anger, or difficulty controlling anger displayed through temper outbursts, physical fights, and/or sarcasm
- stress-related **paranoia** that passes fairly quickly and/or severe dissociative symptoms— feeling disconnected from one's self, as if one is an observer of one's own actions

Demographics

Borderline personality disorder accounts for 30–60% of all **personality disorders**, and is present in approximately 2% of the general population. The disorder appears to affect women more frequently than men—as many as 75% of all diagnosed patients are female.

Diagnosis

Borderline personality disorder typically first appears in early adulthood. Although the disorder may occur in adolescence, it may be difficult to diagnose, since borderline symptoms such as impulsive and experimental behaviors, insecurity, and mood swings are common—even developmentally appropriate—occurrences at this age.

Borderline symptoms may also be the result of chronic substance abuse and/or medical conditions (specifically, disorders of the central nervous system). These should be ruled out before making the diagnosis of borderline personality disorder.

BPD commonly occurs with mood disorders (i.e., depression and anxiety), **post-traumatic stress disorder** (PTSD), eating disorders, attention deficit/hyperactivity disorder (ADHD), and other personality disorders. It has also been suggested by some researchers that borderline personality disorder is not a true pathological condition in and of itself, but rather a number of overlapping personality disorders; it is, however, commonly recognized as a separate and distinct disorder by the American Psychiatric Association and by most mental health professionals. It is diagnosed by interviewing the patient, and matching symptoms to the *DSM* criteria. Supplementary testing may also be necessary.

Treatment

Individuals with borderline personality disorder seek psychiatric help and **hospitalization** at a much higher rate than people with other personality disorders, probably due to their fear of abandonment and their need to seek idealized interpersonal relationships. These patients represent the highest percentage of diagnosed personality disorders (up to 60%).

Providing effective therapy for the borderline personality patient is a necessary, but difficult, challenge. The therapist-patient relationship is subject to the same inappropriate and unrealistic demands that borderline personalities place on all their significant interpersonal relationships. They are chronic "treatment seekers" who become easily frustrated with their therapist if they feel they are not receiving adequate attention or empathy, and symptomatic anger, impulsivity, and self-destructive behavior can impede the therapist-patient relationship. However, their fear of abandonment, and of ending the therapy relationship, may actually cause them to discontinue treatment as soon as progress is made.

Psychotherapy, typically in the form of **cognitive-behavioral therapy**, is usually the treatment of choice for borderline personalities. Dialectical behavior therapy (DBT), a cognitive-behavioral technique, has emerged as an effective therapy for borderline personalities with suicidal tendencies. The treatment focuses on giving the borderline patient self-confidence and coping tools for life outside of treatment through a combination of social skill training, mood awareness and meditative exercises, and education on the disorder. **Group therapy** is also an option for some borderline patients, although some may feel threatened by the idea of "sharing" a therapist with others.

Medication is not considered a first-line treatment choice, but may be useful in treating some symptoms of the disorder and/or the mood disorders that have been diagnosed in conjunction with BPD. Recent clinical studies indicate that **naltrexone** may be helpful in relieving physical discomfort related to dissociative episodes.

Prognosis

The disorder usually peaks in young adulthood and frequently stabilizes after age 30. Approximately 75–80% of borderline patients attempt or threaten **suicide**, and between 8–10% are successful. If the borderline patient suffers from depressive disorder, the risk of suicide is much higher. For this reason, swift diagnosis and appropriate interventions are critical.

Prognosis

Prevention recommendations are scarce. The disorder may be genetic and not preventable. The only known

prevention would be to ensure a safe and nurturing environment during childhood.

See also Dissociation/Dissociative disorders

Resources

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BPD Central, National Alliance for the Mentally Ill. 200 N. Glebe Road, Suite 1015, Arlington, VA 22203-3754. (800) 950-6264. Web site: <http://www.bpdcentral.com>.

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Brain

Definition

The brain is the part of the central nervous system located in the skull. It controls the mental processes and physical actions of a human being.

Description

The brain, along with the spinal cord and network of nerves, controls information flow throughout the body, voluntary actions such as walking, reading, and talking, and involuntary reactions such as breathing and digestion.

The human brain is a soft, shiny, grayish-white, mushroom-shaped structure. The brain of an average adult weighs about 3 lb (1.4 kg). At birth, the average infant's brain weighs 13.7 oz (390 g); by age 15, it has nearly reached full adult size. The brain is protected by the skull and a three-layered membrane called the meninges. The brain's surface is covered with many bright red arteries and bluish veins that penetrate inward. Glucose, oxygen, and certain ions pass easily across the blood-brain barrier into the brain, although other substances, such as antibiotics, do not.

The four principal sections of the human brain are: the brain stem, the diencephalon, the cerebrum (divided into two large paired cerebral hemispheres), and the cerebellum.

The brain stem

The brain stem connects the brain with the spinal cord. Every message transmitted between the brain and spinal cord passes through the *medulla oblongata*—a part of the brain stem—via nerve fibers. The fibers on the right side of the medulla cross to the left and those on the left cross to the right. As a result, each side of the brain controls the opposite side of the body. The medulla regulates the heartbeat, breathing rate, and blood-vessel diameters; it also helps coordinate swallowing, vomiting, hiccuping, coughing, and sneezing.

Another brain stem component, the *pons* (meaning "bridge"), conducts messages between the spinal cord and the rest of the brain, and between the different parts of the brain. The *midbrain* conveys impulses between the cerebral cortex, pons, and spinal cord, and also contains visual and audio reflex centers involving the movement of the eyeballs and head.

Twelve pairs of *cranial nerves* originate in the underside of the brain, mostly from the brain stem. They leave the skull through openings and extend as peripheral nerves to their destinations. Among these cranial nerves are the olfactory nerves that bring messages about smell and the optic nerves that conduct visual information.

The diencephalon

The diencephalon lies above the brain stem and embodies the *thalamus* and *hypothalamus.* The thalamus is an important relay station for sensory information, interpreting sound, smell, taste, pain, pressure, temperature, and touch; it also regulates some emotions and memory. The hypothalamus controls a number of body functions, such as heartbeat and digestion, and helps regulate the endocrine system (hormonal system) and normal body temperature. The hypothalamus signals hunger and thirst, and also helps regulate sleep, anger, and aggression.

The cerebrum

Constituting nearly 90% of the brain's weight, the cerebrum is divided into specific areas that interpret sensory impulses. For example, spoken and written languages are transmitted to a part of the cerebrum called Wernicke's area where meaning is constructed. Motor areas control muscle movements. Broca's area translates thoughts into speech, and coordinates the muscles needed for speaking. Impulses from other motor areas direct hand muscles for writing and eye muscles for physical movement necessary for reading. The cerebrum is divided into left and right hemispheres. A deep fissure separates the two, with the corpus callosum, a large bundle of fibers, connecting them.

By studying patients whose corpora callosa had been destroyed, scientists realized that differences existed between the left and right sides of the cerebral cortex. The left side of the brain functions mainly in speech, logic, writing, and arithmetic. The right side, on the other hand, is more concerned with imagination, art, symbols, and spatial relations. In general, the left half of the brain controls the right side of the body, and vice versa. For most right-handed people (and many left-handed people as well), the left half of the brain is dominant.

The cerebrum's outer layer, the *cerebral cortex,* is composed of gray matter, which is made up of nerve cell bodies. About 0.08 in (2 mm) thick with a surface area about 5 sq ft (0.5 sq m), it's nearly half the size of an office desk. White matter, composed of nerve fibers covered with myelin sheaths, lies beneath the gray matter. During embryonic development, the gray matter grows faster than the white and folds in on itself, giving the brain its characteristic wrinkles, called *convolutions,* or *gyri;* the grooves between them are known as *sulci.*

The cerebellum

The cerebellum is located below the cerebrum and behind the brain stem. It is butterfly-shaped, with the "wings" known as the *cerebellar hemispheres;* the two halves are connected by the *vermis.* The cerebellum coordinates many neuromuscular functions, such as balance and coordination. Disorders related to damage of the cerebellum often result in ataxia (problems with coordination), dysarthria (unclear speech resulting from problems controlling the muscles used in speaking), and nystagmus (uncontrollable jerking of the eyeballs). A brain tumor that is relatively common in children known as medulloblastoma grows in the cerebellum.

KEY TERMS

Corpus callosum—(plural, corpora callosa) A thick bundle of nerve fibers lying deep in the brain that connects the two cerebral hemispheres and coordinates their functions.

Meninges—A membrane covering the brain and spinal cord that consists of three layers: the *pia mater,* the innermost layer; the *arachnoid,* in the middle; and the *dura mater,* the outermost layer.

Myelin sheaths—A fatty layer around nerve cells that aids the transmission of nerve impulses.

Peripheral nerve—A nerve in a distant location from the brain that receives information in the form of an impulse from the brain and spinal cord.

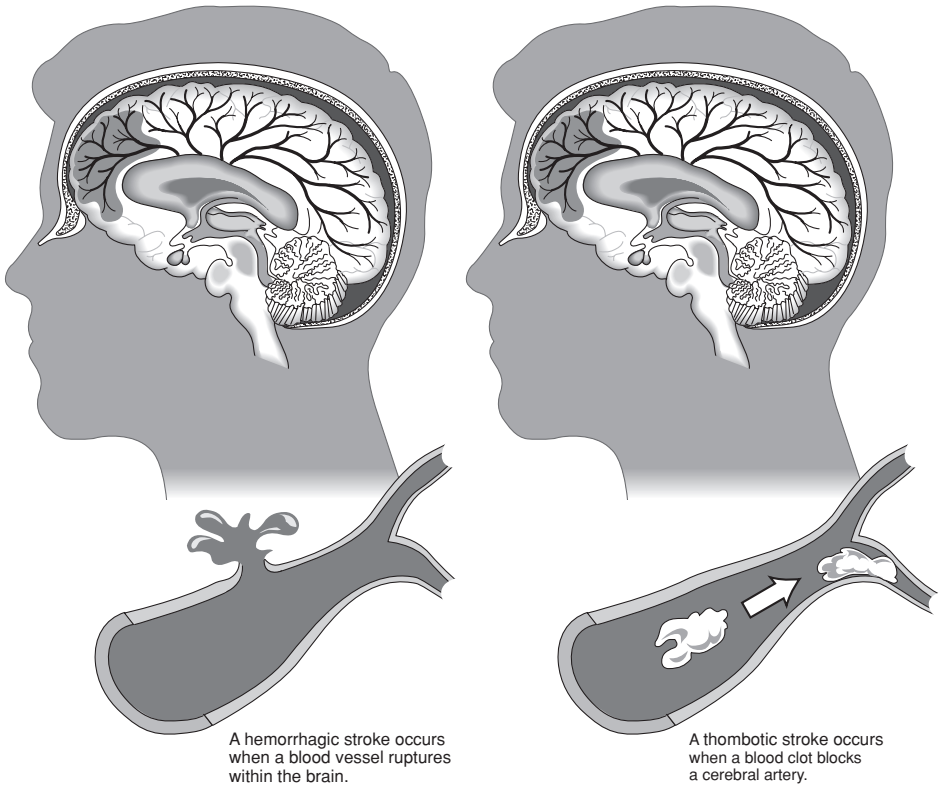
Studying the brain

Neurons carry information through the nervous system in the form of brief electrical impulses called *action potentials.* When an impulse reaches the end of an axon, **neurotransmitters** are released at junctions called *synapses.* The neurotransmitters are chemicals that bind to receptors on the receiving neurons, triggering the continuation of the impulse. Fifty different neurotransmitters have been discovered since the first was identified in 1920. By studying the chemical effects of neurotransmitters in the brain, scientists have developed treatments for mental disorders and are learning more about how drugs affect the brain.

Scientists once believed that brain cells do not regenerate, thereby making brain injuries and brain diseases untreatable. Since the late 1990s, however, researchers have been testing treatment for such patients with neuron transplants, which introduce nerve tissue into the brain, with promising results. They have also been investigating substances such as nerve growth factor (NGF), which may someday help regenerate nerve tissue.

Computerized brain imaging

Technology provides useful tools for researching the brain and helping patients with brain disorders. An electroencephalogram (EEG) records brain waves, which are produced by electrical activity in the brain. It is obtained by positioning electrodes on the head and amplifying the waves with an electroencephalograph. EEGs are valuable in diagnosing brain diseases such as epilepsy and tumors.



The human brain. *(Illustration by Hans & Cassady.)*

Scientists use three other techniques to study and understand the brain and diagnose disorders:

MAGNETIC RESONANCE IMAGING (MRI). Using a magnetic field to display the living brain at various depths, MRI can produce very clear and detailed pictures of brain structures. These images, which often appear as cross-sectional slices, are obtained by altering the main magnetic field of a specific brain area. MRI is particularly valuable in diagnosing damage to soft tissues, such as areas affected by head trauma or **stroke**. This is crucial when early **diagnosis** improves the chances of successful treatment. MRI also reveals tumors and other types of brain lesions.

POSITRON EMISSION TOMOGRAPHY (PET). During this test, a technician injects the patient with a small amount of a substance, such as glucose, that is marked with a radioactive tag. By tracking the radioactive substance as it travels to the brain, physicians can see almost immediately where glucose is consumed in the brain. This indicates brain activity, an important factor in diagnosing epilepsy, Alzheimer's, or Parkinson's. PET is also valuable in locating tumors and brain areas that have been affected by a stroke or blood clot.

MAGNETOENCEPHALOGRAPHY (MEG). Magnetoencephalography measures the electromagnetic fields created between neurons as electrochemical information is passed along. Of all brain-scanning methods, MEG provides the most accurate indicator of nerve cell activity, which can be measured in milliseconds. By combining an MRI with MEG, clinicians can get a noninvasive look at the brain that is especially useful in diagnosing epilepsy or migraines, for example. MEG also helps identify specific brain areas involved with different tasks. Any movement by the patient—wiggling the toes, for example—appears on the computer screen immediately as concentric colored rings. This pinpoints brain signals even before the toes are actually wiggled. Researchers foresee that these techniques could someday help paralysis victims move by supplying information on how to stimulate their muscles or indicating the signals needed to control an artificial limb.

See also Addiction; Nutrition and mental health

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KEY TERMS

Alveolar—Pertaining to alveoli, which are tiny air sacs at the ends of the small air passages in the lungs.

Apnea—A brief suspension or interruption of breathing.

Dyssomnia—A type of sleep disorder characterized by a problem with the amount, quality, or timing of the patient's sleep.

Hypoventilation—An abnormally low level of blood oxygenation in the lungs.

Polysomnogram—A machine that is used to diagnose sleep disorders by measuring and recording a variety of body functions related to sleep, including heart rate, eye movements, brain waves, muscle activity, breathing, changes in blood oxygen concentration, and body position.

Syndrome—A group of symptoms that together characterize a disease or disorder.

Tracheostomy—A surgical procedure in which an artificial opening is made in the patient's windpipe to relieve airway obstruction.

Breathing-related sleep disorder

Definition

Breathing-related sleep disorder is marked by sleep disruption caused by abnormal breathing during sleep. The most common complaint of individuals with breathing-related sleep disorder is excessive daytime sleepiness, brought on by frequent interruptions of nocturnal, or nighttime, sleep. A less frequent complaint is **insomnia** or inability to sleep.

Mental health professionals use the ***Diagnostic and Statistical Manual of Mental Disorders*** also known as the *DSM* to diagnose mental disorders. In the 2000 edition of this manual (the fourth edition, text revision, also known as *DSM-IV-TR*) breathing-related sleep disorder is listed as one of several different primary **sleep disorders**. Within the category of primary sleep disorders, it is classified as one of the dyssomnias, which are characterized by irregularities in the quality, timing, and amount of sleep.

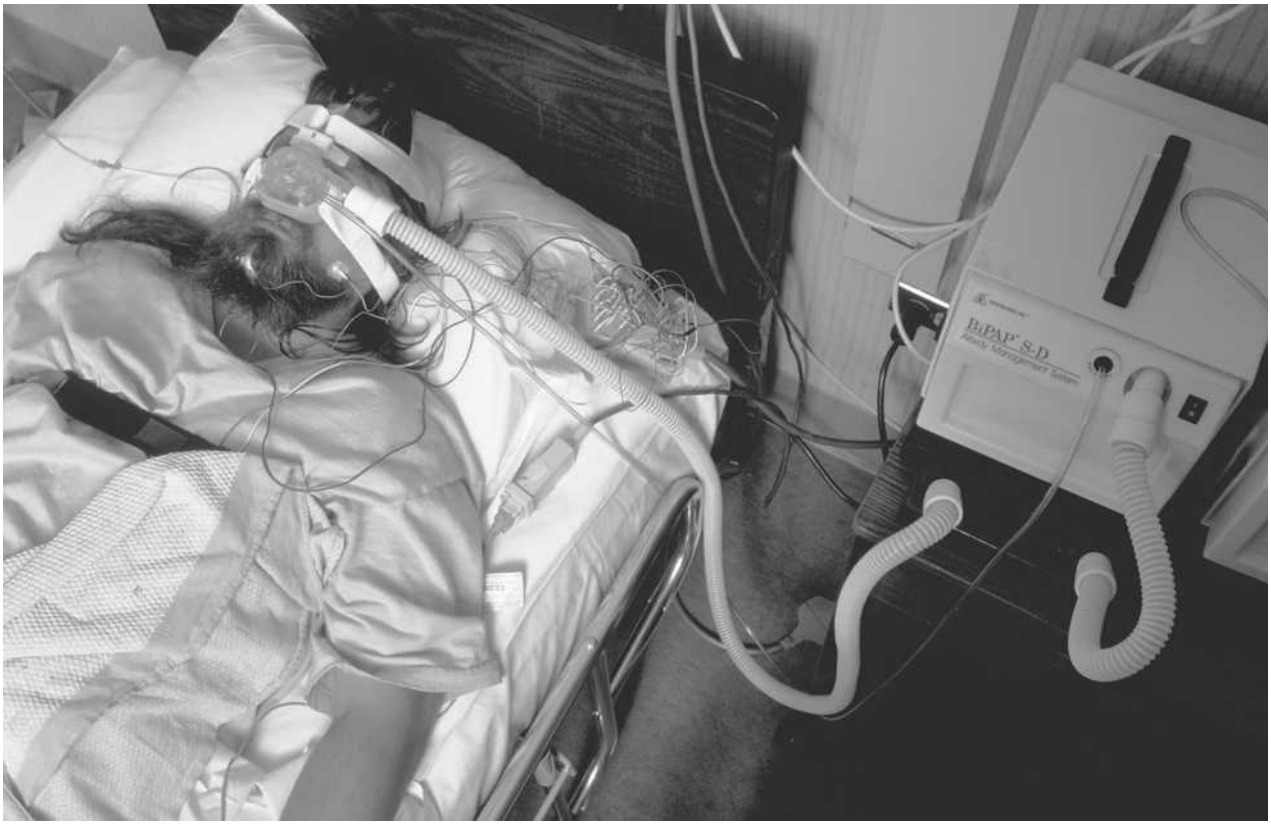
The *DSM-IV-TR* lists three types of breathing-related sleep disorder: obstructive sleep apnea syndrome (the most common type); central sleep apnea syndrome; and central alveolar hypoventilation syndrome.

Description

The most common feature of any breathing-related sleep disorder is interruption of the person's sleep leading to excessive daytime sleepiness. When the regular nighttime sleep of individuals is frequently interrupted, sleepiness at other times of the day is the usual result. People with breathing-related sleep disorder often find that they feel sleepy during such relaxing activities as reading or watching a movie. With extreme cases, the person may feel so sleepy that he or she falls asleep during activities that require alertness, such as talking, walking, or driving.

Other people with breathing-related sleep disorder report having insomnia, or the inability to sleep. Patients also find that their sleep does not refresh them; they may awaken frequently during sleep, or have difficulty breathing while sleeping or lying down.

The two sleep apnea syndromes that are listed as subtypes of breathing-related sleep disorder are characterized by episodes of airway blockage or stopping of breathing during sleep. Sleep apnea is potentially deadly. The other type of breathing-related sleep disorder, central alveolar hypoventilation syndrome, is distinguished from the other two subtypes by the fact that the reduced oxygen content of the blood is caused by shallow breathing. The alveoli, which are the tiny air sacs in the lung tissue, are not able to oxygenate the blood efficiently because the person is not breathing deeply enough. Shallow breathing often occurs when people are awake and is common in severely overweight individuals.



A patient suffering from acute sleep apnea is hooked up to monitors in preparation for a night's sleep at a Stanford University lab. *(Russell D. Curtis. National Audubon Society Collection/ Photo Researchers, Inc. Reproduced by permission.)*

Causes and symptoms

Causes

Many persons with the obstructive sleep apnea syndrome subtype of breathing-related sleep disorder are overweight. The symptoms often grow worse as the person's weight increases. Persons who have obstructive sleep apnea and are not overweight often have breathing passages that are narrowed by swollen tonsils, abnormally large adenoids, or other abnormalities of the various structures of the mouth and throat.

Central sleep apnea syndrome is often associated with cardiac or neurological conditions affecting airflow regulation. It is a disorder that occurs most frequently in elderly patients.

Patients diagnosed with central alveolar hypoventilation syndrome experience a breathing impairment related to abnormally low arterial oxygen levels.

Symptoms

Obstructive sleep apnea syndrome, which is the most common type of breathing-related sleep disorder, is marked by frequent episodes of upper-airway obstruction during sleep. Patients with this syndrome alternate between loud snores or gasps and silent periods that usually last for 20–30 seconds. The snoring is caused by the partial blockage of the airway. The silent periods are caused by complete obstruction of the airway, which makes the patient's breathing stop.

Obstructive sleep apnea syndrome is also common in children with enlarged tonsils. The symptoms of any breathing-related sleep disorder in children are often subtle and more difficult to diagnose. Children under five are more likely to demonstrate such nighttime symptoms as apnea and breathing difficulties. Children over five are more likely to demonstrate such daytime symptoms as sleepiness and attention difficulties.

Persons with central sleep apnea syndrome experience periods when the oxygenation of blood in the lungs temporarily stops during sleep; but they do not suffer airway obstruction. Although these patients may snore, their snoring is usually mild and not a major complaint.

Central alveolar hypoventilation syndrome is characterized by excessive sleepiness and insomnia.

Demographics

The majority of patients with the obstructive sleep apnea type of breathing-related sleep disorder are overweight, middle-aged males. Adult males are two to four times as likely as adult females to experience obstructive sleep apnea syndrome. Among children the male: female ratio is 1:1.

Central sleep apnea syndrome is most common in the elderly.

Diagnosis

A **diagnosis** of breathing-related sleep disorder usually requires a thorough physical examination of the patient. The patient may be referred to an otorhinolaryngologist (a doctor who specializes in disorders of the ear, nose, and throat) for a detailed evaluation of the upper respiratory tract. The physical examination is followed by observation of the patient in a sleep clinic or laboratory. Breathing patterns, including episodes of snoring and apnea, are evaluated when the patient is connected to a device called a polysomnogram. The polysomnogram uses a set of electrodes to measure several different body functions associated with sleep, including heart rate, eye movements, **brain** waves, muscle activity, breathing, changes in blood oxygen concentration, and body position. Interviews are also conducted with the patient and his or her partner.

To meet criteria for the diagnosis of breathing-related sleep disorder, the patient must experience interruptions of sleep leading to insomnia or excessive sleepiness that have been determined to result from one of the following sleep-related breathing conditions: obstructive sleep apnea syndrome; central sleep apnea syndrome; or central alveolar hypoventilation syndrome.

The disturbance in sleep must also not be better accounted for by another mental disorder or by a general medical condition not related to breathing.

The disturbance in sleep must not be due to the direct effects on the body of a prescription medication or drug of abuse.

Treatments

Weight loss is a key to effective treatment of overweight people with breathing-related sleep disorder. It is often considered the first step in treating any disorder involving sleep apnea. Increased exercise and reduced-calorie **diets** are the most important components of an effective weight loss regimen.

Nasal continuous positive airway pressure therapy, also known as nasal CPAP therapy, is a popular form of treatment for the obstructive sleep apnea subtype of breathing-related sleep disorder. Nasal CPAP therapy, which has been in use since 1981, involves the use of a high-pressure blower that delivers continuous air flow to a mask worn by the patient during sleep. The airflow from the nasal CPAP blower is often very effective in reducing or eliminating sleep apnea episodes. Nasal CPAP treatment is, however, inconvenient and somewhat noisy for anyone who must share a bedroom with the patient. Patients do not always comply with this form of treatment; one study indicated that about 25% of patients who are treated with nasal CPAP therapy stop using it within a year.

Medications for patients with the sleep apnea subtype of breathing-related sleep disorder are most commonly such respiratory stimulants as medroxyprogesterone acetate (Depo-Provera) and acetazolamide (Diamox). **Protriptyline** (Vivactil), which is a tricyclic antidepressant, is also used for some patients.

Surgery to relieve airway obstruction is increasingly preferred by many patients. If the airway obstruction is related to anatomical structures that are narrowing the airway, surgical reshaping of the soft palate and uvula (a small, conical-shaped piece of tissue attached to the middle of the soft palate) may be performed. Another surgical procedure that is sometimes done in very obese patients with obstructive sleep apnea is a tracheostomy, or an artificial opening made in the windpipe. This operation has a number of unpleasant side effects, however, and so is usually reserved for patients whose breathing-related disorder is life-threatening.

Patients with sleep apnea are advised to abstain from alcohol and sedative medications, which are often given to patients who display any type of sleeping irregularities. Alcohol and sedatives often increase the likelihood of upper airway problems during sleep.

Prognosis

Breathing-related sleep disorder often has a gradual long-term progression and a chronic course. For this reason, many people have the disorder for years before seeking treatment. For many, symptoms worsen during middle age, causing people to seek treatment at that point.

Successful treatment of other conditions, such as **obesity**, cardiac or neurological conditions in the elderly, or enlarged tonsils in children often aids in the treatment of breathing-related sleep disorder. Weight loss often leads to spontaneous resolution of the disorder.

Prevention

Because overweight people are more likely to develop the more common obstructive sleep apnea type of breathing-related sleep disorder, a good preventive measure is effective weight management. Good general health and treatment of related physiological conditions are also effective in preventing the disorder.

See also Circadian rhythm sleep disorder; Obesity

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American Sleep Apnea Association. 1424 K Street NW, Suite 302, Washington DC 20005. <http://www.sleepapnea.org>.

American Sleep Disorders Association. 6301 Bandel Road NW, Suite 101, Rochester, MN 55901. <http://www.asda.org>.

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Brief psychotic disorder

Definition

Brief psychotic disorder is a short-term, time-limited disorder. An individual with brief psychotic disorder has experienced at least one of the major symptoms of **psychosis** for less than one month. **Hallucinations**, **delusions**, strange bodily movements or lack of movements (catatonic behavior), peculiar speech and bizarre or markedly inappropriate behavior are all classic psychotic symptoms that may occur in brief psychotic disorder.

The cause of the symptoms helps to determine whether or not the sufferer is described as having brief psychotic disorder. If the psychotic symptoms appear as a result of a physical disease, a reaction to medication, or intoxication with drugs or alcohol, then the unusual behaviors are not classified as brief psychotic disorder. If hallucinations, delusions, or other psychotic symptoms occur at the same time that an individual is experiencing major clinical depression or bipolar (manic-depressive) disorder, then the brief psychotic disorder **diagnosis** is not given. The decision rules that allow the clinician to identify this cluster of symptoms as brief psychotic disorder are outlined in the ***Diagnostic and Statistical Manual of Mental Disorders*** Fourth Edition Text Revision, produced by the American Psychiatric Association. This manual is referred to by most mental health professionals as *DSM-IV-TR*.

Description

Positive symptoms

The person experiencing brief psychotic disorder always has one or more "positive" psychotic symptoms. The psychotic symptoms are not "positive" in the everyday sense of something being good or useful. Positive in this context is used with the medical meaning: a factor is present that is not normally expected, or a normal type of behavior is experienced in its most extreme form. **Positive symptoms** of psychosis include hallucinations, delusions, strange bodily movements or lack of movements (catatonic behavior), peculiar speech and bizarre or primitive behavior.

HALLUCINATIONS. Hallucinations involve experiencing sensations that have no corresponding objective reality. Hallucinations can occur in various forms that parallel the human senses. Visual hallucinations involve the sense of sight, or "seeing things." Auditory hallucinations generally involve hearing voices, and are the most common of the hallucinations. Sometimes, a hallucination can include both voices and some visual experience; mental health professionals describe this as an "auditory-visual hallucination." Smelling non-existent smells or feeling things on or under one's skin that do not actually exist are forms of somatic hallucinations. Somatic comes from *soma*, the Greek word for body; thus, somatic hallucinations are bodily hallucinations.

DELUSIONS. Delusions are also a classic psychotic feature. Delusions are strongly held irrational and unrealistic beliefs that are extremely difficult to change, even

when the person is exposed to evidence that contradicts the delusion. The layperson typically thinks of delusions as being "paranoid," or "persecutory" wherein the delusional person is excessively suspicious and continually feels at the mercy of conspirators who are "out to get" him or her. However, delusions can also be unjustified beliefs that are grandiose, involve elaborate love fantasies ("erotomanic" delusions), or extreme and irrational jealousy. Grandiose delusions are persistent irrational beliefs that somehow exaggerate the person's importance, such as believing oneself to be a famous person, or having an enviable position such as being the Prime Minister or President. Often grandiose delusions take on religious overtones; for instance, a person might become convinced that she is the Virgin Mary. Furthermore, delusions can be somatic. Somatic delusions are erroneous but strongly held beliefs about the characteristics or functioning of one's body; an example is a mental health consumer who refuses to eat because of a conviction that the throat muscles are completely paralyzed and that only liquids can be swallowed, when there is no actual physical reason to be unable to swallow.

OTHER PSYCHOTIC SYMPTOMS. Other psychotic symptoms that may occur in brief psychotic disorder are strange bodily movements or lack of movements (catatonic behavior), peculiar speech, and bizarre or child-like behavior. Catatonic behavior or **catatonia** involves both possible extremes related to movement. Catalepsy is the motionless aspect of catatonia—a person with catalepsy may remain fixed in the same position for hours on end. Rapid or persistently repeated movements, frequent grimacing and strange facial expressions, and unusual gestures are the opposite end of the catatonia phenomenon. Peculiar speech is also seen in some cases of brief psychotic disorder. Speech distortions can involve words mixed together in no coherent order, responses that are irrelevant and strange in the context of the conversation in which they occur, or echolalia, the repetition of another person's exact spoken words, repeated either immediately after the speaker or after a delay of minutes to hours. Bizarre behavior can range from child-like behaviors such as skipping, singing, or hopping in inappropriate circumstances to unusual practices such as hoarding food or covering one's head and clothing with aluminum foil wrappings.

Of course, not all of these psychotic symptoms will be observed simultaneously in the person with brief psychotic disorder. Any constellation of these positive psychotic symptoms that occurs for one entire day up to one month is considered to be brief psychotic disorder, unless there is some other syndrome or biological cause that caused the symptoms to appear.

Causes and symptoms

Causes

Brief psychotic disorder is not a simple or consistent disorder with a single cause. Because many phenomena can prompt a short-term experience of psychotic symptoms, there are several ways of viewing the causes of the disorder.

AN EARLY PHASE OF SCHIZOPHRENIA. Because of the similarities between brief psychotic disorder, **schizophreniform disorder** and **schizophrenia**, many clinicians have come to think of brief psychotic disorder as being the precursor to a lengthier psychotic disorder. Although this can only be identified retrospectively, brief psychotic disorder is often the diagnosis that was originally used when an individual (who later develops schizophrenia) experiences a first "psychotic break" from more typical functioning.

A STRESS RESPONSE. At times, under severe **stress**, temporary psychotic reactions may appear. The source of stress can be from typical events encountered by many people in the course of a lifetime, such as being widowed or divorced. The severe stress may be more unusual, such as being in combat, enduring a natural disaster, or being taken hostage. The person generally returns to a normal method of functioning when the stress decreases or more support is available, or better coping skills are learned.

POSTPARTUM PSYCHOSIS. In some susceptible women, dramatic hormonal changes in childbirth and shortly afterward can result in a form of brief psychotic disorder often referred to as *postpartum psychosis*. Unfortunately, postpartum conditions are often misidentified and improperly treated. In many cases of a mother killing her infant or committing **suicide**, postpartum psychosis is involved.

DEFENSE MECHANISM IN PERSONALITY DISORDER. Persons with **personality disorders** appear to be more susceptible to developing brief psychotic reactions in response to stress. Individuals with personality disorders have not developed effective adult mechanisms for coping with life. When life becomes more demanding and difficult than can be tolerated, the person may lapse into a brief psychotic state.

CULTURALLY DEFINED DISORDER. Culture is a very important factor in understanding mental health and psychological disturbance, and brief psychotic disorder is an excellent example. The types of behavior that occur during brief psychotic disorder are very much shaped by the expectations and traditions of the individual's culture. Many cultures have some form of mental disorder that would meet criteria for brief psychotic disorder the features of which are unique to that culture, wherein most

KEY TERMS

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

sufferers have similar behaviors that are attributed to causes that are localized to that community. The *DSM-IV-TR* calls disorders unique to certain societies or groups "culture-bound." An example of a culture-bound syndrome is *koro*, a syndrome observed in Japan and some other areas of Asia but not elsewhere. Koro is an **obsession** to the point of delusion with the possibility that the genitals will retract or shrink into the body and cause death.

Conversely, while culture shapes the form a psychotic reaction may take, culture also determines what is *not* to be considered psychotic. Behaviors that in one culture would be thought of as bizarre or psychotic, may be acceptable in another. For example, some cultural groups and religions view "speaking in tongues" as a valuable expression of the gifts of God, whereas viewed out of context, the unrecognizable speech patterns might be viewed as psychotic. If the behaviors shown are culturally acceptable in the person's society or religion, and happen in an approved setting such as a religious service, then brief psychotic disorder would not be diagnosed.

Symptoms

DSM-IV-TR provides three major criteria for brief psychotic disorder:

- At least one positive symptom of psychosis, from the following symptoms: delusions; hallucinations; disorganized speech which is strange, peculiar, difficult to comprehend; disorganized (bizarre or child-like) behavior; or catatonic behavior.
- Limited duration. The psychotic symptoms have occurred for at least one day but less than one month. There is an eventual return to normal level of functioning.
- The symptoms are not biologically influenced or attributable to another disorder. In other words, the symptoms cannot be occurring as part of a mood disorder, **schizoaffective disorder**, or schizophrenia, and they cannot be due to intoxication with drugs or alcohol. Further, the symptoms cannot be an adverse reaction to a medication, and they cannot be caused by a physical injury or medical illness.

Demographics

The actual rate of brief psychotic disorder is unknown, although it appears to be fairly rare in the United States and other developed countries. While psychotic reactions that occur and subside in under a month are more common in non-industrialized nations, the mental disorders wherein psychotic symptoms last longer than one month are more prevalent in developed countries. The disorder appears to be more common in adolescents and young adults than in those of middle age or older.

Diagnosis

Using the *DSM-IV-TR* criteria previously listed makes identification of the disorder relatively clear-cut. However, an unusual aspect to this diagnosis is the emphasis on the length of time that symptoms have been evident. Most mental health disorder diagnoses do not include the duration of the symptoms as part of their definitions. However, the length of time the person has had psychotic symptoms is one of the major distinctions among three different psychotic disorders. Brief psychotic disorder involves the shortest duration of suffering psychotic symptoms: one day to one month. Schizophreniform disorder also involves the individual showing signs of psychosis, but for a longer period (one month or more, but less than six months). Schizophrenia is diagnosed in individuals who have evidenced psychotic symptoms that are not associated with physical disease, mood disorder or intoxication, for six months or longer. Another complicating factor in making the diagnosis is the context in which the "psychotic symptoms" are experienced. If the psychotic-like behaviors evidenced are acceptable in the person's culture or religion and these behaviors happen in a traditionally expected context such as a religious service or **meditation**, then brief psychotic disorder would not be diagnosed.

The disorder is usually diagnosed by obtaining information in interview from the client and possibly from immediate family. Also, the diagnostician would be likely to perform a semi-structured interview called a mental status examination, which examines the person's ability to concentrate, to remember, to realistically understand the situation, and to think logically.

Treatments

Antipsychotic medications are very effective in ending a brief psychotic episode. A number of different antipsychotics are used for the purpose of terminating

acute psychotic episodes. **Haloperidol** (Haldol) is most commonly used if the psychotic symptoms are accompanied by *agitation*. Agitation is a state of frantic activity that is often accompanied by anger or fearfulness; when in an agitated state, the client is more likely to cause harm to self or others. In agitated psychotic states, the haloperidol is often given as an injection, accompanied by other medications that decrease anxiety (**lorazepam**, also known as Ativan) and slow behavior (**diphenhydramine**, also known as Benadryl). If the client is not agitated, usually a newer-generation antipsychotic is used, given daily as tablets, capsules or liquid, for a lengthier period of time. The novel antipsychotic that would be used is likely to be one of the following: **olanzapine** (Zyprexa), **quetiapine** (Seroquel), or **risperidone** (Risperdal). Hormones may also be prescribed for postpartum psychosis. Supportive therapy may also prove helpful in some situations, in decreasing the client's anxiety and educating the client about the psychiatric illness. In culture-bound syndromes, the most effective treatment is often the one that is societally expected; for example, bathing in a river viewed as sacred might be a usual method of curing the psychotic-like state, in a particular culture.

Prognosis

The prognosis is fairly positive in brief psychotic disorder because by its own definition, a return to normal functioning is expected. If there is a major life event as a stress or an unusual traumatic experience that initiated the episode, chances are very good that there will be no recurrence. If there is not a particular triggering event or if the episode occurred in an individual with a personality disorder, the likelihood of recurrence is higher. If an episode is a recurrence without a specific triggering event, then the beginnings of the development of schizophrenia or **bipolar disorder** may be at hand, in which case the prognosis is poor. In the individual with personality disorder, the pattern may recur in response to stress, so that there are intermittent experiences of brief psychotic disorder over the course of a lifetime.

Prevention

In women who have experienced brief postpartum psychosis, one prevention option is to forgo having additional children. If a postpartum psychosis has occurred in the past, in subsequent pregnancies the physician may be proactive in prescribing an antipsychotic medication regimen to be taken in the postpartum period in order to prevent psychotic symptoms from recurring. Severe stressors can be a trigger for brief psychotic disorder in many cases. Therefore, in response to identifiable extreme stressors, such as natural disasters or terrorist attacks, strong social support and immediate post-crisis counseling could possibly prevent the development of brief psychotic disorder in susceptible persons.

See also Borderline personality disorder; Delirium; Dementia; Postpartum depression; Post-traumatic stress disorder; Schizotypal personality disorder; Substance abuse

Resources

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Bulimia nervosa

Definition

Bulimia nervosa is an eating disorder characterized by **binge eating** and engaging in inappropriate ways of counteracting the bingeing (using laxatives, for example) in order to prevent weight gain. The word "bulimia" is the Latin form of the Greek word *boulimia*, which means "extreme hunger." A binge is consuming a larger amount of food within a limited period of time than most people would eat in similar circumstances. Most people with bulimia report feelings of loss of control associated with bingeing, and some have mildly dissociative experiences in the course of a binge, which means that they feel disconnected from themselves and from reality when they binge.

KEY TERMS

Binge—An excessive amount of food consumed in a short period of time. Usually, while a person binge eats, he or she feels disconnected from reality, and feels unable to stop. The bingeing may temporarily relieve depression or anxiety, but after the binge, the person usually feels guilty and depressed.

Body image—A term that refers to a person's inner picture of his or her outward appearance. It has two components: perceptions of the appearance of one's body, and emotional responses to those perceptions.

Comorbidity—Association or presence of two or more mental disorders in the same patient. A disorder that is said to have a high degree of comorbidity is likely to occur in patients diagnosed with other disorders that may share or reinforce some of its symptoms.

Cortisol—A steroid hormone released by the cortex (outer portion) of the adrenal gland when a person is under stress.

Diuretic—A medication or substance given to increase the amount of urine excreted.

Dysthymic disorder—A mood disorder that is less severe than depression but usually more chronic.

Electrolytes—Substances or elements that dissociate into electrically charged particles (ions) when dissolved in the blood. The electrolytes in human blood include potassium, magnesium, and chloride.

Hypokalemia—Abnormally low levels of potassium in the blood. Hypokalemia is a potential medical emergency, as it can lead to disturbances in of the heart rhythm. Muscle cramps and pain are a common symptom of hypokalemia in bulimic patients.

Incisors—The four teeth in the front of each jaw in humans. The incisors of patients with bulimia frequently show signs of erosion from stomach acid.

Ipecac—The dried root of *Caephalis ipecacuanha*, a South American plant. Given in syrup form, ipecac is most commonly used to induce vomiting in cases of accidental poisoning.

Petechiae—Pinpoint-sized hemorrhages in the skin or a mucous membrane. In bulimia, petechiae may appear in the skin around the eyes as a result of increased pressure in the capillaries caused by vomiting.

Purging—Inappropriate actions taken to prevent weight gain, often after bingeing, including self-induced vomiting or the misuse of laxatives, diuretics, enemas, or other medications.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

The handbook for mental health professionals to aid in **diagnosis** is the ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM-IV-TR*. This book categorizes bulimia nervosa as an eating disorder, along with **anorexia nervosa**.

Description

Bulimia nervosa is classified into two subtypes according to the methods used by the patient to prevent weight gain after a binge. The purging subtype of bulimia is characterized by the use of self-induced vomiting, laxatives, enemas, or diuretics (pills that induce urination); in the nonpurging subtype, fasting or overexercising is used to compensate for binge eating.

The onset of bulimia nervosa is most common in late adolescence or early adult life. Dieting efforts and body dissatisfaction, however, often occur in the teenage years. For these reasons, it is often described as a developmental disorder. Although genetic researchers have identified specific genes linked to susceptibility to eating disorders, the primary factor in the development of bulimia nervosa is environmental **stress** related to the onset of puberty. Girls who have strongly negative feelings about their bodies in response to puberty are at high risk for developing bulimia.

The binge eating associated with bulimia begins most often after a period of strict dieting. Most people with bulimia develop purging behaviors in response to the bingeing. Vomiting is used by 80%–90% of patients

diagnosed with bulimia. The personal accounts of recovered bulimics suggest that most "discover" vomiting independently as a way of ridding themselves of the food rather than learning about it from other adolescents. Vomiting is often done to relieve an uncomfortable sensation of fullness in the stomach following a binge as well as to prevent absorption of the calories in the food. Vomiting is frequently induced by touching the gag reflex at the back of the throat with the fingers or a toothbrush, but a minority of patients use syrup of ipecac to induce vomiting. About a third of bulimics use laxatives after binge eating to empty the digestive tract, and a minority use diuretics or enemas. Purging behaviors lead to a series of digestive and metabolic disturbances that then reinforce the behaviors.

A small proportion of bulimics exercise excessively or fast after a binge instead of purging.

Patients with bulimia may come to the attention of a **psychiatrist** because they develop medical or dental complications of the eating disorder. In some cases, the adolescent's dentist is the "case finder." In many cases, however, the person with bulimia seeks help.

Causes and symptoms

Causes

As of 2002, bulimia nervosa is understood to be a complex disorder with multiple factors contributing to its development. Researchers presently disagree about the degree of influence exerted by genetic factors, psychological patterns in the family of origin, and social trends.

GENETIC. Two recently published reviews (in 1999 and 2000) suggest that there is some heritability for bulimia. In other words, these articles suggest that there is a genetic component to bulimia. **Neurotransmitters** are chemicals that pass chemical messages along from nerve cell to nerve cell, and people with bulimia have abnormal levels of certain neurotransmitters. Some observers have suggested that these abnormalities in the levels of central nervous system neurotransmitters may also be influenced by genetic factors.

FAMILY OF ORIGIN. A number of recent studies point to the interpersonal relationships in the family of origin (the patient's family while growing up) as a factor in the later development of bulimia. People with bulimia are more likely than people with anorexia to have been sexually abused in childhood; studies have found that abnormalities in blood levels of serotonin (a neurotransmitter associated with mood disorders) and cortisol (the primary stress hormone in humans) in bulimic patients with a history of childhood sexual **abuse** resemble those in patients with **post-traumatic stress disorder**. Post-traumatic stress disorder is a mental disorder that can develop after someone has experienced a traumatic event (horrors of war, for example) and is unable to put that event behind him or her— the disorder is characterized by very realistic flashbacks of the traumatic event.

A history of eating conflicts and struggles over food in the family of origin is also a risk factor for the development of bulimia nervosa. Personal accounts by recovered bulimics frequently note that one or both parents were preoccupied with food or dieting. Fathers appear to be as influential as mothers in this regard.

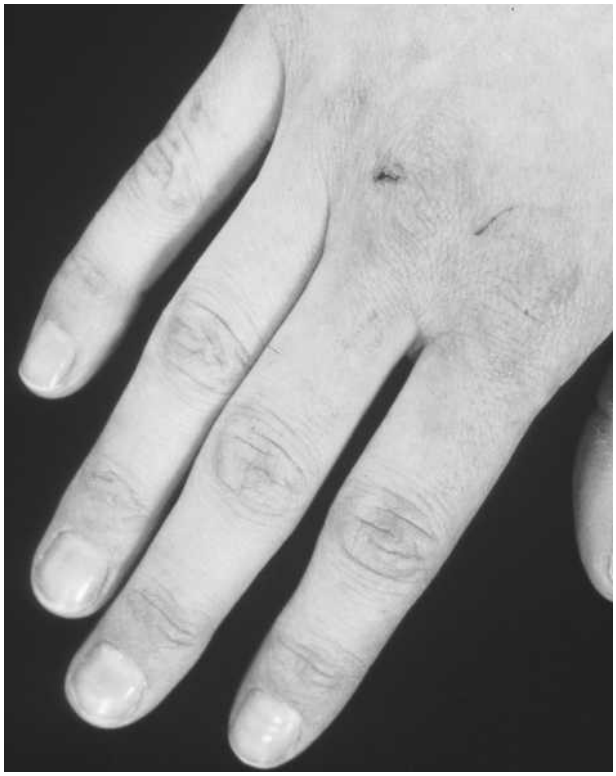
An additional risk factor for early-onset bulimia is interest in or preparation for a sport or occupation that requires strict weight control, such as gymnastics, figure skating, ballet, and modeling.

SOCIOCULTURAL CAUSES. Emphasis in the mass media on slenderness in women as the primary criterion of beauty and desirability is commonly noted in studies of bulimia. Historians of fashion have remarked that the standard of female attractiveness has changed over the past half century in the direction of greater slenderness; some have commented that Marilyn Monroe would be considered "fat" by contemporary standards. The ideal female figure is not only unattainable by the vast majority of women, but is lighter than the standards associated with good health by insurance companies. In 1965 the average model weighed 8% less than the average American woman; as of 2001 she weighs 25% less.

Another factor mentioned by intellectual historians is the centuries-old split in Western philosophy between mind and body. Instead of regarding a human person as a unified whole comprised of body, soul, and mind, Western thought since Plato has tended to divide human nature in a dualistic fashion between the life of the mind and the needs of the body. Furthermore, this division was associated with gender symbolism in such a way that the life of the mind was associated with masculinity and the needs of the body with femininity. The notion that the "superior" mind should control the "inferior" physical dimension of human life was correlated with men's physical, legal, and economic domination of women. Although this dualistic pattern of symbolic thought is no longer a conscious part of the Western mindset, it appears to influence Western culture on a subterranean level.

A number of different theories have been put forward to explain the connections between familial and social factors and bulimia. Some of these theories maintain that:

- Bulimia results from a conflict between mother and daughter about nurturing and dependency. Girls are typically weaned earlier than boys and fed less. The bulimic's bingeing and purging represent a conflict



The cuts on the knuckles shown in this photograph are due to the teeth breaking the skin during self-induced vomiting. *(B. Bodine/Custom Medical Stock Photo, Inc. Reproduced by permission.)*

between wanting comfort and believing that she does not deserve it.

- Bulimia develops when an adolescent displaces larger conflicts about being a woman in a hypersexualized society onto food. Many writers have commented about the contradictory demands placed on women in contemporary society— for example, to be sexually appealing yet "untouchable" at the same time. Controlling body size and food intake becomes a simplified solution to a very complex problem of personal identity and moral standards.
- Bulimia is an **obsession** with food that the culture encourages in order to protect men from competition from intellectually liberated women. Women who are spending hours each day thinking about food, or bingeing and purging, do not have the emotional and intellectual energy to take their places in the learned professions and the business world.
- Bulimia expresses a fear of fat rooted in childhood memories of mother's size relative to one's own.
- Bulimia results from intensified competition among women for professional achievement (getting a desirable job or a promotion, or being accepted into graduate or professional school) as well as personal success (getting a husband), because studies have indicated that businesses and graduate programs discriminate against overweight applicants.
- Bulimia results from attempts to control emotional chaos in one's interpersonal relationships by imposing rigid controls on food intake.

Nutrition experts have pointed to the easy availability of foods high in processed carbohydrates in developed countries as a social factor that contributes to the incidence of bulimia. One study found that subjects who were given two slices of standard mass-produced white bread with some jelly had their levels of serotonin increased temporarily by 450%. This finding suggests that bulimics who binge on ice cream, bread, cookies, pizza, and fast food items that are high in processed carbohydrates are simply manipulating their neurochemistry in a highly efficient manner. The incidence of bulimia may be lower in developing countries because **diets** that are high in vegetables and whole-grain products but low in processed carbohydrates do not affect serotonin levels in the **brain** as rapidly or as effectively.

Symptoms

The *DSM-IV-TR* specifies that bingeing and the inappropriate attempts to compensate for it must occur twice a week for three months on average to meet the diagnostic criteria for bulimia nervosa.

A second criterion of bulimia nervosa is exaggerated concern with body shape and weight. Bulimia can be distinguished from **body dysmorphic disorder** (BDD) by the fact that people with BDD usually focus on a specific physical feature— most commonly a facial feature— rather than overall shape and weight. Bulimics do, however, resemble patients with BDD in that they have distorted body images.

Bulimia is associated with a number of physical symptoms. Binge eating by itself rarely causes serious medical complications, but it is associated with nausea, abdominal distension and cramping, slowed digestion, and weight gain.

Self-induced vomiting, on the other hand, may have serious medical consequences, including:

- Erosion of tooth enamel, particularly on the molars and maxillary incisors. Loss of tooth enamel is irreversible.
- Enlargement of the salivary glands.
- Scars and calloused areas on the knuckles from contact with the teeth.

- Irritation of the throat and esophagus from contact with stomach acid.
- Tearing of mucous membranes in the upper gastrointenstinal tract or perforation of the esophagus and stomach wall. Perforation of part of the digestive tract is a rare complication of bulimia but is potentially fatal.
- Electrolyte imbalances. The loss of fluids from repeated vomiting and laxative abuse can deplete the body's stores of hydrogen chloride, potassium, sodium, and magnesium. Hypokalemia (abnormally low levels of potassium in the blood) is a potential medical emergency that can lead to muscle cramps, **seizures**, and heart arrhythmias.

Other physical symptoms associated with bulimia include irregular menstrual periods or amenorrhea; petechiae (pinhead-sized bruises from capillaries ruptured by increased pressure due to vomiting) in the skin around the eyes and rectal prolapse (the lowering of the rectum from its usual position).

Demographics

Bulimia nervosa affects between 1% and 3% of women in the developed countries; its prevalence is thought to have increased markedly since 1970. The rates are similar across cultures as otherwise different as the United States, Japan, the United Kingdom, Australia, South Africa, Canada, France, Germany, and Israel. About 90% of patients diagnosed with bulimia are female as of 2002, but some researchers believe that the rate of bulimia among males is rising faster than the rate among females.

The average age at onset of bulimia nervosa appears to be dropping in the developed countries. A study of eating disorders in Rochester, Minnesota over the 50 years between 1935 and 1985 indicated that the incidence rates for women over 20 remained fairly constant, but there was a significant rise for women between 15 and 20 years of age. The average age at onset among women with bulimia was 14 and among men, 18.

In terms of sexual orientation, gay men appear to be as vulnerable to developing bulimia as heterosexual women, while lesbians are less vulnerable.

Recent studies indicate that bulimia in the United States is no longer primarily a disorder of Caucasian women; the rates among African American and Hispanic women have risen faster than the rate of bulimia for the female population as a whole. One report indicates that the chief difference between African American and Caucasian bulimics in the United States is that the African American patients are less likely to eat restricted diets between episodes of binge eating.

Diagnosis

The diagnosis of bulimia nervosa is made on the basis of a physical examination, a psychiatric assessment, the patient's eating history, and the findings of laboratory studies. Patients who do not meet the full criteria for bulimia nervosa may be given the diagnosis of subsyndromal bulimia or of eating disorder not otherwise specified (EDNOS).

Physical examination

Patients suspected of having bulimia nervosa should be given a complete physical examination because the disorder has so many potential medical complications. In addition, most bulimics are close to normal weight or only slightly overweight, and so do not look outwardly different from most people of their sex in their age group. The examination should include not only vital signs and an assessment of the patient's height and weight relative to age, but also checking for such signs of bulimia as general hair loss, abdominal soreness, swelling of the parotid glands, telltale scars on the back of the hand, petechiae, edema, and teeth that look ragged or "moth-eaten."

Psychiatric assessment

Psychiatric assessment of patients with bulimia usually includes four components:

- A thorough history of body weight, eating patterns, diets, typical daily food intake, methods of purging (if used), and concept of ideal weight.
- A history of the patient's significant relationships with parents, siblings, and peers, including present or past physical, emotional, or sexual abuse.
- A history of previous psychiatric treatment (if any) and assessment of comorbid (occurring at the same time as the bulimia) mood, anxiety, substance abuse, or **personality disorders**.
- Administration of standardized instruments that measure attitudes toward eating, body size, and weight. Common tests for eating disorders include the Eating Disorder Examination; the Eating Disorder Inventory; the Eating Attitude Test, or EAT; and the Kids Eating Disorder Survey.

Laboratory findings

Laboratory tests ordered for patients suspected of having bulimia usually include a complete blood cell count, blood serum chemistry, thyroid tests, and urinalysis. If necessary, the doctor may also order a chest x ray and an electrocardiogram (EKG). Typical findings in

patients with bulimia include low levels of chloride and potassium in the blood, and higher than normal levels of amylase, a digestive enzyme found in saliva.

Treatments

Treatment for bulimia nervosa typically involves several therapy approaches. It is, however, complicated by several factors.

First, patients diagnosed with bulimia nervosa frequently have coexisting psychiatric disorders that typically include major depression, **dysthymic disorder**, anxiety disorders, substance abuse disorders, or personality disorders. In the case of depression, the mood disorder may either precede or follow the onset of bulimia, and, with bulimia, the prevalence of depression is 40%–70%. With regard to substance abuse, about 30% of patients diagnosed with bulimia nervosa abuse either alcohol or stimulants over the course of the eating disorder. The personality disorders most often diagnosed in bulimics are the so-called Cluster B disorders— borderline, narcissistic, histrionic, and antisocial. **Borderline personality disorder** is a disorder characterized by stormy interpersonal relationships, unstable self-image, and impulsive behavior. People with **narcissistic personality disorder** believe that they are extremely important and are unable to have empathy for others. Individuals with **histrionic personality disorder** seek attention almost constantly and are very emotional. **Antisocial personality disorder** is characterized by a behavior pattern of a disregard for others' rights— people with this disorder often deceive and mainpulate others. A number of clinicians have noted that patients with bulimia tend to develop impulsive and unstable personality disturbances whereas patients with anorexia tend to be more obsessional and perfectionistic. Estimates of the prevalence of personality disorders among patients with bulimia range between 2% and 50%. The clinician must then decide whether to treat the eating disorder and the comorbid conditions concurrently or sequentially. It is generally agreed, however, that a substance abuse disorder, if present, must be treated before the bulimia can be effectively managed. It is also generally agreed that mood disorders and bulimia can be treated concurrently, often using antidepressant medication along with therapy.

Second, the limitations on treatment imposed by **managed care** complicate the treatment of bulimia nervosa. When the disorder first received attention in the 1970s, patients with bulimia were often hospitalized until the most significant physical symptoms of the disorder could be treated. As of 2002, however, few patients with bulimia are hospitalized, with the exception of medical emergencies related to electrolyte imbalances and gastrointestinal injuries associated with the eating disorder. Most treatment protocols for bulimia nervosa now reflect cost-containment measures.

Medications

The most common medications given to patients are antidepressants, because bulimia is so closely associated with depression. Short-term medication trials have reported that tricyclic antidepressants— **desipramine**, **imipramine**, and **amitriptyline** — reduce episodes of binge eating by 47%–91% and vomiting by 45%–78%. The monoamine oxidase inhibitors are not recommended as initial medications for patients diagnosed with bulimia because of their side effects. The most promising results have been obtained with the selective serotonin reuptake inhibitors, or SSRIs. **Fluoxetine** (Prozac) was approved in 1998 by the Food and Drug Administration (FDA) for the treatment of bulimia nervosa. Effective dosages of fluoxetine are higher for the treatment of bulimia than they are for the treatment of depression. Although a combination of medication and **cognitive-behavioral therapy** is more effective in treating most patients with bulimia than medication alone, one team of researchers reported success in treating some bulimics who had not responded to **psychotherapy** with fluoxetine by itself.

A newer type of medication that shows promise in the treatment of bulimia nervosa is ondansetron, a drug that was originally developed to control nausea from chemotherapy and radiation therapy for cancer. Ondansetron acts to control the transmission of signals in nerves leading to the vagus nerve, which in turn governs feelings of fullness and the vomiting reflex. A British study reported that ondansetron normalized several aspects of eating behaviors in all the patients who received it during the study.

In addition to antidepressant or antinausea medications, such acid-reducing medications as cimetidine and ranitidine, or antacids, may be given to patients with bulimia to relieve discomfort in the digestive tract associated with irritation caused by stomach acid.

Psychotherapy

Cognitive-behavioral therapy (CBT) is regarded as the most successful psychotherapeutic approach to bulimia nervosa. CBT is intended to interrupt the faulty thinking processes associated with bulimia, such as preoccupations with food and weight, black-white thinking ("all or nothing" thinking, or thinking thoughts only at extreme ends of a spectrum) and low self-esteem, as well as such behaviors as the binge-purge cycle. Patients are first helped to regain control over their food intake by keeping

food diaries and receiving feedback about their meal plans, symptom triggers, nutritional balance, etc. They are then taught to challenge rigid thought patterns as well as receiving **assertiveness training** and practice in identifying and expressing their feelings in words rather than through distorted eating patterns. About 50% of bulimic patients treated with CBT are able to stop bingeing and purging. Of the remaining half, some show partial improvement and a small minority do not respond at all.

Family therapy is sometimes recommended as an additional mode of treatment for patients with bulimia who come from severely troubled or food-obsessed families that increase their risk of relapsing.

Other mainstream therapies

Medical nutrition therapy, or MNT, is a recognized component of the treatment of eating disorders. Effective MNT for patients with bulimia involves an understanding of cognitive-behavioral therapy as well as the registered dietitian's usual role of assisting the physician with monitoring the patient's physical symptoms, laboratory values, and vital signs. In the treatment of bulimia, the dietitian's specialized knowledge of nutrition may be quite helpful in dealing with the myths about food and fad diets that many bulimic patients believe. The dietitian's most important task, however, is helping the patient to normalize her or his eating patterns in order to break the deprivation/bingeing cycle that is characteristic of bulimia nervosa. Calorie intake is usually based on retaining the patient's weight in order to prevent hunger, since hunger increases susceptibility to bingeing.

Recent studies in upstate New York have found that bright **light therapy**, of the type frequently prescribed for **seasonal affective disorder** (SAD), appears to be effective in reducing binge eating in patients diagnosed with bulimia. It also significantly relieved depressive symptoms, as measured by the patients' scores on the **Beck Depression Inventory**.

Alternative and complementary treatments

Alternative therapies that have been shown to be helpful for some patients in relieving the anxiety and muscular soreness associated with bulimia nervosa include **acupuncture**, massage therapy, hydrotherapy, and shiatsu.

Herbal remedies that have been used to calm digestive upsets in bulimic patients include teas made from **chamomile** or peppermint. Peppermint helps to soothe the intestines by slowing down the rate of smooth muscle contractions (peristalsis). Chamomile has been used to help expel gas from the digestive tract, a common complaint of bulimics. Both herbs have a wide margin of safety.

Some bulimic patients have responded well to **yoga** because its emphasis on focused breathing and **meditation** calls attention to and challenges the distorted thought patterns that characterize bulimia. In addition, the stretching and bending movements that are part of a yoga practice help to displace negative thoughts focused on the body's outward appearance with positive appreciation of its strength and agility. Lastly, since yoga is noncompetitive, it allows bulimics to explore the uniqueness of their bodies rather than constantly comparing themselves to other people.

Prognosis

The prognosis of bulimia depends on several factors, including age at onset, types of purging behaviors used (if any), and the presence of other psychiatric conditions or disorders. In many cases, the disorder becomes a chronic (long-term) condition; 20%–50% of patients have symptoms for at least five years in spite of treatment. The usual pattern is an alternation between periods of remission and new episodes of bingeing. Patients whose periods of remission last for a year or longer have a better prognosis; patients diagnosed with major depression or a personality disorder have a less favorable prognosis. Overall, however, the prognosis for full recovery from bulimia nervosa is considered relatively poor compared to other eating disorders.

Bulimia nervosa appears to produce changes in the functioning of the serotonin system in the brain. Serotonin is a neurotransmitter. A team of researchers at the University of Pittsburgh who compared brain images taken by **positron emission tomography** (PET) from bulimic women who had been in remission for a year or longer with brain images from healthy women found that the recovered bulimics did not have a normal age-related decline in serotonin binding. Since serotonin helps to regulate mood, appetite, and impulse control, the study may help to explain why some women may be more susceptible to developing bulimia than others.

Prevention

As of 2002, the genetic factors in bulimia are not well understood. With regard to family influences, an important study published in December 2001 reported that the presence of eating problems in early childhood is a strong predictor of eating disorders in later life. The longitudinal study of 800 children and their mothers was based on psychiatric assessments of the subjects made in 1975, 1983, 1985, and 1992. The researchers found that a diagnosis of bulimia nervosa in early adolescence is associated with a nine-fold increase in risk for late adolescent bulimia and a 20-fold increase in risk for adult bulimia. Late adolescent

bulimia nervosa is associated with a 35-fold increase in risk for adult bulimia nervosa. Given these findings, the most important preventive measure that can be taken in regard to bulimia nervosa is the establishment of healthful eating patterns and attitudes toward food in the family of origin.

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Academy for Eating Disorders. Montefiore Medical School, Adolescent Medicine, 111 East 210th Street, Bronx, NY 10467. (718) 920-6782.

American Academy of Child and Adolescent Psychiatry. 3615 Wisconsin Avenue, NW, Washington, DC 20016-3007. (202) 966-7300. Fax: (202) 966-2891. <www.aacap.org>.

American Anorexia/Bulimia Association. 165 West 46th Street, Suite 1108, New York, NY 10036. (212) 575-6200.

American Dietetic Association. (800) 877-1600. <www.eatright.org>.

Anorexia Nervosa and Related Eating Disorders, Inc. (ANRED). P.O. Box 5102, Eugene, OR 97405. (541) 344-1144. <www.anred.com>.

Center for the Study of Anorexia and Bulimia. 1 W. 91st St., New York, NY 10024. (212) 595-3449.

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Bupropion

Definition

Bupropion is an antidepressant drug used to elevate mood and promote recovery of a normal range of emotions in patients with depressive disorders. In addition, bupropion is used to as an aid in smoking cessation treatment. In the United States, bupropion is sold as an antidepressant under the brand name Wellbutrin. As a smoking cessation treatment, the drug is marketed under the brand name Zyban.

Purpose

Bupropion is principally known as an antidepressant drug used to promote recovery of depressed patients. It also has therapeutic uses in smoking cessation treatment, **panic disorder**, and **attention-deficit/hyperactivity disorder** (ADHD).

Description

Bupropion is a non-tricyclic antidepressant drug. Tricyclic antidepressants, which have a three-ring chemical structure, may cause troublesome side effects, including sedation, dizziness, faintness, and weight gain. Until the 1980s, such drugs were the mainstay of the pharmacological treatment of depression. Bupropion was one of the first antidepressants with a significantly different chemical structure to be developed by pharmaceutical researchers seeking drugs effective in treating depression but without the unwanted actions of the tricyclic antidepressants.

The exact way that bupropion works in the **brain** is not understood. Its mechanism of action appears to be different from that of most other antidepressant drugs, although bupropion does act on some of the same **neurotransmitters** and neurotransmission pathways. Neurotransmitters are naturally occurring chemicals that regulate the transmission of nerve impulses from one cell to another. Mental well-being is partially dependent on maintaining the proper balance among the various neurotransmitters in the brain. Bupropion may restore normal emotional feelings by counteracting abnormalities of neurotransmission that occur in depressive disorders.

In contrast to the drowsiness frequently caused by other antidepressants, bupropion is a mild stimulant. Bupropion is also less likely to cause weight gain and adverse effects on blood pressure and the heart. However, it is more likely to trigger epileptic **seizures**.

KEY TERMS

Antipsychotic drug—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Epilepsy—A neurological disorder characterized by the onset of seizures. Seizures are caused by a disturbance in the electrical activity in the brain and can cause loss of consciousness, muscle spasms, rhythmic movements, abnormal sensory experiences, or altered mental states.

Neurotransmission—The conduction of a nerve impulse along a chain of nerve cells, which occurs when a cell in the chain secretes a chemical substance, called a neurotransmitter, onto a subsequent cell.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness.

Recommended dosage

The usual adult dose of bupropion (Wellbutrin) is 100 mg, taken three times per day, with at least six hours between doses. The extended release form of the drug (Wellbutrin SR) is taken as 150 mg twice a day with at least eight hours between doses. For smoking cessation, bupropion (Zyban) is taken as 150-mg extended release tablets twice a day, with at least eight hours between doses. Bupropion treatment should be started at a lower dose, then gradually increased to a therapeutic dosage, as directed by the physician. Generally, the total dosage should not exceed 300 mg per day, except as directed by the physician.

The therapeutic effects of bupropion, like other antidepressants, appear slowly. Maximum benefit is often not evident for several weeks after starting the drug. People taking bupropion should be aware of this and continue taking the drug as directed even if they do not see immediate improvement in mood.

Since higher doses of bupropion increase the risk of seizures, no more than 150 mg should be given at any one time, and the total daily dosage should not be increased by more than 100 mg every three days.

Increasing the dosage gradually also minimizes agitation, restlessness, and **insomnia** that may occur.

Healthy elderly patients do not appear to be more sensitive to side effects of bupropion than younger adults and do not require reduced doses. Certain medical conditions, especially liver and kidney disease, may necessitate dose reduction. Although bupropion has been taken by children and adolescents under age 18, it has not been systematically studied in these age groups.

Precautions

Bupropion is more likely to trigger epileptic seizures than other antidepressants. The drug should not be given to patients who have a history of epilepsy, take other medication to help control seizures, or have some other condition associated with seizures, such as head trauma or alcoholism. Nevertheless, in fewer than 1% of healthy people taking bupropion at the recommended dose have seizures. The possibility of seizures is increased at higher doses and following a sudden increase in dose. Patients should minimize alcohol intake while taking bupropion, since alcohol consumption increases the chance of seizures.

Because of the possibility of overdose, potentially suicidal patients should be given only small quantities of the drug at one time. Increases in blood pressure have occurred in patients taking bupropion along with nicotine treatment for smoking cessation. Monitoring blood pressure is recommended in such cases. Excessive stimulation, agitation, insomnia, and anxiety have been troublesome side effects for some patients, especially when treatment is first begun or when the dose is increased. Such adverse effects may be less intense and less frequent when the dose is increased gradually.

It has not been determined whether bupropion is safe to take during pregnancy. Pregnant women should take bupropion only if necessary. The drug is secreted in breast milk. Women taking bupropion should consult their physicians about breast-feeding.

Side effects

Bupropion is a mild stimulant and may cause insomnia, agitation, confusion, restlessness, and anxiety. These effects may be more pronounced at the beginning of therapy and after dose increases. Headache, dizziness, and tremor may occur. Despite stimulating effects, bupropion may also cause sedation.

Weight loss is more common with bupropion than weight gain, but both have been reported. Excessive sweating, dry mouth, sore throat, nausea, vomiting, decreased appetite, constipation, blurred vision, and rapid heart rate may occur.

Interactions

Bupropion should not be administered along with other medications that lower the seizure threshold, such as steroids and the asthma medication theophylline. Many psychiatric medications also lower the seizure threshold. Monoamine oxidase inhibitors (MAOs), another type of antidepressant medication, should not be taken with bupropion. Adverse effects may increase in patients taking levodopa and other medications for Parkison's disease along with bupropion. Patients should inform their doctors about all other medications they are taking before starting this drug.

Nicotine patch therapy may be administered concurrently with bupropion in smoking cessation treatment. If this is done, blood pressure must be monitored, since increased blood pressure has been reported with this combination of medications.

Certain drugs, especially those eliminated by the liver, may interfere with the elimination of bupropion from the body, causing higher blood levels and increased side effects. Conversely, bupropion may retard the elimination of other medicines, including many antidepressants, antipsychotic drugs, and heart medications, resulting in higher blood levels and potentially increased side effects.

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Richard Kapit, M.D.

BuSpar *see* **Buspirone**

Buspirone

Definition

Buspirone is an anti-anxiety (anxiolytic) drug sold in the United States under the brand name of BuSpar. It is also available under its generic name.

Purpose

Buspirone is used for the treatment of generalized anxiety disorders and for short term relief of symptoms of anxiety.

Description

Buspirone's mechanism of action is unclear but probably involves actions on such central nervous system chemicals as dopamine, serotonin, acetylcholine, and norepinephrine. These chemicals are called **neurotransmitters** and are involved in the transmission of nervous impulses from cell to cell. Mental well-being is partially dependent on maintaining a balance among different neurotransmitters.

Buspirone's actions are different from a common class of sedatives called benzodiazepines. The primary action of benzodiazepines is to reduce anxiety, relax skeletal muscles, and induce sleep. The earliest drugs in this class were **chlordiazepoxide** (Librium) and **diazepam** (Valium). Buspirone also acts through a different mechanism than **barbiturates** such as phenobarbital. Unlike benzodiazepines, buspirone has no anticonvulsant or muscle-relaxant properties, and unlike benzodiazepines or barbiturates, it does not have strong sedative properties. If **insomnia** is a component of the patient's anxiety disorder, a sedative/hypnotic drug may be taken along with buspirone at bedtime. Buspirone also diminishes anger and hostility for most people. Unlike benzodiazepines, which may aggravate anger and hostility in some patients, (especially older patients), buspirone may help patients with anxiety who also have a history of aggression.

The benefits of buspirone take a long time to become evident. Unlike benzodiazepines, where onset of action and time to maximum benefit are short, patients must take buspirone for three to four weeks before feeling the maximum benefit of the drug. In some cases, four to six weeks of treatment may be required. Patients should be aware of this and continue to take the drug as prescribed even if they think they are not seeing any improvement.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anxiolytic—A preparation or substance given to relieve anxiety; a tranquilizer.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Buspirone is available in 5-, 10-, 15-, and 30-mg tablets.

Recommended dosage

The usual starting dose of buspirone is 10 to 15 mg per day. This total amount is divided into two or three doses during the day. For example, a dose of 5 mg may be given two or three times per day to make a total dose of 10 to 15 mg per day. The dose may be increased in increments of 5 mg daily every two to four days. Most patients will respond to a dose of 15 to 30 mg daily. Patients should not take a total dose of more than 60 mg daily. When patients are receiving certain other drugs (see below) in addition to buspirone, starting doses of buspirone may need to be lowered (for example, 2.5 mg twice daily), and any dosage increases should be done with caution and under close physician supervision. Dosages may need to be reduced in patients with kidney or liver problems.

Precautions

Buspirone is less sedating (causes less drowsiness and mental sluggishness) than other anti-anxiety drugs. However, some patients may still experience drowsiness and mental impairment. Because it is impossible to predict which patients may experience sedation with buspirone, those starting this drug should not drive or operate dangerous machinery until they know how the drug will affect them.

Patients who have been taking benzodiazepines for a long time should be gradually withdrawn from them while they are being switched over to buspirone. They should also be observed for symptoms of benzodiazepine withdrawal.

Patients with kidney damage should take buspirone with caution in close consultation with their physician. They may require a lower dosage of buspirone to prevent buildup of the drug in the body. Patients with severe kidney disease should not take buspirone. Patients with liver damage should likewise be monitored for a buildup of buspirone and have their doses lowered if necessary.

Side effects

The most common side effects associated with buspirone involve the nervous system. Ten percent of patients may experience dizziness, drowsiness, and headache, and another 5% may experience **fatigue**, nervousness, insomnia, and light-headedness. Patients may also experience excitement, depression, anger, hostility, confusion, nightmares, or other **sleep disorders**, lack of coordination, tremor, and numbness of the extremities. Although buspirone is considered non-sedating, some patients will experience drowsiness and lack of mental alertness at higher doses and especially early in therapy. In most patients, these side effects decrease with time.

The following side effects have also been associated with buspirone:

- nausea (up to 8% of patients)
- dry mouth, abdominal distress, gastric distress, and diarrhea, constipation (up to 5% of patients)
- rapid heart rate and palpitations (up to 2% of patients)
- blurred vision (up to 2% of patients)
- increased or decreased appetite
- flatulence
- non-specific chest pain
- rash
- irregular menstrual periods and/or breakthrough bleeding

Interactions

Dangerously high blood pressure has resulted from the combination of buspirone, and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Because of this, buspirone should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 10 days before starting buspirone. The same holds true when discontinuing buspirone and starting an MAO inhibitor.

Certain drugs may inhibit the enzyme system in the liver that breaks down buspirone. Examples of drugs that might inhibit this system are erythromycin, a broad-spectrum antibiotic, itraconazole, an oral antifungal agent, and **nefazodone**, an antidepressant. When these drugs are combined with buspirone, buspirone concentrations may increase to the point of toxicity (poisoning). These combinations should either be avoided or doses of buspirone decreased to compensate for this interaction.

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Jack Raber, Pharm.D.

C

Caffeine-related disorders

Definition

Caffeine is a white, bitter crystalline alkaloid derived from coffee or tea. It belongs to a class of compounds called xanthines, its chemical formula being 1,3,7-trimethylxanthine. Caffeine is classified together with cocaine and **amphetamines** as an analeptic, or central nervous system stimulant. Coffee is the most abundant source of caffeine, although caffeine is also found in tea, cocoa, and cola beverages as well as in over-the-counter and prescription medications for pain relief.

In the clinician's handbook for diagnosing mental disorders (the ***Diagnostic and Statistical Manual of Mental Disorders***, known as the *DSM-IV-TR*), caffeine-related disorders are classified under the rubric of substance-related disorders. *DSM-IV-TR* specifies four caffeine-related disorders: caffeine intoxication, caffeine-induced anxiety disorder, caffeine-induced sleep disorder, and caffeine-related disorder not otherwise specified. A fifth, caffeine withdrawal, is listed under the heading of "Criteria Sets and Axes Provided for Further Study."

Caffeine-related disorders are often unrecognized for a number of reasons:

- Caffeine has a "low profile" as a drug of abuse. Consumption of drinks containing caffeine is unregulated by law and is nearly universal in the United States; one well-known textbook of pharmacology refers to caffeine as "the most widely used psychoactive drug in the world." In many countries, coffee is a social lubricant as well as a stimulant; the "coffee break" is a common office ritual, and many people find it difficult to imagine eating a meal in a fine restaurant without having coffee at some point during the meal. It is estimated that 10–12 billion pounds of coffee are consumed worldwide each year.
- People often underestimate the amount of caffeine they consume on a daily basis because they think of caffeine only in connection with coffee as a beverage. Tea, cocoa, and some types of soft drink, including root beer and orange soda as well as cola beverages, also contain significant amounts of caffeine. In one British case study, a teenager who was hospitalized with muscle weakness, nausea, vomiting, diarrhea, and weight loss was found to suffer from caffeine intoxication caused by drinking 8 liters (about 2 gallons) of cola on a daily basis for the previous two years. She had been consuming over a gram of caffeine per day. Chocolate bars and coffee-flavored yogurt or ice cream are additional sources of measurable amounts of caffeine.
- Caffeine has some legitimate medical uses in athletic training and in the relief of tension-type headaches. It is available in over-the-counter (OTC) preparations containing aspirin or acetaminophen for pain relief as well as in such OTC stimulants as NoDoz and Vivarin.
- Caffeine is less likely to produce the same degree of physical or psychological dependence as other drugs of abuse. Few coffee or tea drinkers report loss of control over caffeine intake, or significant difficulty in reducing or stopping consumption of beverages and food items containing caffeine.
- The symptoms of caffeine intoxication are easy to confuse with those of an anxiety disorder.

The *DSM-TR-IV* states that it is unclear as of 2000 whether the tolerance, withdrawal symptoms, and "some aspects of dependence on caffeine" seen in some people who drink large amounts of coffee "are associated with clinically significant impairment that meets the criteria for Substance Abuse or Substance Dependence." On the other hand, a research team at Johns Hopkins regards caffeine as a model drug for understanding substance abuse and dependence. The team maintains that 9%–30% of caffeine consumers in the United States may be caffeine-dependent according to *DSM* criteria for substance dependency.

KEY TERMS

Adenosine—A compound that serves to modulate the activities of nerve cells (neurons) and to produce a mild sedative effect when it activates certain types of adenosine receptors. Caffeine is thought to produce its stimulating effect by competing with adenosine for activation of these receptors.

Analeptic—A substance that acts as a stimulant of the central nervous system. Caffeine is classified as an analeptic.

Caffeinism—A disorder caused by ingesting very high doses of caffeine (10g or more per day) and characterized by seizures and respiratory failure.

Dependence—The adaptation of neurons and other physical processes to the use of a drug, followed by withdrawal symptoms when the drug is removed; physiological and/ or psychological addiction.

Hematemesis—Vomiting blood. Hematemesis is a symptom that sometimes occurs with gastrointestinal ulcers made worse by high levels of caffeine consumption.

Norepinephrine—A catecholamine neurotransmitter that acts to constrict blood vessels, raise blood pressure, and dilate the bronchi of the respiratory system. Caffeine increases the secretion of norepinephrine.

Reinforcement—A term that refers to the ability of a drug or substance to produce effects that will make the user want to take it again.

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

Xanthine—A class of crystalline nitrogenous compounds that includes caffeine, which is 1,3,7-trimethylxanthine.

Description

Pharmacological aspects of caffeine

An outline of the effects of caffeine on the central nervous system (CNS) and other organ systems of the body may be helpful in understanding its potential for physical dependence. When a person drinks a beverage containing caffeine (or eats coffee-flavored ice cream), the caffeine is absorbed from the digestive tract without being broken down. It is rapidly distributed throughout the tissues of the body by means of the bloodstream. If a pregnant woman drinks a cup of coffee or tea, the caffeine in the drink will cross the placental barrier and enter the baby's bloodstream.

When the caffeine reaches the **brain**, it increases the secretion of norepinephrine, a neurotransmitter that is associated with the so-called fight or flight **stress** response. The rise in norepinephrine levels and the increased activity of the neurons, or nerve cells, in many other areas of the brain helps to explain why the symptoms of caffeine intoxication resemble the symptoms of a **panic attack**.

The effects of caffeine are thought to occur as a result of competitive antagonism at adenosine receptors. Adenosine is a water-soluble compound of adenine and ribose; it functions to modulate the activities of nerve cells and produces a mild sedative effect when it activates certain types of adenosine receptors. Caffeine competes with adenosine to bind at these receptors and counteracts the sedative effects of the adenosine. If the person stops drinking coffee, the adenosine has no competition for activating its usual receptors and may produce a sedative effect that is experienced as **fatigue** or drowsiness.

Caffeine content of food items and OTC preparations

The caffeine content of various food items and medications is as follows:

- Brewed coffee, 8-oz cup: 135–150 mg
- Instant coffee, 8-oz cup: 95 mg
- Powdered cappuccino beverage, 8-oz cup: 45–60 mg
- Tea brewed from leaves or bag, 8-oz cup: 50 mg
- Iced tea from mix, 8-oz glass: 25–45 mg
- Snapple iced tea, 8-oz glass: 21 mg
- Mountain Dew, 8-oz glass: 38 mg
- Dr. Pepper, 8-oz. glass: 28 mg
- Diet cola, 8-oz glass: 31 mg
- Root beer, 8-oz glass: 16 mg
- Coffee ice cream, 8-oz serving: 60–85 mg
- Coffee yogurt, 8-oz serving: 45 mg.
- Dark chocolate candy bar, 1.5 oz: 31 mg
- NoDoz, regular strength, 1 tablet: 100 mg

- NoDoz, maximum strength, 1 tablet: 200 mg
- Excedrin, 2 tablets: 130 mg

Caffeine can produce a range of physical symptoms following ingestion of as little as 100 mg, although amounts of 250 mg or higher are usually needed to produce symptoms that meet the criteria of caffeine intoxication.

Caffeine intoxication

To meet *DSM-IV-TR* criteria for caffeine intoxication, a person must develop five or more of the twelve symptoms listed below; the symptoms must cause significant distress or impair the person's social or occupational functioning; and the symptoms must not be caused by a medical disorder or better accounted for by an anxiety disorder or other mental disorder.

Because people develop tolerance to caffeine fairly quickly with habitual use, caffeine intoxication is most likely to occur in those who consume caffeine infrequently or who have recently increased their intake significantly.

Caffeine-induced anxiety and sleep disorders

DSM-IV-TR criteria for caffeine-induced anxiety and **sleep disorders** specify that the symptoms of anxiety and **insomnia** respectively must be more severe than the symptoms associated with caffeine intoxication. In addition, the anxiety or insomnia must be severe enough to require separate clinical attention.

Causes and symptoms

Causes

The immediate cause of caffeine intoxication and other caffeine-related disorders is consumption of an amount of caffeine sufficient to produce the symptoms specified by *DSM-IV-TR* as criteria for the disorder. The precise amount of caffeine necessary to produce symptoms varies from person to person depending on body size and degree of tolerance to caffeine. Tolerance of the stimulating effects of caffeine builds up rapidly in humans; mild withdrawal symptoms have been reported in persons who were drinking as little as one to two cups of coffee per day.

Some people may find it easier than others to consume large doses of caffeine because they are insensitive to its taste. Caffeine tastes bitter to most adults, which may serve to limit their consumption of coffee and other caffeinated beverages. Slightly more than 30% of the American population, however, has an inherited inability to taste caffeine.

Symptoms

The symptoms of caffeine intoxication include:

- restlessness
- nervousness
- excitement
- insomnia
- flushed face
- diuresis (increased urinary output)
- gastrointestinal disturbance
- muscle twitching
- talking or thinking in a rambling manner
- tachycardia (speeded-up heartbeat) or disturbances of heart rhythm
- periods of inexhaustibility
- psychomotor agitation

People have reported ringing in the ears or seeing flashes of light at doses of caffeine above 250 mg. Profuse sweating and diarrhea have also been reported. Doses of caffeine higher than 10 g may produce respiratory failure, **seizures**, and eventually death.

Side effects and complications

High short-term consumption of caffeine can produce or worsen gastrointestinal problems, occasionally leading to peptic ulcers or hematemesis (vomiting blood).

In addition to the symptoms produced by high short-term doses, long-term consumption of caffeine has been associated with fertility problems and with bone loss in women leading to osteoporosis in old age. Some studies have found that pregnant women who consume more than 150 mg per day of caffeine have an increased risk of miscarriage and low birth weight babies, but the findings are complicated by the fact that most women who drink large amounts of coffee during pregnancy are also heavy smokers. Some researchers believe that long-term consumption of caffeine is implicated in cardiovascular diseases, but acknowledge that further research is required.

On the other hand, moderate doses of caffeine improve athletic performance as well as alertness. Caffeine in small doses can relieve tension headaches, and one study found that a combination of ibuprofen and caffeine was more effective in relieving tension headaches than either ibuprofen alone or a placebo. Coffee consumption also appears to lower the risk of alcoholic and nonalcoholic cirrhosis of the liver.

Coffee is the most abundant source of caffeine, although caffeine is also found in tea, cocoa, and cola beverages as well as in over-the-counter and prescription medications for pain relief. *(Patrik Giardino/ CORBIS. Photo reproduced by permission.)*

Drug interactions

Caffeine is often combined with aspirin or acetaminophen in over-the-counter and prescription analgesics (pain relievers). It can also be combined with ibuprofen. On the other hand, certain groups of drugs should not be combined with caffeine or taken with beverages containing caffeine. Oral contraceptives, cimetidine (Tagamet), mexiletine (Mexitil), and **disulfiram** (Antabuse) interfere with the breakdown of caffeine in the body. Caffeine interferes with the body's absorption of iron, and with drugs that regulate heart rhythm, including quinidine and **propranolol** (Inderal). Caffeine may produce serious side effects when taken together with monoamine oxidase inhibitors or with certain decongestant medications.

Combinations of ephedra and caffeine have been used in weight-loss programs because they produce greater weight loss than can be achieved by caloric restriction alone. Major studies were underway as of 2001 at Harvard and Vanderbilt to determine the safety of these regimens.

Practitioners of homeopathy have traditionally advised patients not to drink beverages containing caffeine in the belief that caffeine "antidotes" homeopathic remedies. Contemporary homeopaths disagree on the antidoting effects of caffeine, observing that homeopathy is used widely and effectively in Europe and that Europeans tend to drink strong espresso coffee more frequently than Americans.

Demographics

The general population of the United States has a high level of caffeine consumption, with an average intake of 200 mg per day. About 85% of the population uses caffeine in any given year. Among adults in the United States, about 30% consume 500 mg or more each day. These figures are lower, however, than the figures for Sweden, the United Kingdom, and other parts of Europe, where the average daily consumption of caffeine is 400 mg or higher. In developing countries, the average consumption of caffeine is much lower— about 50 mg per day.

In the United States, levels of caffeine consumption among all races and ethnic groups are related to age, with usage beginning in the late teens and rising until the early 30s. Caffeine consumption tapers off in adults over 40 and decreases in adults over 65. Caffeine intake is higher among males than among females in North America.

The prevalence of caffeine-related disorders in the United States is not known as of 2002.

Diagnosis

Diagnosis of a caffeine-related disorder is usually based on the patient's recent history, a physical examination, or laboratory analysis of body fluids. In addition to medical evidence, the examiner will rule out other mental disorders, particularly manic episodes, **generalized anxiety disorder**, **panic disorder**, amphetamine intoxication, or withdrawal from sedatives, tranquilizers, sleep medications, or nicotine. All of these disorders or syndromes may produce symptoms resembling those of caffeine intoxication. In most cases, the temporal relationship of the symptoms to high levels of caffeine intake establishes the diagnosis.

In some cases, the examiner may consider the possibility of depression during the differential diagnosis, as many people with depression and eating disorders self-medicate with caffeine.

Treatments

Treatment of caffeine-related disorders involves lowering consumption levels or abstaining from beverages containing caffeine. Some people experience mild withdrawal symptoms that include headaches, irritability, and occasionally nausea, but these usually resolve quickly.

Caffeine consumption has the advantage of having relatively weak (compared to alcohol or cigarettes) social **reinforcement**, in the sense that one can easily choose a noncaffeinated or decaffeinated beverage in a restaurant or at a party without attracting comment. Thus physical dependence on caffeine is less complicated by the social factors that reinforce nicotine and other drug habits.

Prognosis

With the exception of acute episodes of caffeinism, people recover from caffeine intoxication without great difficulty.

Prevention

Prevention of caffeine-related disorders requires awareness of the caffeine content of caffeinated beverages, OTC drugs, and other sources of caffeine; monitoring one's daily intake; and substituting decaffeinated coffee, tea, or soft drinks for the caffeinated versions of these beverages.

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American Dietetic Association. (800) 877-1600. <www.eatright.org>.

Center for Science in the Public Interest (CSPI). <www.cspinet.org>.

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Cannabis and related disorders

Definition

Cannabis, more commonly called marijuana, refers to the several varieties of *Cannabis sativa*, or Indian hemp plant, that contains the psychoactive drug delta-9-tetrahydrocannabinol (THC). Cannabis-related disorders refer to problems associated with the use of substances derived from this plant.

KEY TERMS

Amotivational syndrome—Loss of ambition associated with chronic cannabis (marijuana) use.

Anandamide—One type of endocannabinoid that appears to help regulate early pregnancy.

Cannabis—The collective name for several varieties of Indian hemp plant. Also known as marijuana.

Cannabis abuse—Periodic use of cannabis, less serious than dependence, but still capable of causing problems for the user.

Cannabis dependence—The compulsive need to use cannabis, leading to problems.

Cannabis intoxication—The direct effects of acute cannabis use and the reactions that accompany those effects.

Delta-9-tetrahydrocannabinol(THC)—The primary active ingredient in marijuana.

Endocannabinoids—Cannabis-like compounds produced naturally in the human body.

Hashish—The dark, blackish resinous material that exudes from the leaves of the Indian hemp plant.

Marijuana—The dried and shredded or chopped leaves of the Indian hemp plant.

Description

Cannabis—in the form of marijuana, hashish (a dried resinous material that seeps from cannabis leaves and is more potent than marijuana), or other cannabinoids—is considered the most commonly used illegal substance in the world. Its effects have been known for thousands of years, and were described as early as the fifth century B.C., when the Greek historian Herodotus told of a tribe of nomads who, after inhaling the smoke of roasted hemp seeds, emerged from their tent excited and shouting for joy.

Cannabis is the abbreviation for the Latin name for the hemp plant—*Cannabis sativa*. All parts of the plant contain psychoactive substances, with THC making up the highest percentage. The most potent parts are the flowering tops and the dried, blackish-brown residue that comes from the leaves known as hashish, or "hash."

There are more than 200 slang terms for marijuana, including "pot," "herb," "weed," "Mary Jane," "grass," "tea," and "ganja." It is usually chopped and/or shredded and rolled into a cigarette, or "joint," or placed in a pipe (sometimes called a "bong") and smoked. An alternative method of using marijuana involves adding it to foods and eating it, such as baking it into brownies. It can also be brewed as a tea. Marijuana has appeared in the form of "blunts"—cigarettes emptied of their tobacco content and filled with a combination of marijuana and another drug such as crack cocaine.

Between 1840 and 1900, European and American medical journals published numerous articles on the therapeutic uses of marijuana. It was recommended as an appetite stimulant, muscle relaxant, painkiller, sedative, and anticonvulsant. As late as 1913, Sir William Osler recommended it highly for treatment of migraine. Public opinion changed, however, in the early 1900s, as alternative medications such as aspirin, opiates, and **barbiturates** became available. In 1937, the United States passed the Marijuana Tax Act, which made the drug essentially impossible to obtain for medical purposes.

By the year 2000, the debate over the use of marijuana as a medicine continued. THC is known to successfully treat nausea caused by cancer treatment drugs, stimulate the appetites of persons diagnosed with acquired immune deficiency syndrome (AIDS), and possibly assist in the treatment of glaucoma. Its use as a medicinal agent is still, however, highly controversial. Even although the states of Arizona and California passed laws in 1996 making it legal for physicians to prescribe marijuana in the form of cigarettes for treatment of the diseases listed above, governmental agencies continue to oppose strongly its use as a medicine, and doctors who do prescribe it may find their licenses at risk.

Cannabis-related disorders reflect the problematic use of cannabis products to varying degrees. These disorders include:

- Cannabis dependence: The compulsive need to use the drug, coupled with problems associated with chronic drug use.
- Cannabis abuse: Periodic use that may cause legal problems, problems at work, home, or school, or danger when driving.
- Cannabis intoxication: The direct effects of acute cannabis use and reactions that accompany it such as feeling "high," euphoria, sleepiness, lethargy, impairment in short-term memory, stimulated appetite, impaired judgment, distorted sensory perceptions, impaired motor performance, and other symptoms.

Causes and symptoms

Causes

Cannabis-related disorders share many of the same root causes with other addictive substances. The initial desire for a "high," combined with the widely held perception that cannabis use is not dangerous, often leads to experimentation in the teen years.

Recent research challenges the notion that cannabis use is not physically addictive. According to the National Institute of Drug Abuse (NIDA), daily cannabis users experience withdrawal symptoms including irritability, stomach pain, aggression, and anxiety. Many frequent cannabis users are believed to continue using in order to avoid these unpleasant symptoms. Long-term use may lead to changes in the **brain** similar to those seen with long-term use of other addictive substances. It is believed that the greater availability, higher potency, and lower price for cannabis in recent years all contribute to the increase in cannabis-related disorders.

Beginning in the 1990s, researchers began to discover that cannabis-like compounds are naturally produced in various parts of the human body. These compounds, called "endocannabinoids," appear to suppress inflammation and other responses of the immune system. One of these endocannabinoids— anandamide—appears to help regulate the early stages of pregnancy.

Symptoms

CANNABIS DEPENDENCE AND ABUSE. The handbook used by mental health professionals to diagnose mental disorders is the ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM-IV-TR*. This manual states that the central features of cannabis dependence are compulsive use, tolerance of its effects, and withdrawal symptoms. Use may interfere with family, school, and work, and may cause legal problems.

Regular cannabis smokers may show many of the same respiratory symptoms as tobacco smokers. These include daily cough and phlegm, chronic bronchitis, and more frequent chest colds. Continued use can lead to abnormal functioning of the lung tissue, which may be injured or destroyed by the cannabis smoke.

Recent research indicates that smoking marijuana has the potential to cause severe increases in heart rate and blood pressure, particularly if combined with cocaine use. Even with marijuana use alone, however, the heart rate of subjects increased an average of 29 beats per minute when smoking marijuana.

A study of heavy marijuana users has shown that critical skills related to attention, memory, and learning can be impaired, even after use is discontinued for at least 24 hours. Heavy users, compared to light users, made more errors on tasks and had more difficulty sustaining attention and shifting attention when required. They also had more difficulty in registering, processing, and using information. These findings suggest that the greater impairment in mental functioning among heavy users is most likely due to an alteration of brain activity directly produced by the marijuana use.

Recent studies have found that babies born to mothers who used marijuana during pregnancy were smaller than those born to nonusing mothers. Smaller babies are more likely to develop health problems. Additionally, nursing mothers who use marijuana pass some of the THC to the baby in their breast milk. Research shows that use of marijuana during the first month of breastfeeding can impair an infant's motor development.

Cannabis abuse is characterized by less frequent use and less severe problems. However, as with cannabis dependence, abuse can interfere with performance at school or work, cause legal problems, and interfere with motor activities such as driving or operating machinery.

CANNABIS INTOXICATION. Cannabis intoxication refers to the occurrence of problematic behaviors or psychological changes that develop during, or shortly after, cannabis use. Intoxication usually starts with a "high" feeling followed by euphoria, inappropriate laughter, and feelings of grandiosity. Other symptoms include sedation, lethargy, impaired short-term memory, difficulty with motor tasks, impaired judgment, distorted sensory perceptions, and the feeling that time is passing unusually slowly. Sometimes severe anxiety, feelings of depression, or social withdrawal may occur. Along with these symptoms, common signs of cannabis intoxication include reddening of the membranes around the eyes, increased appetite, dry mouth, and increased heart rate.

Demographics

The NIDA conducts an annual nationwide study of twelfth-, tenth-, and eighth-grade students and young adults. This study is known as the *Monitoring the Future Study*, or *MTF*. Results show that after a decade of decreased use in the 1980s, marijuana use among students began to rise in the early 1990s. Data show that, between 1998 and 1999, marijuana use continued to increase among twelfth and tenth graders. For twelfth graders, the lifetime rate (use of marijuana at least one or more times) is higher than for any year since 1987. However, these rates remain well below those seen in the late 1970s and early 1980s. Daily marijuana use among students in all three grades also showed a slight increase.

Another method by which the government measures marijuana use is the *Community Epidemiology Work Group*, or *CEWG*. This method examines rates of emergency room admissions related to marijuana use in 20 major metropolitan areas. In 1998, use of marijuana showed an upward trend in most of the areas monitored, with the largest increases occurring in Dallas, Boston, Denver, San Diego, and Atlanta. The highest percentage increase in emergency room visits related to marijuana was among 12- to 17-year-olds.

Treatment data for marijuana abuse increased in six of the metropolitan areas surveyed but remained stable elsewhere. Marijuana treatment admissions were highest in Denver, Miami, New Orleans, and Minneapolis/St. Paul. Half of the admissions in Minneapolis/St. Paul were under the age of 18 years.

Marijuana remains the most commonly used illicit drug in the United States. As with most other illicit drugs, cannabis use disorders appear more often in males and is most common among people between the ages of 18 and 30 years.

An estimated 2.1 million people started using marijuana in 1998. According to data from a study released in the late 1990s called the *National Household Survey on Drug Abuse*, or *NHSDA*, more than 72 million Americans ages 12 years and older (33%) tried marijuana at least once during their lifetime, while almost 18.7 million (8.6%) used marijuana in the previous year. The reader can compare these figures to the figures from 1985, when 56.5 million Americans (29.4%) had tried marijuana at least once in their life, and 26.1 million (13.6%) had used marijuana within the past year.

Diagnosis

Diagnosis of cannabis-related disorders is made in a number of ways. Intoxication is easiest to diagnose because of clinically observable signs, including reddened eye membranes, increased appetite, dry mouth, and increased heart rate. It is also diagnosed by the presence of problematic behavioral or psychological changes such as impaired motor coordination, judgment, anxiety, euphoria, and social withdrawal. Occasionally, panic attacks may occur, and there may be impairment of short-term memory. Lowered immune system resistance, lowered testosterone levels in males, and chromosomal damage may also occur. Psychologically, chronic use of marijuana has been associated with a loss of ambition known as the "amotivational syndrome."

Cannabis use is often paired with the use of other addictive substances, especially nicotine, alcohol, and cocaine. Marijuana may be mixed and smoked with opiates, phencyclidine ("PCP" or "angel dust"), or hallucinogenic drugs. Individuals who regularly use cannabis often report physical and mental lethargy and an inability to experience pleasure when not intoxicated (known as "anhedonia"). If taken in sufficiently high dosages, cannabinoids have psychoactive effects similar to hallucinogens such as lysergic acid diethylamide (LSD), and individuals using high doses may experience adverse effects that resemble hallucinogen-induced "bad trips." Paranoid ideation is another possible effect of heavy use, and, occasionally, **hallucinations** and **delusions** occur. Highly intoxicated individuals may feel as if they are outside their body ("depersonalization") or as if what they are experiencing isn't real ("derealization"). Fatal traffic accidents are more common among individuals testing positive for cannabis use.

Urine tests can usually identify metabolites of cannabinoids. Because cannabinoids are fat soluble, they remain in the body for extended periods. Individuals who have used cannabis may show positive urine tests for as long as two to four weeks after using.

Examination of the nasopharynx and bronchial lining may also show clinical changes due to cannabis use. Marijuana smoke is known to contain even larger amounts of carcinogens than tobacco smoke. Sometimes cannabis use is associated with weight gain.

Treatments

Treatment options for individuals with cannabis-related disorders are identical to those available for people with alcohol and other substance abuse disorders. The goal of treatment is abstinence. Treatment approaches range from in-patient **hospitalization**, drug and alcohol rehabilitation facilities, and various outpatient programs. Twelve-step programs such as Narcotics Anonymous are also treatment options. For heavy users suffering from withdrawal symptoms, treatment with anti-anxiety and/or antidepressant medication may assist in the treatment process.

Prognosis

According to the *DSM-IV-TR*, cannabis dependence and abuse tend to develop over a period of time. It may, however, develop more rapidly among young people with other emotional problems. Most people who become dependent begin using regularly. Gradually, over time, both frequency and amount increase. With chronic use, there can sometimes be a decrease in or loss of the pleasurable effects of the substance, along with increased feelings of anxiety and/or depression. As with alcohol and nicotine, cannabis use tends to begin early in the course of

substance abuse and many people later go on to develop dependence on other illicit substances. Because of this, cannabis has been referred to as a "gateway" drug, although this view remains highly controversial. There is much that remains unknown about the social, psychological, and neurochemical basis of drug use progression, and it is unclear whether marijuana use actually causes individuals to go on to use other illicit substances.

One long-term effect of chronic use has been termed the "amotivational syndrome." This refers to the observation that many heavy, chronic users seem unambitious in relation to school and/or career.

Prevention

Many drug education programs focus strongly on discouraging marijuana experimentation among young teenagers. Recent research reported by the NIDA indicates that high-sensation-seekers—that is, individuals who seek out new, emotionally intense experiences and are willing to take risks to obtain these experiences—are at greater risk for using marijuana and other drugs, and for using them at an earlier age. As a result, the NIDA developed a series of public service announcements (PSAs) for national television. These PSAs were dramatic and attention getting, and were aired during programs that would appeal to high sensation-seekers, such as action-oriented television shows. These PSAs were aired in a limited television area and the results monitored. Marijuana use declined substantially among teens during the PSA campaigns, and long-term effects were shown for several months afterwards. In one county, marijuana use decreased by 38%, and in another, by 26.7%.

Drug education programs such as the "DARE" (Drug Awareness and Resistance Education) programs target fifth graders. These and other antidrug programs focus on peer pressure resistance and the use of older teens who oppose drug use as models of a drug-free lifestyle. These programs show mixed results.

See also Addiction; Disease concept of chemical dependency; Dual diagnosis; Nicotine and related disorders; Opioids and related disorders; Relapse and relapse prevention; Self-help groups; Substance abuse and related disorders; Support groups

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American Council for Drug Education, 136 E. 64th St., NY, NY 10021.

Narcotics Anonymous. PO box 9999, Van Nuys, CA 91409. (818) 780-3951.

National Institute on Drug Abuse (NIDA). US Department of Health and Human Services, 5600 Fishers Ln., Rockville, MD 20857. <http://www.nida.nih.gov>

National Organization for the Reform of Marijuana Laws (NORML). 2001 S St. NW, Suite 640, Washington, DC 20009. (202) 483-5500.

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Carbamazepine

Definition

Carbamazepine is an anticonvulsant that is structurally related to tricyclic antidepressants such as **amitriptyline** and **imipramine**. In the United States, carbamazepine is sold under the trade names Tegretol and Carbatrol.

Purpose

Carbamazepine is effective in the treatment of psychomotor and grand mal **seizures** and a type of facial pain called trigeminal neuralgia and, in combination with other drugs, for psychiatric disorders such as mania and extreme aggression. Carbamazepine is also occasionally used to control pain in persons with cancer.

Description

Carbamazepine was first marketed as an anti-seizure medication and as a first-line treatment for trigeminal

KEY TERMS

Absence seizure—An epileptic seizure characterized by a sudden, momentary loss of consciousness, occasionally accompanied by some minor, jerky movements in the neck or upper arms, a twitching of the face, or a loss of muscle tone.

Aplastic anemia—A form of anemia in which the bone marrow does not produce adequate amounts of peripheral blood components such as red cells, white cells and platelets.

Bipolar disorder—A mental disorder characterized by dramatic, and sometimes rapid mood swings, resulting in both manic and depressive episodes; formerly called manic-depressive disorder.

Convulsion—A violent, involuntary contraction or series of contractions of muscles.

Grand mal seizure—A seizure characterized by a sudden loss of consciousness that is immediately followed by generalized convulsions. Such a seizure is usually preceded by a sensory experience, called an aura, which provides a warning as to an impending convulsion.

Psychomotor seizure—A seizure characterized by electrical activity that is characterized by variable degrees of loss of consciousness and often accompanied by bizarre behavior.

neuralgia. Because it was later noted to be effective in patients with certain psychiatric disorders, psychiatrists began combining it with other drugs such as lithium and major tranquilizers in severe cases of bipolar disease and aggressive behavior that could not be managed with single-drug therapy.

Carbamazepine is available in 100-mg chewable tablets, 200-mg capsules and a suspension at 100 mg per 5 ml of liquid.

Recommended dosage

When used to treat seizure disorders or psychiatric disease, the recommended initial dosage of carbamazepine is 200 mg two times each day. If needed, the daily dosage may be increased by 200 mg once each week. Total daily dosages should not exceed 1,000 mg in children between the ages of 12 and 15 years. Total daily dosages for adults should not exceed 1,200 mg. Carbamazepine should be taken with meals.

Precautions

Carbamazepine should be used with caution in persons who also experience other types of seizure disorders such as atypical absence seizures. Among such individuals, carbamazepine usage has been associated with an increased risk of initiating, rather than controlling, generalized convulsions.

Carbamazepine should never be discontinued abruptly unless another treatment for seizures is initiated at the same time. If this does not happen, acute withdrawal of carbamazepine may result in seizures.

Patients should be alert for signs and symptoms of bone marrow toxicity such as fever, sore throat, infection, mouth sores, easy bruising, or bleeding which occurs just under the skin.

Because carbamazepine may affect mental alertness, especially early in therapy, patients receiving this drug should not operate dangerous machinery or drive a car until they understand how the drug will affect them.

Side effects

The most commonly reported adverse reactions to carbamazepine include dizziness, drowsiness, unsteadiness, nausea and vomiting. These are more common when therapy is just beginning.

Carbamazepine has been reported to cause aplastic anemia. This is a form of anemia that is generally does not respond to treatment. The bone marrow of persons with aplastic anemia does not produce adequate amounts of red blood cells, white blood cells, and platelets. Blood counts should be monitored for individuals using this drug. Some people with previously diagnosed depression of the bone marrow should not take carbamazepine.

Carbamazepine may cause birth defects and should be avoided by women who are pregnant. An effective contraceptive method should be used while taking carbamazepine. It is important to note that this medication may decrease the effectiveness of oral contraceptives.

The drug can cross into breast milk and should be avoided by women who are breast-feeding.

Carbamazepine may also cause a skin rash or sensitivity to the sun.

Interactions

Blood levels of carbamazepine may be reduced when it is used in combination with other drugs such as phenobarbitol, phenytoin or primidone. This means that inadequate amounts of carbamazepine are available to the body, limiting the ability of the drug to control seizure

activity or treat psychiatric disease. Carbamazepine also causes reductions in the blood levels of the following drugs when they are used simultaneously: phenytoin, warfarin, doxycycline, **haloperidol**, **valproic acid**, and theophylline.

The simultaneous administration of carbamazepine with erythromycin, cimetidine, propoxyohene, isoniacid, **fluoxetine** and calcium channel blockers such as nifedipine or verapamil may increase the blood level of carbamazepine to a toxic range.

The simultaneous use of carbamazepine and oral contraceptives may increase the possibility that the oral contraceptive won't be effective in preventing pregnancy. Some physicians recommend that a different method of contraception be used while carbamazepine is being used.

People taking carbamazepine should not drink grapefruit juice. Grapefruit juice slows the breakdown of carbamazepine, increasing the concentration of carbamazepine in the bloodstream.

Due to the potential of many interactions with other drugs, individuals should consult with a physician or pharmacist prior to starting any new medications either bought over the counter or initiated by another physician.

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CASE *see* **Clinical Assessment Scales for the Elderly**

Case management

Definition

Case management assigns the administration of care for an outpatient individual with a serious mental illness to a single person (or team); this includes coordinating all necessary medical and mental health care, along with associated supportive services.

Purpose

Case management tries to enhance access to care and improve the continuity and efficiency of services. Depending on the specific setting and locale, case managers are responsible for a variety of tasks, ranging from linking clients to services to actually providing intensive clinical or rehabilitative services themselves. Other core functions include outreach to engage clients in services, assessing individual needs, arranging requisite support services (such as housing, benefit programs, job training), monitoring medication and use of services, and advocating for client rights and entitlements.

Case management is not a time-limited service, but is intended to be ongoing, providing clients whatever they need whenever they need it, for as long as necessary.

KEY TERMS

Medicaid—A program jointly funded by state and federal governments that reimburses hospitals and physicians for the care of individuals who cannot pay for their own medical expenses. These individuals may be in low-income households or may have chronic disabilities.

Medicare—A federally funded health insurance program for individuals age 65 and older, and certain categories of younger persons with disabilities.

Meta-analysis—The statistical analysis of a large collection of analyses from individual studies for the purpose of integrating the findings.

Neuroleptic—Another name for the older antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Supplemental Security Income—A federal program that provides cash to meet basic needs for food, shelter and clothing for aged, blind, and disabled individuals who have little or no income.

Historical background

Over the past 50 years, there have been fundamental changes in the system of mental health care in America. In the 1950s, mental health care for persons with severe and persistent mental illnesses (like **schizophrenia**, **bipolar disorder**, severe depression, and **schizoaffective disorder**) was provided almost exclusively by large public mental hospitals. Created as part of a reform movement, these state hospitals provided a wide range of basic life supports in addition to mental health treatment, including housing, meals, clothing and laundry services, and varying degrees of social and **vocational rehabilitation**.

During the latter half of the same decade, the introduction of neuroleptic medication provided symptomatic management of seriously disabling psychoses. This breakthrough, and other subsequent reforms in mental health policy (including the introduction of Medicare and Medicaid in 1965 and the Supplemental Security Income [SSI] program in 1974), provided incentives for policy makers to discharge patients to the community and transfer state mental health expenditures to the federal government.

These advances—coupled with new procedural safeguards for involuntary patients, court decisions establishing the right to treatment in the least restrictive setting, and changed philosophies of care—led to widespread **deinstitutionalization**. In 1955 there were 559,000 persons in state hospitals; by 1980, that number had dropped to 132,000. According to the most recent data from the U.S. Center for Mental Health Services, while the number of mental health organizations providing 24-hour services (hospital inpatient and residential treatment) more than doubled in the United States from 1970 to 1998, the number of psychiatric beds provided by these organizations decreased by half.

As a result of deinstitutionalization policies, the number of patients discharged from hospitals has risen, and the average length of stay for newly admitted patients has decreased. An increasing number of patients are never admitted at all, but are diverted to a more complex and decentralized system of community-based care. Case management was designed to remedy the confusion created by multiple care providers in different settings, and to assure accessibility, continuity of care, and accountability for individuals with long-term disabling mental illnesses.

Models of case management

The two models of case management mentioned most often in the mental health literature are assertive community treatment (ACT) and intensive case management.

A third model, clinical case management, refers to a program where the case manager assigned to a client also functions as their primary therapist.

Assertive community treatment

The ACT model originated in an inpatient research unit at Mendota State Hospital in Madison, Wisconsin in the late 1960s. The program's architects, Arnold Marx, M.D., Leonard Stein, M.D. and Mary Ann Test, Ph.D., sought to create a "hospital without walls." In this model, teams of 10–12 professionals— including case managers, a **psychiatrist**, nurses, **social workers**, and vocational specialists—are assigned ongoing responsibility 24 hours a day, seven days a week, 365 days a year, for a caseload of approximately 10 clients with severe and persistent mental illnesses.

ACT uses multidisciplinary teams, low client-to-staff ratios, an emphasis on assertive outreach, provision of in-vivo services (in the client's own setting), an

emphasis on assisting the client in managing their illness, assistance with ADL (activities of daily living) skills, emphasis on relationship building, and emotional support, **crisis intervention** (as necessary) and an orientation, whenever possible, towards providing clients with services rather than linking them to other providers.

Compared to other psychosocial interventions the program has a remarkably strong evidence base. Twenty-five randomized controlled clinical trials have demonstrated that these programs reduce **hospitalization**, **homelessness**, and inappropriate hospitalization; increase housing stability; control psychiatric symptoms; and improve quality of life, especially among individuals who are high users of mental health services. The ACT model has been implemented in 33 states.

Intensive case management

Intensive case management practices are typically targeted to individuals with the greatest service needs, including individuals with a history of multiple hospitalizations, persons dually diagnosed with substance abuse problems, individuals with mental illness who have been involved with the criminal justice system, and individuals who are both homeless and severely mentally ill.

A recent (2002) mail survey of 22 experts found that while intensive case management shares many critical ingredients with ACT programs, its elements are not as clearly articulated. Another distinction between intensive case management and ACT appears to be that the latter relies more heavily on a team versus individual approach. In addition, intensive case managers are more likely to "broker" treatment and rehabilitation services rather than provide them directly. Finally, intensive case management programs are more likely to focus on client strengths, empowering clients to fully participate in all treatment decisions.

Clinical case management

A meta-analytic study comparing ACT and clinical case management found that while the generic approach resulted in increased hospital admissions, it significantly decreased the length of stay. This suggests that the overall impact of clinical case management is positive. Consistent with prior research, the study concluded that both ACT and high-quality clinical case management should be essential features of any mental health service system. One of the greatest tragedies of deinstitutionalization has been that most families, without any training or support, often become de facto case managers for their family members.

Case management for children and adolescents

Case management is also used to coordinate care for children with serious emotional disturbances—diagnosed mental health problems that substantially disrupt a child's ability to function socially, academically, and emotionally. Although not a formal **diagnosis** in the ***Diagnostic and Statistical Manual of Mental Disorders*** *(DSM),* the handbook published by the American Psychiatric Association used by mental health professionals to diagnose mental disorders, the term "serious emotional disturbance" is commonly used by states and the federal government to identify children with the greatest service needs. While the limited research on case management for children and youth with serious emotional disturbances has been primarily focused on service use rather than clinical outcomes, there is growing evidence that case management is an effective **intervention** for this population.

Case management models used for children vary considerably. One model, called "wraparound," helps families develop a plan to address the child's individual needs across multiple life domains (home and school, for example). Research on the effectiveness of this model is still in an early stage. Another model, known as the children and youth intensive case management or expanded broker model had been evaluated in two controlled studies. Findings suggest that this broker/advocacy model results in behavioral improvements and fewer days in hospital settings.

Conclusion

In recent years, many case management programs have expanded their teams to successfully utilize consumers as peer counselors and family members as outreach workers. The programs have also been adapted to serve older individuals with severe and persistent mental illnesses. While the ACT model offers the strongest evidential base for its effectiveness, research into the clinical and service system outcomes of this and other models of case management is ongoing.

The effectiveness of any case management program depends upon the availability of high-quality treatment and support services in a given community, the structure and coordination of the service system, and on the ability of an individual or family to pay for care either through private insurance or (more often) through public benefit and entitlement programs. With recent policy directives from the Centers for Medicaid and Medicare Services (formerly the Health Care Financing Administration or HCFA) promoting the use of Medicaid funds

for ACT, more states are funding case management through Medicaid. While some policy makers express concern about costs, the expense of these programs is usually offset by the savings realized from keeping patients out of jails, hospitals, and emergency rooms. Compared to traditional outpatient programs, case management also offers a level of care that is far more comprehensive and humane for a disabled population.

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Irene S. Levine, Ph.D.

CAT *see* **Children's Apperception Test**

CAT scan *see* **Computed tomography**

Catapres *see* **Clonidine**

Catatonia

Definition

Catatonia is a disturbance of motor behavior that can have either a psychological or neurological cause. Its most well-known form involves a rigid, immobile position that is held by a person for a considerable length of time—often days, weeks, or longer. It can also refer to agitated, purposeless motor activity that is not stimulated by something in the environment. A less extreme form of catatonia involves very slowed motor activity. Often, the physical posture of a catatonic individual is unusual and/or inappropriate, and the individual may hold a posture if placed in it by someone else. According to the handbook used by mental health professionals to diagnose mental disorders, the *Diagnostic and Statistical Manual of the American Psychiatric Association*, 4th Edition, Text Revision, also known as the *DSM-IV-TR*, some 5–9% of all psychiatric inpatients show some catatonic symptoms. Of these, 25–50% are associated with mood disorders, 10–15% are associated with **schizophrenia**, and the remainder are associated with other mental disorders.

Description

Types of catatonia

CATATONIC SCHIZOPHRENIA. As with all types of schizophrenia, the catatonic type, fortunately rare today, involves a marked disturbance in all spheres of life. As a schizophrenic disorder, the individual shows disturbances in thinking, feeling, and behavior. Most schizophrenics are unable to form meaningful intimate relationships or train for and sustain meaningful employment.

The catatonic type of schizophrenia is characterized by severe psychomotor disturbance. Individuals with this disorder show extreme immobility. They may stay in the same position for hours, days, weeks, or longer. The position they assume may be unusual and appear uncomfortable to the observer. If another person moves part of the catatonic individual's body, such as a limb, he or she may maintain the position into which they are placed, a condition known as "waxy flexibility." Sometimes catatonia presents itself as excessive motor activity, but the activity seems purposeless, and does not appear to fit with what is happening in the environment. In its most severe forms, whether stupor or agitation, the individual may need close supervision to keep from injuring him- or herself, or others.

DEPRESSION WITH CATATONIC FEATURES. Individuals who are severely depressed may show disturbances of motor behavior that is similar to that of catatonic schizophrenics, as previously described. They may be essential-

KEY TERMS

Catatonia—Disturbance of motor behavior with either extreme stupor or random, purposeless activity.

Catatonic schizophrenia—A subtype of a severe mental disorder that affects thinking, feeling and behavior, and that is also characterized by catatonic behaviors—either extreme stupor or random, purposeless activity.

Echolalia—Meaningless repetition of words or phrases spoken by another.

Echopraxia—Imitation of another person's physical movements in a repetitious or senseless manner.

Waxy flexibility—A condition in which a person can be molded into a strange position and hold that position for a long period of time.

ly immobile, or exhibit excessive but random-seeming motor activity. Extreme negativism, elective mutism (choosing not to speak), peculiar movements, mimicking words or phrases (known as "echolalia") or mimicking movements (known as "echopraxia") may also be part of the picture. Again, in its most extreme forms, catatonic stupor (not moving for hours, days, weeks, or longer), and catatonic activity (random-seeming activity) may necessitate supervision so that the individual does not hurt him- or herself, or others. Catatonic behaviors may also be seen in persons with other mood disorders, such as manic or mixed-mood states; these are also known as Bipolar I and Bipolar II disorders.

CATATONIC DISORDER DUE TO GENERAL MEDICAL CONDITION. Individuals with catatonia due to a medical condition may show symptoms similar to persons with catatonic schizophrenia and catatonic depression. However, the cause is believed to be physiological. Certain neurologic diseases, such as encephalitis, may cause catatonic symptoms that can be either temporary, or lasting.

See also Affect; Bipolar disorders; Catatonic disorder; Major depressive disorder; Manic episode; Schizophrenia

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Catatonic disorders

Definition

Catatonic disorders are a group of symptoms characterized by disturbances in motor (muscular movement) behavior that may have either a psychological or a physiological basis. The best-known of these symptoms is immobility, which is a rigid positioning of the body held for a considerable length of time. Patients diagnosed with a catatonic disorder may maintain their body position for hours, days, weeks or even months at a time. Alternately, catatonic symptoms may look like agitated, purposeless movements that are seemingly unrelated to the person's environment. The condition itself is called **catatonia**.

A less extreme symptom of catatonic disorder is slowed-down motor activity. Often, the body position or posture of a catatonic person is unusual or inappropriate; in addition, he or she may hold a position if placed in it by someone else.

Description

Types of catatonic disorder

CATATONIC SCHIZOPHRENIA. Schizophrenia is a severe, usually life-long mental illness that affects every aspect of human functioning. Thinking, feeling, and behavior are all affected by the disorder; and the person with schizophrenia usually has difficulties in interpersonal relationships as well as in obtaining and keeping meaningful employment. The catatonic subtype of schizophrenia is, fortunately, rare today in North America and Europe. It is characterized by severe disturbances in motor behavior. Individuals with catatonic schizophrenia often show extreme immobility. They may stay in the same position for hours, days, weeks, or longer. The position they assume may be unusual and appear uncomfortable to the observer; for example, the person may stand on one leg like a stork, or hold one arm outstretched for a long time. If an observer moves a hand or limb of the catatonic person's body, he or she may maintain the new

KEY TERMS

Akinesia—Absence of physical movement.

Catalepsy—An abnormal condition characterized by postural rigidity and mental stupor, associated with certain mental disorders.

Catatonic disorder—A severe disturbance of motor behavior characterized by either extreme immobility or stupor, or by random and purposeless activity.

Catatonic schizophrenia—A subtype of a severe mental disorder that affects thinking, feeling and behavior, and that is also characterized by catatonic behaviors—either extreme stupor or random, purposeless activity.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Echolalia—Meaningless repetition of words or phrases spoken by another.

Echopraxia—Imitation of another person's physical movements in a repetitious or senseless manner.

Hypomania—A milder form of mania which is characteristic of bipolar II disorder.

Mutism—Inability to speak due to conscious refusal or psychological inhibition.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Stupor—A trance-like state that causes a person to appear numb to their environment.

Waxy flexibility—A condition in which a person's body part, usually a limb, can be moved by others into diffferent positions, where it remains for long periods of time.

position. This condition is known as waxy flexibility. In other situations, a person with catatonic schizophrenia may be extremely active, but the activity appears bizarre, purposeless, and unconnected to the situation or surroundings. The patient may, for example, run up and down a flight of stairs repeatedly. Catatonic stupor is characterized by extremely slowed motor activity, often to the point of being motionless and appearing unaware of surroundings. The patient may exhibit negativism, which means that he or she resists all attempts to be moved, or all instructions or requests to move, without any apparent motivation.

Catatonic symptoms were first described by the **psychiatrist** Karl Ludwig Kahlbaum in 1874. Kahlbaum described catatonia as a disorder characterized by unusual motor symptoms. His description of individuals with catatonic behaviors remains accurate to this day. Kaulbaum carefully documented the symptoms and the course of the illness, providing a natural history of this unusual disorder.

DEPRESSION WITH CATATONIC FEATURES. People who are severely depressed may show disturbances of motor behavior resembling those of patients diagnosed with catatonic schizophrenia. These depressed persons may remain virtually motionless, or move around in an extremely vigorous but apparently random fashion. Extreme negativism, elective mutism (choosing not to speak), peculiar movements, and imitating someone else's words or phrases (echolalia) or movements (echopraxia) may also be part of the symptomatic picture. These behaviors may require caregivers to supervise the patient, to insure that he or she does not hurt him- or herself or others.

Catatonic behaviors may also occur in persons with other mood disorders. Persons experiencing manic or mixed mood states (a simultaneous combination of manic and depressive symptoms) may at times exhibit either the immobility or agitated random activity seen in catatonia. A severely depressed person may experience intense emotional pain from simply moving a finger. Even getting up out of a chair can be a painful chore that may take hours for the severely depressed individual. As the depression begins to lift, the catatonic symptoms diminish.

CATATONIC DISORDER DUE TO A GENERAL MEDICAL CONDITION. Persons with catatonic disorder due to a medical condition show symptoms similar to those of catatonic schizophrenia and catatonic depression, except that the cause is believed to be physiological. Such neurological diseases as encephalitis may cause catatonic symptoms that can be temporary or lasting.

Psychiatric symptoms caused by physiological illnesses can appear early in the course of an illness. For this reason, it is important to consider possible physical causes when catatonic symptoms appear. Persons with catatonic symptoms of physical origin generally show

greater self-awareness or insight, and more distress about their symptoms than those suffering from schizophrenia. This difference can help clinicians distinguish between patients whose catatonic symptoms stem from psychiatric causes versus those whose symptoms have a medical origin.

Causes and symptoms

Causes

CATATONIC SCHIZOPHRENIA. The cause of schizophrenia remains unknown. During the past decade, however, research has pointed to abnormalities in structure or function of certain areas of the **brain**, including the limbic system, the frontal cortex, and the basal ganglia. These three regions are interconnected, so that dysfunction in one area may be related to structural problems in another. Brain imaging of living people and studies of the brains of deceased persons point to the limbic system as the potential site of pathology in at least some, if not most, schizophrenic patients.

DEPRESSION WITH CATATONIC FEATURES. Mood disorders are believed to be at least partially caused by irregularities in production of **neurotransmitters** within the brain. Neurotransmitters are chemicals that conduct impulses along a nerve from one nerve cell to another. Two of the most important neurotransmitters associated with depression are norepinephrine and serotonin. In animal studies, virtually all effective antidepressant medications affect the receptors for these neurotransmitters. Dopamine is another neurotransmitter that plays a role in the development of depressive disorders.

CATATONIC DISORDER DUE TO A GENERAL MEDICAL CONDITION. Numerous medical conditions can cause psychiatric symptoms. Some of the more common are infectious, metabolic, and neurological conditions. Catatonic symptoms have been linked to earlier infection with encephalitis and to Parkinson's disease. Although the appearance of patients with post-encephalitis catatonia may be similar to that of catatonic schizophrenic patients, the majority of post-encephalitic patients are not psychotic. Oliver Sacks vividly describes catatonic disorder due to encephalitis and Parkinson's disease in his 1973 book *Awakenings*.

Symptoms

CATATONIC SCHIZOPHRENIA. Catatonic schizophrenia is a form of thought disorder with prominent motor symptoms and abnormalities. These symptoms include:

- Catalepsy, or motionlessness maintained over a long period of time.
- Catatonic excitement, marked by agitation and seemingly pointless movement.
- Catatonic stupor, with markedly slowed motor activity, often to the point of immobility and seeming unawareness of the environment.
- Catatonic rigidity, in which the person assumes a rigid position and holds it against all efforts to move him or her.
- Catatonic posturing, in which the person assumes a bizarre or inappropriate posture and maintains it over a long period of time.
- Waxy flexibility, in which the limb or other body part of a catatonic person can be moved into another position that is then maintained. The body part feels to an observer as if it were made of wax.
- Akinesia, or absence of physical movement.

DEPRESSION WITH CATATONIC FEATURES. Within the category of mood disorders, catatonic symptoms are most commonly associated with bipolar I disorder. Bipolar I disorder is a mood disorder involving periods of mania interspersed with depressive episodes. Symptoms of catatonic excitement, such as random activity unrelated to the environment or repetition of words, phrases and movements may occur during manic phases. Catatonic immobility may appear during the most severe phase of the depressive cycle. The actual catatonic symptoms are indistinguishable from those seen in catatonic schizophrenia. It is also possible for catatonic symptoms to occur in conjunction with other mood disorders, including bipolar II disorder (in which a milder form of mania called hypomania occurs); mixed disorders (in which mania and depression occur at the same time); and major depressive disorders.

CATATONIC DISORDER DUE TO A GENERAL MEDICAL CONDITION. Symptoms of catatonic disorder caused by medical conditions are indistinguishable from those that occur in schizophrenia and mood disorders. Unlike persons with schizophrenia, however, those with catatonic symptoms due to a medical condition demonstrate greater insight and awareness into their illness and symptoms. They have periods of clear thinking, and their affect (emotional response) is generally appropriate to the circumstances. Neither of these conditions is true of patients with schizophrenia or severe depression.

Demographics

According to the handbook used by mental health professionals to diagnose mental disorders, the *Diagnostic and Statistical Manual of Mental Disorders,* fourth edition, Text Revision, also known as the *DSM-IV-*

A patient suffering from catatonic schizophrenia. *(Grunnitus Studios. Photo Researchers, Inc. Reproduced by permission.)*

TR, between 5% and 9% of all psychiatric inpatients show some catatonic symptoms. Of these, 25%–50% are associated with mood disorders, 10%–15% are associated with schizophrenia, and the remainder are associated with other mental disorders. Catatonic symptoms can also occur in a wide variety of general medical conditions, including infectious, metabolic and neurological disorders. They may also appear as side effects of various medications, including several drugs of abuse.

Diagnosis

Catatonic symptoms are quite noticeable. Important diagnostic distinctions, however, must be made to determine their cause. Catatonic schizophrenia is diagnosed when the patient's other symptoms include thought disorder, inappropriate affect, and a history of peculiar behavior and dysfunctional relationships. Catatonic symptoms associated with a mood disorder are diagnosed when there is a prior history of mood disorder, or after careful psychiatric evaluation. Medical tests are necessary to determine the cause of catatonic symptoms caused by infectious diseases, metabolic abnormalities, or neurological conditions. The patient should be asked about recent use of both prescribed and illicit drugs in order to determine whether the symptoms are drug-related.

Treatment

Treatment for catatonic symptoms depends on the underlying cause. Catatonic schizophrenia is treated by a variety of pharmacological and psychotherapeutic methods. **Hospitalization** may be necessary to protect the patient's safety. Supportive **psychotherapy** and **family education** can help persons with schizophrenia and their families adjust to problems created by the illness. Such other supportive services as sheltered workshops and special education may also be necessary.

Treatment of catatonic symptoms due to mood disorder involves therapy directed at the underlying mood disorder. Manic episodes are treated with such mood stabilizers as lithium and **valproic acid** (Depakote). Depressive episodes are treated with antidepressant medications or, if necessary, electroconvulsive treatment (ECT).

Catatonic symptoms caused by a medical disorder require correct **diagnosis** of the underlying medical condition, followed by appropriate treatment. Levodopa and **amantadine** (Symmetrel) have shown some effectiveness in reducing catatonic symptoms due to post-encephalitic Parkinson'sdisease. Hospitalization and careful supervision of persons with catatonic symptoms may be necessary to insure that they do not hurt themselves or others.

Prognosis

Catatonic schizophrenia is usually a debilitating lifelong illness. Symptoms typically emerge in adolescence. Social and environmental stressors, such as leaving home for college or military service, use of an illicit drug, or the death of a close friend or relative may trigger the initial symptoms of schizophrenia. The classic pattern is one of worsened symptoms alternating with remissions rather than cure, although about 20% of patients eventually resume their previous level of functioning. Following the initial episode, most patients suffer a relapse within five years of the diagnosis. The course of the disorder varies, with women having a somewhat better prognosis, but persons with schizophrenia remain vulnerable to **stress** for their lifetime.

Catatonia associated with mood disorders is somewhat more treatable, although it may also recur from time to time throughout the patients life.

Catatonic symptoms caused by medical conditions can be treated and sometimes cured. Infections are the most completely curable. Metabolic and neurological

conditions may be treatable, but various degrees of impairment may remain throughout the patient's life.

Prevention

There are no specific preventive measures for most causes of catatonia. Infectious disease can sometimes be prevented. Catatonic symptoms caused by medications or drugs of abuse can be reversed by suspending use of the drug.

See also Affect; Bipolar disorders; Major depressive disorder; Manic episode; Schizophrenia

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American Psychiatric Association. 1400 K Street NW, Washington, DC 20002. (202) 336-5500.

Mental Illness Foundation. 420 Lexington Avenue, Suite 2104, New York, NY 10170. (212) 682-4699.

National Alliance for the Mentally Ill (NAMI). 2101 Wilson Blvd., Suite 302, Arlington, VA 22201.

National Mental Health Association. 1021 Prince Street, Alexandria, VA, 22314. (703) 684-7722.

Barbara Sternberg, Ph.D.

Causes of mental illness *see* **Origin of mental illnesses**

Celexa *see* **Citalopram**

Chamomile

Definition

Chamomile is a plant that has been used since ancient Egypt in a variety of healing applications. Chamomile is a native of the Old World; it is related to the daisy family, having strongly scented foliage and flowers with white petals and yellow centers. The name chamomile is derived from two Greek words that mean "ground" and "apple," because chamomile leaves smell somewhat like apples, and because the plant grows close to the ground.

There are two varieties of chamomile commonly used in herbal preparations for internal use and for **aromatherapy**. One is called Roman chamomile (*Anthemis nobilis*), with contemporary sources in Belgium and southern England. Roman chamomile grows to a height of 9 in (23 cm) or less, and is frequently used as a ground cover along garden paths because of its pleasant apple scent. German chamomile (*Matricaria recutita*) is grown extensively in Germany, Hungary, and parts of the former Soviet Union. German chamomile grows to a height of about 3 ft (1 m) and is the variety most commonly cultivated in the United States, where it is used medicinally.

Purpose

Chamomile has been used internally for a wide variety of complaints. The traditional German description of chamomile is *alles zutraut*, which means that the plant "is good for everything."

Chamomile has been used internally for the following purposes:

- Antispasmodic: A preparation given to relieve intestinal cramping and relax the smooth muscles of the internal organs. Chamomile is used as an antispasmodic to relieve digestive disorders, menstrual cramps, premenstrual syndrome (PMS), headache, and other stress-related disorders.
- Anthelminthic: Chamomile has been used to expel parasitic worms from the digestive tract.
- Carminative: Chamomile is given to help expel gas from the intestines.
- Sedative: Perhaps the most frequent internal use of chamomile is in teas prepared to relieve anxiety and insomnia.
- Anti-inflammatory: Roman chamomile has been used to soothe the discomfort of gingivitis (inflamed gums), earache, and arthritis. German chamomile is used in

KEY TERMS

Anthelminthic—A type of medication given to expel or eliminate intestinal worms.

Antispasmodic—A medication or preparation given to relieve muscle or digestive cramps.

Carminative—A substance or preparation that relieves digestive gas.

Essential oil—The product of special ducts or cells in the tissues of aromatic plants (or the sap of certain trees) that gives the plant its characteristic aroma and therapeutic properties. Essential oils are sometimes called volatile oils because they evaporate readily at room temperature.

Flavonoids—Plant pigments that have a variety of effects on human physiology. Some of these pigments have anti-inflammatory, anti-carcinogenic, and antioxidant effects, for example.

Middle note—A term used in perfumery and aromatherapy to designate essential oils whose odors emerge later than top notes but evaporate more rapidly than bottom notes. Chamomile is considered a middle note in aromatherapy.

Tannin—An astringent compound found in chamomile, oak bark, and certain other plants. Tannin in large quantities can interfere with iron absorption.

Topical—A type of medication or preparation intended for use on the skin or external surface of the body. Chamomile is commonly used in topical preparations for acne, open skin irritations, and similar conditions because of its antibacterial properties.

Europe to treat oral mucosities in cancer patients following chemotherapy treatment.

- Antiseptic: Chamomile has mild antibacterial properties, and is sometimes used as a mouthwash or eyewash. It can be applied to compresses to treat bruises or small cuts.
- Other: Mexican Americans, especially the elderly, have been reported to use chamomile for the treatment of asthma and urinary incontinence. It is one of the two most popular herbs in use among this population.

The external uses of chamomile include blending its essential oil with **lavender** or rose for scenting perfumes, candles, creams, or other aromatherapy products intended to calm or relax the user. Chamomile is considered a middle note in perfumery, which means that its scent lasts somewhat longer than those of top notes but is less long lasting than scents extracted from resinous or gum-bearing plants. Chamomile is also a popular ingredient in shampoos, rinses, and similar products to add highlights to blonde or light brown hair.

Other external uses of chamomile include topical preparations for the treatment of bruises, scrapes, skin irritations, and joint pain. The antibacterial and anti-inflammatory properties of chamomile make it a widely used external treatment for acne, arthritis, burns, ulcerated areas of skin, and even diaper rash. The German E Commission, regarded as an authority on herbal treatments, has recommended chamomile to "combat inflammation, stimulate the regeneration of cell tissue, and promote the healing of refractory wounds and skin ulcers."

Description

The flowers are the part of the chamomile plant that are harvested for both internal and external use. Chamomile flowers can be dried and used directly for teas and homemade topical preparations, but they are also available commercially in prepackaged tea bags and in capsule form. The essential oil of chamomile is pressed from the leaves as well as the flowers of the plant; it costs about $22–$35 for 5 ml. Chamomile is also available as a liquid extract.

The chemically active components of chamomile include alpha bisabobol, chamozulene, polyines, tannin, coumarin, flavonoids, and apigenin. However, no single factor has been credited with all the major healing properties of whole chamomile; it is assumed that the various components work together to produce the plant's beneficial effects.

Recommended dosage

Children may be given 1–2 ml of a glycerine preparation of German chamomile three times a day for colic; or 2–4 oz (57–100 g) of tea, one to three times a day, depending on the child's weight.

Adults may take a tea made from 0.7–1 oz (2–3 g) of dried chamomile steeped in hot water, three to four times daily for relief of heartburn, gas, or stomach cramps. Alternately, adults may take 5 ml of 1:5 dilution of chamomile tincture three times daily.

For use as a mouthwash, one may prepare a tea from 0.7–1 oz (2–3 g) of dried chamomile flowers, allow the tea to cool, and then gargle as often as desired. To soothe an irritated upper respiratory tract during cold season, adults may pour a few drops of essential oil of

chamomile on top of steaming water and inhale the fragrant vapors.

For relief of eczema, insect bites, and other skin irritations, adults may add 4 oz (110 g) of dried chamomile flowers to a warm bath. Topical ointments containing 3–10% chamomile may be used for psoriasis, eczema, or dry, irritated skin.

Precautions

Because chamomile is related botanically to the ragweed plant, persons who are highly allergic to ragweed should use chamomile with caution.

Chamomile is generally safe to drink when prepared using the recommended quantity of dried flowers. Highly concentrated tea made from Roman chamomile has been reported to cause nausea; this reaction is caused by a compound found in Roman chamomile called anthemic acid.

Women who are pregnant or lactating should not use chamomile.

Persons taking warfarin or similar blood-thinning medications should use chamomile only after consulting their physician, as it may intensify the effects of anticoagulant drugs.

Side effects

Chamomile can cause allergic reactions in people who are sensitive to ragweed.

Interactions

Chamomile can increase the effects of anticoagulant medications. In addition, its tannin content may interfere with iron absorption. Chamomile may also add to the effects of benzodiazepines, including Valium, Ativan, and Versed. No other noteworthy medication interactions have been reported.

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National Association for Holistic Aromatherapy (NAHA). 4509 Interlake Avenue North, #233, Seattle, WA 98103-6773. (888) ASK-NAHA or (206) 547-2164. <www.naha.org>.

Rebecca J. Frey, Ph.D.

Child abuse *see* **Abuse**

Child Depression Inventory

Definition

The Child Depression Inventory (CDI) is a symptom-oriented instrument for assessing depression in children between the ages of seven and 17 years. The basic CDI consists of 27 items, but a 10-item short form is also available for use as a screener.

Purpose

The CDI was first published by Maria Kovacs in 1992. It was developed because depression in young children is often difficult to diagnose, and also because depression was regarded as an adult disorder until the 1970s. It was thought that children's nervous systems were not sufficiently mature to manifest the neurochemical changes in **brain** function associated with depression.

In 2002 the National Institute of Mental Health (NIMH) estimated that as many as 2.5% of children and 8.3% of adolescents under the age of 18 in the United States suffer from depression. A study sponsored by the NIMH of 9- to 17-year-olds found that 6% developed depression in a six-month period, with 4.9% diagnosed as having major depression. Research also indicates that children and adolescents experience the onset of depression at earlier ages than previous generations, are more likely to experience recurrences, and are more likely to experience severe depression as adults.

KEY TERMS

Dysthymic disorder—A mood disorder that is less severe than depression but usually more chronic. Dysthymic disorder is diagnosed in children and adolescents when a depressed mood persists for a least one year and is accompanied by at least two other symptoms of major depression.

Epidemiology—The study of the causes, incidence, transmission, and control of diseases.

Frequency distribution—In statistics, the correspondence between a set of frequencies and the set of categories used to classify the group being tested.

Psychometric—Pertaining to testing and measurement of mental or psychological abilities. Psychometric tests convert an individual's psychological traits and attributes into a numerical estimation or evaluation.

Self-rated—A term in psychological testing that means that the person taking the test is the one who decides whether a question applies to them and records the answer, as distinct from an examiner's evaluating and recording answers.

Standard deviation—A measure of variability in a set of scores. The standard deviations are based on a comparison to others in the same age group. Standardizing the scores in this way allows scores across age groups to be compared.

The CDI is intended to detect and evaluate the symptoms of a **major depressive disorder** or **dysthymic disorder** in children or adolescents, and to distinguish between children with those disorders and children with other psychiatric conditions. The CDI can be administered repeatedly in order to measure changes in the depression over time and to evaluate the results of treatment for depressive disorders. It is regarded as adequate for assessing the severity of the depressive symptoms.

The CDI has also been used in research studies of the epidemiology of depression in children as well as studies of dissociative symptoms and post-traumatic syndromes in children. It has been rated as having adequate to excellent psychometric properties by research psychologists.

Precautions

The CDI shares certain drawbacks with other self-report measures used in children, namely that children do not have the same level of ability as adults to understand and report strong internal emotions. On the other hand, children have the same ability as adults to modify their answers on the CDI and similar tests to reflect what they think are the desired answers rather than what they actually feel. This phenomenon is variously known as "faking good" or "faking bad," depending on the bias of the modified answers. Some researchers have also observed that children who do not have age-appropriate reading skills may receive an inaccurate **diagnosis** on the basis of their CDI score.

The results of the CDI should be evaluated only by a trained professional **psychologist** or **psychiatrist**, not by a parent, teacher, or school nurse.

Because depressive symptoms fluctuate somewhat in children as well as in adults, the author of the test recommends retesting children who score positive on the CDI, with a two- to four-week interval between the test and the retest. A child who screens positive on the CDI should receive a comprehensive diagnostic evaluation by a licensed mental health professional. The evaluation should include interviews with the child or adolescent; the parents or other caregivers; and, when possible, such other observers as teachers, social service personnel, or the child's primary care physician.

Description

The CDI is self-rated, which means that the child or adolescent being evaluated records their answers to the questions on the test sheet, as distinct from giving verbal answers to questions that are then analyzed and recorded by the examiner. Other self-rated instruments for assessing depression in children include the **Beck Depression Inventory** (BDI) and the Weinberg Screening Affective Scale (WSAS).

Each question on the CDI consists of three possible responses; the child or adolescent being evaluated selects the response that most closely describes him or her over the preceding two weeks. The CDI is designed to make quantitative measurements of the following symptoms of depression: mood disturbances; capacity for enjoyment; depressed self-evaluation; disturbances in behavior toward other people; and vegetative symptoms, which include **fatigue**, oversleeping, having difficulty with activities requiring effort, and other symptoms of passivity or inactivity.

Results

The test administrator totals the responses and plots them onto a profile form. A score that falls below a cut-

off point, or is 1.0 to 2.0 standard deviations above the mean, is considered to be positive for depression.

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American Psychological Association. 750 First Street, NE, Washington, DC 20002. (202) 336-5500. <www.apa.org>.

National Depressive and Manic-Depressive Association. 730 North Franklin Street, Suite 501, Chicago, IL 60610-3526. (800) 826-3632. <www.ndmda.org>.

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Rebecca J. Frey, Ph.D.

Childhood disintegrative disorder

Definition

Childhood disintegrative disorder (CDD) is a developmental disorder that resembles **autism**. It is characterized by at least two years of normal development, followed by loss of language, social skills, and motor skills before age ten. Other names for childhood disintegrative disorder are Heller's syndrome, **dementia** infantilis, and disintegrative **psychosis.**

Description

Thomas Heller, an Austrian educator, first described childhood disintegrative disorder in 1908. It is a complex disorder that affects many different areas of the child's development. It is grouped with the **pervasive developmental disorders** (PDDs) and is related to the better known and more common disorder of autism.

Initially CDD was considered strictly a medical disorder and was believed to have identifiable medical causes. After researchers reviewed the reported cases of CDD, however, no specific medical or neurological cause was found to account for all occurrences of the disorder. For that reason, CDD was included in the fourth edition of the ***Diagnostic and Statistical Manual of Mental Disorders***, or *DSM-IV*, in 1994. The *Diagnostic and Statistical Manual* is the standard reference work consulted by mental health professionals in the United States and Canada.

Causes and symptoms

Causes

The cause of childhood disintegrative disorder is unknown. Research findings suggest, however, that it may arise in the neurobiology of the **brain**. About half the children diagnosed with CDD have an abnormal electroencephalogram (EEG). EEGs measure the electrical activity in the brain generated by nerve transmission (brain waves). CDD is also sometimes associated with **seizures**, another indication that the neurobiology of the brain may be involved. CDD is occasionally associated with such diagnosed medical disorders of the brain as leukodystrophy and Schilder's disease; but no one disease, brain defect, disorder, or condition can account for all symptoms and all cases. Research is hampered by the rarity of this disorder.

Symptoms

Children with CDD have at least two years of normal development in all areas—language understanding, speech, skill in the use of large and small muscles, and social development. After this period of normal growth, the child begins to lose the skills he or she has acquired. This loss usually takes place between ages three and four, but it can happen any time up to age ten.

KEY TERMS

Autism—A developmental disability that appears early in life, in which normal brain development is disrupted and social and communication skills are retarded, sometimes severely.

Dementia infantilis—Another term for childhood disintegrative disorder, used more frequently in the European medical literature. The Latin name literally means "early childhood dementia."

Leukodystrophy—A disturbance of the white matter of the brain

Schilder's disease—A disturbance of the white matter of the brain that causes blindness, deafness, and mental deterioration

Sensory integration therapy—A treatment that was originally designed for children with autism. Sensory integration therapy is often performed by occupational or physical therapists; its goal is to help the child with autism or CDD process information acquired through the senses (hearing, touch, taste, and smell as well as sight) more effectively.

The loss of skills may be gradual, but more often occurs rapidly over a period of six to nine months. The transition may begin with unexplained changes in behavior, such as anxiety, unprovoked anger, or agitation. Behavioral changes are followed by loss of communication, social, and motor skills. Children may stop speaking or revert to single words. They often lose bowel or bladder control and withdraw into themselves, rejecting social interaction with adults or other children. They may perform repetitious activities and often have trouble moving from one activity to the next.

In this way CDD resembles autism. In autism, however, previously acquired skills are not usually lost. According to the *Handbook of Autism and Pervasive Developmental Disorders*, virtually all children with CDD lose speech and social skills. About 90% lose self-help skills (the ability to feed, wash, and toilet themselves); and about the same number develop non-specific overactivity. After a time, the regression stops, but the child does not usually regain the skills that were lost.

Demographics

CDD is a rare disease, much less common than autism. About 1 in 100,000 children are thought to have CDD. It is possible, however, that the disorder is underdiagnosed. For a long time, it was thought that CDD occurred equally among boys and girls. Newer research suggests that it is about four times more common in boys, and that many girls who were diagnosed with CDD actually had **Rett's disorder**, a disorder that shares many of the symptoms of CDD but occurs almost always in girls.

Diagnosis

CDD is most commonly diagnosed when the parents of the affected child consult the pediatrician about the child'sloss of previously acquired skills. The doctor will first give the child a medical examination to rule out epilepsy or other medical conditions. The child'shead may also be x rayed to rule out head trauma or a brain tumor. Following the medical examinations and tests, the child will be referred to a **psychiatrist** who specializes in treating children and adolescents. The psychiatrist will then make the differential **diagnosis** of CDD.

To be diagnosed with CDD, a child must show loss or regression in at least two of the areas listed below. Usually regression occurs in more than two areas. These are:

- receptive language skills (language understanding)
- expressive language skills (spoken language)
- social or self-help skills
- play with peers
- motor skills
- bowel or bladder control, if previously established

Children with CDD are unable to start conversations with other people and often do not communicate with nonverbal signals (smiles, gestures, nodding the head, etc.) either. They also lose interest in playing games and in relationships with other people. They may engage in strange repetitive behavior, such as bobbing the head up and down, or other repeated movements. These changes must not be caused by a general medical condition or another diagnosed mental disorder.

CDD must be differentiated from autism and such other specific pervasive developmental disorders as Rett's disease. It also must be differentiated from **schizophrenia**. One of the differences between CDD and other PDDs is that to be diagnosed with CDD, a child must develop normally for at least two years before loss of skills occurs, and the loss must occur before age ten. Parents' reports of the child's development, records in baby books, medical records kept by the child's pediatrician, and home movies are often used to document normal development through the first two years of life.

Treatments

Treatment for CDD is very similar to treatment for autism. The emphasis falls on early and intense educational interventions. Most treatment is behavior-based and highly structured. Educating the parents so that they can support the child'streatments at home is usually part of the overall treatment plan. Speech and language therapy, occupational therapy, social skills development, and sensory integration therapy may all be used according to the needs of the individual child

Families with a child who has CDD often find themselves highly stressed. Practical demands on caregivers are high, and CDD takes an emotional toll on family members. Finding appropriate providers with experience delivering services for a child with CDD is sometimes difficult, especially outside large cities. **Support groups** for families can help reduce their isolation and frustration. Because CDD is rare, autism support groups and organizations include families of children with CDD in their services.

Prognosis

The prognosis for children with CDD is very poor; it is worse than the prognosis for children with autism. Once skills are lost, they are not usually regained. Only about 20% of children diagnosed with the disorder reacquire the ability to speak in sentences. Most adults with CDD remain dependent on full-time caregivers or are institutionalized.

Prevention

Since the causes of CDD are unknown, there are no known ways to prevent this disorder.

Resources

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ORGANIZATIONS

Autism Society of America. 7910 Woodmont Avenue, Suite 300, Bethesda, MD 20814-3067. (301) 657-0881 or 800-3AUTISM. <htto://www.autism-society.org>.

National Association of Rare Disorders (NORD). P.O. Box 8923, New Fairfield, CT 06812-8923. (800) 999-NORD or (203) 746-6518.

Tish Davidson, A.M.

Children's Apperception Test

Definition

The Children's Apperception Test, often abbreviated as CAT, is an individually administered projective personality test appropriate for children aged three to 10 years.

Purpose

The CAT is intended to measure the personality traits, attitudes, and psychodynamic processes evident in prepubertal children. By presenting a series of pictures and asking a child to describe the situations and make up stories about the people or animals in the pictures, an examiner can elicit this information about the child.

The CAT was originally developed to assess psychosexual conflicts related to certain stages of a child's development. Examples of these conflicts include relationship issues, sibling rivalry, and aggression. Today, the CAT is more often used as an assessment technique in clinical evaluation. Clinical diagnoses can be based in part on the Children's Apperception Test and other projective techniques.

Precautions

A **psychologist** or other professional person who is administering the CAT must be trained in its usage and interpretation, and be familiar with the psychological theories underlying the pictures. Because of the subjective nature of interpreting and analyzing CAT results, caution should be used in drawing conclusions from the test results. Most clinical psychologists recommend using the CAT in conjunction with other psychological tests designed for children.

The CAT is frequently criticized for its lack of objective scoring, its reliance on the scorer's own scoring method and bias, and the lack of accepted evidence for its reliability (consistency of results) and validity (effectiveness in measuring what it was designed to measure). For example, no clear evidence exists that the test measures needs, conflicts, or other processes related to human motivations in a valid and reliable way.

KEY TERMS

Apperception—The process of understanding through linkage with previous experience. The term was coined by one of the authors of the TAT to underscore the fact that people don't "perceive" the story cards in a vacuum; rather, they construct their stories on the basis of past experiences as well as present personality traits.

Defense—An unconscious mental process that protects the conscious mind from unacceptable or painful thoughts, impulses, or desires. Examples of defenses include denial, rationalization, projection, and repression. Some defenses are considered to represent lower levels of maturation than others; thus identifying a child's defenses may be helpful in evaluating his or her level of psychological maturity.

Ego—In Freudian psychology, the conscious, rational part of the mind that experiences and reacts to the outside world.

Projective test—A psychological test in which the test taker responds to or provides ambiguous, abstract, or unstructured stimuli, often in the form of pictures or drawings.

Psychodynamic—Referring to the motivational forces, unconscious as well as conscious, that form human attitudes and behavior.

Psychosexual conflicts—In Freudian categories, internal conflicts related to problems at a particular stage of childhood development. Freud associated each developmental stage with a particular part of the human body, such as the mouth or the phallus.

Reliability—The ability of a test to yield consistent, repeatable results.

Sibling rivalry—Competition among brothers and sisters in a nuclear family. It is considered to be an important influence in shaping the personalities of children who grow up in middle-class Western societies but less relevant in traditional African and Asian cultures.

Superego—According to Freud, the part of the mind that represents traditional parental and societal values. The superego is the source of guilt feelings.

Validity—The ability of a test to measure accurately what it claims to measure.

Older children between the ages of seven and 10 years may feel that the animal pictures in the original version of the CAT are too childish for them. They may respond better to the pictures of human beings available in the Children's Apperception Test-Human Figures (CAT-H), a version of the CAT in which human beings replace animals in the pictures.

Description

The Children's Apperception Test was developed in 1949 by Leopold Bellak and Sonya Sorel Bellak. It was an offshoot of the widely used **Thematic Apperception Test** (TAT), which was based on Henry Murray's need-based theory of personality. Bellak and Bellak developed the CAT because they saw a need for an apperception test specifically designed for children. The most recent revision of the CAT was published in 1993.

The original CAT featured ten pictures of animals in such human social contexts as playing games or sleeping in a bed. Today, this version is known as the CAT or the CAT-A (for animal). Animals were chosen for the pictures because it was believed that young children relate better to animals than humans. Each picture is presented by a test administrator in the form of a card. The test is always administered to an individual child; it should never be given in group form. The test is not timed but normally takes 20–30 minutes. It should be given in a quiet room in which the administrator and the child will not be disturbed by other people or activities.

The second version of the CAT, the CAT-H, was developed in 1965 by Bellak and Bellak. The CAT-H includes ten pictures of human beings in the same situations as the animals in the original CAT. The CAT-H was designed for the same age group as the CAT-A but appeals especially to children aged seven to 10, who may prefer pictures of humans to pictures of animals.

The pictures on the CAT were chosen to draw out children's fantasies and encourage storytelling. Descriptions of the ten pictures are as follows: baby chicks seated around a table with an adult chicken appearing in the background; a large bear and a baby bear playing tug-of-war; a lion sitting on a throne being watched by a mouse through a peephole; a mother kangaroo with a joey (baby kangaroo) in her pouch and an older joey beside her; two baby bears sleeping on a small bed in front of a larger bed containing two bulges; a cave in which two large bears are lying down next to a baby bear; a ferocious tiger leaping toward a monkey who is trying to climb a tree; two adult monkeys sitting on a sofa while another adult monkey talks to a baby monkey; a rabbit sitting on a child's bed viewed through a doorway; and a puppy being spanked by an adult dog in front of a bathroom. The cards

in the human version substitute human adults and children for the animals but the situations are the same. Gender identity, however, is more ambiguous in the animal pictures than in the human ones. The ambiguity of gender can allow for children to relate to all the child animals in the pictures rather than just the human beings of their own sex.

The pictures are meant to encourage the children to tell stories related to competition, illness, injuries, body image, family life, and school situations. The CAT test manual suggests that the administrator should consider the following variables when analyzing a child's story about a particular card: the protagonist (main character) of the story; the primary needs of the protagonist; and the relationship of the main character to his or her personal environment. The pictures also draw out a child's anxieties, fears, and psychological defenses.

One theoretical basis for the CAT and other apperception tests is Murray's theory of personality. Murray is credited with clarifying the concept of human needs. He believed that a person's needs affect the way in which he or she interacts with the environment. The pictures on the CAT often address the manner in which individuals interact with their environment in terms of need fulfillment. Murray developed the Thematic Apperception Test, or TAT, in order to assess the relative strength of a person's needs. The needs that Murray particularly emphasized include the need for achievement and the need for recognition.

Because the primary content of the CAT consists of pictures, it is widely used in countries outside the United States.

Results

Scoring of the Children's Apperception Test is not based on objective scales; it must be performed by a trained test administrator or scorer. The scorer's interpretation should take into account the following variables: the story's primary theme; the story's hero or heroine; the needs or drives of the hero or heroine; the environment in which the story takes place; the child's perception of the figures in the picture; the main conflicts in the story; the anxieties and defenses expressed in the story; the function of the child's superego; and the integration of the child's ego.

Consider, for example, the card in which a ferocious tiger leaps toward a monkey who is trying to climb a tree. A child may talk about his or her fears of aggression or punishment. The monkey may be described as a hero escaping punishment from the evil tiger. This story line may represent the child's perceived need to escape punishment from an angry parent or a bully. Conversely, a child may perceive the picture in a relatively harmless way, perhaps seeing the monkey and tiger playing an innocent game.

A projective test like the CAT allows for a wide variety of acceptable responses. There is no "incorrect" response to the pictures. The scorer is responsible for interpreting the child's responses in a coherent way in order to make the test useful as a clinical assessment technique. It is recommended practice for the administrator to obtain the child's personal and medical history before giving the CAT, in order to provide a context for what might otherwise appear to be abnormal responses. For example, it would be normal under the circumstances for a child whose pet has just died to tell stories that include themes of **grief** or loss even though most children would not respond to the cards in that way.

A person scoring the CAT has considerable flexibility in interpretation. He or she can use the analysis of a child's responses to support a psychological **diagnosis**, provide a basis for a clinical evaluation, or gain insight into the child's internal psychological structure.

See also Rorschach technique

Resources

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Ali Fahmy, Ph.D.

Chloral hydrate

Definition

Chloral hydrate is a drug that is used to help sedate persons before and after surgery, to help relieve anxiety or tension, and to help promote sleep in persons with **insomnia**. It is sold in the United States under the brand names Aquachloral and Noctec. It is also available under its generic name.

KEY TERMS

Hydrated—Combining a substance with water.

Hypnotic—A type of medication that induces sleep.

Porphyria—A group of disorders that arise from changes in the metabolism of porphyrin, a naturally occurring compound in the body, and that are characterized by acute abdominal pain and neurological problems.

Sedative—A medication that induces relaxation and sleep.

Purpose

Chloral hydrate is primarily used to help sedate persons, especially children, before and after surgery. It has a calming effect on persons as they prepare for surgery. It is also used to help persons who have sleep difficulties fall asleep. Chloral hydrate can be used to help calm tense or nervous persons.

Description

Chloral hydrate is classified as a sedative-hypnotics drug. The entire mechanism by which chloral hydrate works is not completely understood. It is believed that a chemical produced by chloral hydrate called trichloroethanol causes a mild depressive effect on the **brain**.

Recommended dosage

Chloral hydrate is available in oral and suppository forms. The oral form includes both capsules and a syrup. Adults usually receive 500 mg–1000 mg taken 15–30 minutes before bedtime or one to two hours before surgery. These dosages are for hypnotic effects. For sedative effects, 250 mg is usually taken three times daily after meals. Total daily dosage should not be more than 2 g (2000 mg). The hypnotic dose for children is usually 50 mg for every kilogram of body weight. The maximum amount per single dose is 1 g. Daily dosage is usually divided into several smaller doses and taken throughout the day. The sedative dose is typically one-half of the hypnotic dose. The syrup form should be combined with a half glass of fruit juice or water. The capsules should be taken with a full glass of water or juice to help prevent stomach upset.

The typical hypnotic dose using suppositories is 10–20 grains before bedtime in adults. The sedative dose for adults is 5–10 grains three times daily. The total maximum suppository dose should not be more than 30 grains per day. The hypnotic suppository dose in children is 5 grains for every 40 pounds (18.2 kg) of body weight. The sedative dose is one-half of this amount. The amount of absorption of these suppositories is primarily based on how well the body is hydrated and not on body temperature. It helps to moisten the suppository and finger before inserting the suppository.

Precautions

The treating doctor needs to check the progress of any patients taking this drug for more than a few days to ensure significant side effects are not developing. Patients should not stop taking chloral hydrate suddenly. Instead, the dosage should be gradually decreased over time. Chloral hydrate can produce increased effects when combined with other central nervous depressants such as alcohol, antihistamines, and tranquilizers. The combination of chloral hydrate with these agents can cause significant drowsiness. Chloral hydrate can sometimes cause persons to become drowsy, lightheaded, or dizzy. Chloral hydrate should generally not be used in patients with a history of severe kidney disease, severe liver disease, or those with a history of significant heart disease.

Chloral hydrate should be used with great caution only where necessary in persons with a history of heart disease, gastrointestinal problems, porphyria, drug abuse, and in the elderly. Chloral hydrate should be used with caution in pregnant women and in women who are nursing. Chloral hydrate, as with most drugs, can be taken in excess to the point of overdose. Signs of overdose include difficulty in swallowing, extreme weakness, confusion, **seizures**, extreme drowsiness, low body temperature, staggering, changes in heart rate, and breathing problems.

Side effects

Uncommon but serious side effects of chloral hydrate use include skin rash or hives. Even more rare side effects include confusion, hallucination, and excessive excitement. The development of any of these side effects should be promptly reported to a doctor.

Less serious but more common side effects of chloral hydrate use include nausea, stomach pain, and vomiting. Less common and not particularly serious side effects include diarrhea, lightheadedness, drowsiness, and clumsiness.

Interactions

Chloral hydrate should not be combined with alcohol because of additive depressant effects on the central nervous system. This combination can lead to significant drowsiness. Likewise, chloral hydrate should not be combined with the antidepressants drugs called tricyclic antidepressants because of the additive depressive effect. Chloral hydrate should not be combined with the blood-thinning drug called warfarin. The combination of these drugs may require adjustments in the amount of the warfarin taken.

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Chlordiazepoxide

Definition

Chlordiazepoxide is used for the treatment of anxiety. It is a member of the benzodiazepine family of compounds, which slow the central nervous system in order to ease tension or nervousness. In the United States, it is sold under the trade name of Librium.

Purpose

Chlordiazepoxide is used for the short-term relief of symptoms of anxiety and management of anxiety disorders. It is also used for treating symptoms of withdrawal from acute alcoholism and alcoholic intoxication.

Description

Chlordiazepoxide is useful when treating anxiety for short periods of time. It has sedative properties that are useful for brief periods of use. It is occasionally used to stimulate appetites and is a weak analgesic. The precise mechanism of action is not known. Several hours are needed for peak levels of the drug to be achieved. Chlordiazepoxide is available in 5-, 10-, and 25-mg capsules.

KEY TERMS

Analgesic—A substance that provides relief from pain.

Edema—Abnormal accumulation of fluid in the interstitial spaces of bodily tissue.

Libido—Psychic energy or instinctual drive associated with sexual desire, pleasure, or creativity.

Porphyria—A group of disorders that arise from changes in the metabolism of porphyrin, a naturally occurring compound in the body, and that are characterized by acute abdominal pain and neurological problems.

Porphyrin—Any iron- or magnesium-free pyrrole derivative occurring in many plant and animal tissues.

Recommended dosage

Recommended dosage varies with **diagnosis**. The lowest possible dosage that provides relief from symptoms should be used as the drug has a high potential to cause physiological and psychological dependence. When used in adults for the treatment of moderate anxiety, the usual oral dosage is 5–10 mg three or four times per day. When used for the treatment of more severe **anxiety and anxiety disorders**, the usual oral dosage is 20–25 mg three or four times per day. When used by older persons, or to relieve symptoms of preoperative apprehension or anxiety, the usual oral dosage is 5 mg two to four times per day. If used as a preoperative medication, the usual dosage is 50–100 mg via intramuscular (IM) injection. When used to treat symptoms of acute alcoholism, the usual initial oral dosage is 50–100 mg, repeated as needed until agitation is adequately controlled. The recommended maximum dosage is 300 mg per day. The usual dosage for children is 5 mg two to four times per day.

Precautions

Persons with suicidal tendencies should be closely monitored, as chlordiazepoxide may lower the threshold for action and attempting **suicide**. The drug has a high

potential to cause physiological or psychological dependence.

Side effects

Other than physiological and psychological dependence, few adverse effects have been reported. The most commonly reported include drowsiness, confusion, and difficulty in moving. These are most common among older persons. Occasionally, transient loss of consciousness has been reported.

Other adverse effects include edema (abnormal accumulation of fluid in bodily tissues), minor menstrual irregularities, nausea, constipation and, infrequently, changes in libido (sex drive). Also, it may impair mental or physical skills needed to perform complex motor tasks. For this reason, persons using this drug are advised not to drive automobiles or operate machinery.

Interactions

Chlordiazepoxide may increase the effect of alcohol or other substances that depress central nervous system functions. For this reason, they should not be used at the same time. A small number of reports of interaction with oral anticoagulants have been received, and it may exacerbate porphyria—a group of inherited disorders in which there is abnormally increased production of substances called porphyrins.

See also Addiction; Alcohol and related disorders; Anti-anxiety drugs and abuse-related disorders

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax: (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

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Chlorpromazine

Definition

Chlorpromazine is an antipsychotic drug. It is a member of the phenothiazine family of compounds and is used to alleviate the symptoms and signs of **psychosis**. Psychosis is a form of severe mental illness, which is characterized by loss of contact with reality, **hallucinations**, **delusions**, agitation, and unusual behavior. In the United States, chlorpromazine is also sold under the brand name Thorazine.

Purpose

Chlorpromazine is principally used to reduce the signs and symptoms of psychosis. For this purpose, the drug is used in **schizophrenia** and the manic phase of bipolar (formerly manic-depressive) disorder. The drug is also used in the management of severe behavioral disorders with aggression, combativeness, or excessive excitability. Chlorpromazine may sometimes be used as a sedative in non-psychotic patients with excessive anxiety and agitation. In addition, the drug has been used to relieve nausea, vomiting, and persistent hiccups.

Description

Chlorpromazine was the first antipsychotic drug. It is not an exaggeration to say that the development of this medication began a revolution in the treatment of severe mental illness, which continues to this day. Patients with schizophrenia and other psychoses, who once would have been considered hopelessly untreatable and relegated to the back wards of state institutions, are often able today, as a result of treatment with chlorpromazine or similar medications, to live in the community and lead fuller lives.

The discovery of chlorpromazine resulted from efforts of pharmaceutical researchers in the first half of the twentieth century to develop sedative medications. Several drugs of a chemical class known as phenothiazines were investigated and shown to be effective sedatives, but they had little effect on agitated patients with psychosis. A new phenothiazine drug, chlorpromazine, was synthesized in France in 1950 and was tested on such patients. In 1952, two French psychiatrists, Delay and Deniker, announced that the drug exerted a specific effect in diminishing the symptoms and signs of psychosis in patients with severe mental illnesses.

The mechanism of action of chlorpromazine is not completely understood. Its antipsychotic effects are believed to be related to its action in selectively blocking the transmission of nerve impulses from cell to cell in a region of the **brain** called the limbic system. This part of the brain is involved with emotions and motivation.

Chlorpromazine, when sold under the name Thorazine, is available in many forms: tablets of 10, 25, 50, 100, and 200 mg; spansules (sustained release capsules) of 30, 75, and 150 mg; ampules for injection of 25 and 50 mg; multidose vial of 10 mL of 25 mg/mL; syrup 10mg/5mL, 4 fl oz.; suppositories of 25 and 100 mg. Generic chlorpromazine manufacturers may supply a somewhat different set of dosages and products.

KEY TERMS

Antipsychotic drug—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Epilepsy—A neurological disorder characterized by the onset of seizures. Seizures are caused by a disturbance in the electrical activity in the brain and can cause loss of consciousness, muscle spasms, rhythmic movements, abnormal sensory experiences, or altered mental states.

Neurotransmission—The conduction of a nerve impulse along a chain of nerve cells, which occurs when one cell in the chain secretes a chemical substance, called a neurotransmitter, onto a subsequent cell.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness.

Phenothiazine—A class of drugs widely used in the treatment of psychosis.

Recommended dosage

For acutely disturbed adult patients suffering from a psychosis, such as schizophrenia or mania, the usual daily dosage ranges from 100 mg to 1000 mg per day. Some patients may require a higher dosage. There is great variation in individual dosage requirements for chlorpromazine and for other antipsychotic medications. It is usually advisable to begin with a lower dosage, and increase the dosage until sufficient reduction of symptoms is achieved. Maximum reduction of symptoms may take many weeks of continued treatment. Because of the possibility of side effects, which may be severe, lower dosages should be used in outpatients, children, the elderly, and patients with serious health problems. For nonpsychotic patients with excessive anxiety or agitation, amounts used are generally less than 200 mg per day, divided among two or three doses.

For nausea and vomiting in adults, the usual dosage is 10–25 mg every four to six hours as needed, given by injection. Alternatively, doses of 50–100 mg may be given rectally. Persistent hiccups may be treated with 25–50 mg three or four times per day, orally or by injection.

Precautions

Elderly patients (those over age 65), especially women, and patients receiving long-term antipsychotic treatment are prone to develop **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements that may not disappear or may only partially improve after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw,

mouth or face or other groups of skeletal muscles. Tardive dyskinesia may also appear after chlorpromazine use has stopped. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period. The need for long-term antipsychotic medication should be weighed against the risk of tardive dyskinesia, which increases with duration of treatment.

Neuroleptic malignant syndrome (NMS), a dangerous condition with high fever, muscular rigidity, rapid pulse, sweating, and altered mental state, may occur with antipsychotic medication. NMS requires immediate medical treatment.

Phenothiazine drugs, such as chlorpromazine, may cause sedation and may interfere with driving and other tasks requiring alertness. They may increase the effects of alcohol and sedatives. The adverse effects of chlorpromazine may be increased in people with diseases of the heart, liver, or kidney, or other debilitating illnesses. Phenothiazines may lower the seizure threshold, making it more likely that a seizure will occur in people who have a history of **seizures**. People with epilepsy may require adjustment of their anti-seizure medications. Chlorpromazine may cause acute muscle spasms, particularly of the head and neck, and sudden decreases of blood pressure. Patients may need to be hospitalized during the initial phase of treatment, particularly when receiving high doses or treatment by injection.

Chlorpromazine reduces the body's ability to sweat, thus interfering with the regulation of body temperature. This may be a problem for some people in very hot weather. The problem most commonly occurs in elderly people in hot buildings without air conditioning. Body temperature may reach fatal levels. People taking chlorpromazine should be aware of the possibility of developing hyperthermia (high body temperature) in very hot weather. They should seek cool places in very hot weather.

Children may especially susceptible to neurologic reactions to phenothiazines, such as muscle spasms. Elderly patients may be particularly sensitive to sedation, low blood pressure, and other side effects. These patients should start with lower doses and increase their dosage gradually under physician supervision. Chlorpromazine may decrease salivation in older patients, predisposing to tooth decay, gum disease and mouth infections. Candy and other sugary foods should be limited, and oral hygiene should be maintained.

Chlorpromazine, like all phenothiazines, should not be taken by pregnant women because they harm the developing fetus. Breast-feeding is not recommended while taking the drug. Phenothiazines are secreted in breast milk and may cause harm to nursing infants.

Side effects

Chlorpromazine and other phenothiazines may cause many side effects. The following more common side effects are grouped by the body system affected:

- Cardiovascular: decreases of blood pressure, especially on arising, which may cause dizziness or fainting; rapid heart rate, changes in heart rhythm and electrocardiogram.
- Nervous system: sedation, muscle spasms of the head and neck, muscle rigidity, restlessness, tremors, slowed movement, shuffling gait, increased seizure tendency.
- Digestive system: dry mouth, nausea, constipation, abnormal liver tests.
- Autonomic: blurred vision, nasal congestion, reduced sweating, difficulty urinating, problems with ejaculation, impotence.
- Hormonal: lactation, breast enlargement.
- Skin: rashes, sensitivity to sunlight.
- Body as a whole: weight gain.

Interactions

Chlorpromazine interacts with a long list of other medications. Anyone starting this drug should review the other medications they are taking with their physician and pharmacist for possible interactions. Chlorpromazine and other phenothiazines may intensify the effects of drugs causing sedation, including alcohol, **barbiturates**, narcotic pain medications, minor tranquilizers, and antihistamines. Similarly, chlorpromazine may cause excessive reductions of blood pressure in patients taking other medicines that lower blood pressure. Chlorpromazine may also intensify side effects of drugs that also cause blurred vision, dry mouth, diminished sweating in hot weather, and constipation. Many other antipsychotics and antidepressants cause such effects.

Chlorpromazine may enhance the effects of medications that lower the seizure threshold, such as steroid drugs, the asthma medication theophylline, and many other psychiatric drugs. Patients with epilepsy may require dosage adjustments of their anti-seizure medications. The effectiveness of medications for Parkinson's disease may be reduced by chlorpromazine and other antipsychotics. The likelihood of changes in heart rhythm may be increased when the drug is taken with other medications that have the same effect, including other antipsychotic drugs, antidepressants, certain heart medicines, and erythromycin.

Certain drugs that are eliminated by the liver may interfere with the elimination of chlorpromazine from the

body, causing higher blood levels and increased side effects. Chlorpromazine may retard the elimination of other medicines, including many antidepressants, antipsychotic drugs, and heart medications, resulting in higher levels of these other medications and possibly increased side effects.

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Chronic motor or vocal tic disorder *see* **Tic disorders**

Cigarettes *see* **Nicotine and related disorders**

Circadian rhythm sleep disorder

Definition

Circadian rhythm sleep disorder is a persistent or recurring pattern of sleep disruption resulting either from an altered sleep-wake schedule or an inequality between a person's natural sleep-wake cycle and the sleep-related demands placed on him or her. The term circadian rhythm refers to a person's internal sleep and wake-related rhythms that occur throughout a 24-hour period. The sleep disruption leads to **insomnia** or excessive sleepiness during the day, resulting in impaired functioning.

The Fourth Edition Text Revision of the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*, a handbook used by mental health professionals to diagnose mental disorders) defines circadian rhythm sleep disorder as one of several primary **sleep disorders**. Within the category of primary sleep disorders, it is classified as one of the dyssomnias, characterized by irregularities in an individual's quality, timing, and amount of sleep. In earlier versions of the *DSM*, the disorder is called sleep-wake schedule disorder.

Description

Circadian rhythm sleep disorder involves an alteration of an individual's circadian system or a mismatch between a person's natural, or endogenous, circadian system and the external, or exogenous, demands placed on it. It can lead to insomnia at certain times of the day or excessive sleepiness throughout the day. The insomnia or excessive sleepiness results in impaired functioning in social, occupational, or other environments.

The *DSM-IV-TR* lists four types of circadian rhythm sleep disorder: delayed sleep phase type, jet lag type, shift work type, and unspecified type.

Causes and symptoms

Causes

The delayed sleep phase type of circadian rhythm sleep disorder is marked by a delay of the sleep-wake cycle as it relates to the demands of society. It is often due to a psychosocial stressor (an event in a person's environment that causes **stress** or discomfort), especially for adolescents. The delayed sleep-wake cycle leads to chronic sleep deprivation and habitually late sleeping hours. Individuals with this type often have difficulty changing their sleeping patterns to an earlier and more socially acceptable time. Their actual sleep, once it begins, is normal. It is the timing of their sleeping and waking that is persistently delayed.

The jet lag type of circadian rhythm sleep disorder is characterized by disruptions arising from a mismatch between a person's circadian cycle and the cycle required by a different time zone. The more time zones that are traveled, the greater the disruption. Eastbound travel, in which sleep-wake hours are advanced, typically causes more problems than westbound travel, in which sleep-wake hours are delayed. People who travel often and cross many time zones when they travel are most susceptible to this type.

The shift work type of circadian rhythm sleep disorder is distinguished by disruptions due to a conflict

The jet lag type of circadian rhythm sleep disorder is characterized by disruptions arising from a mismatch between a person's circadian cycle and the cycle required by a different time zone. People who travel often and cross many time zones when they travel are most susceptible to this type of circadian rhythm sleep disorder. *(Will McIntyre. Photo Researchers, Inc. Reproduced by permission.)*

between a person's endogenous circadian cycle and the cycle required by shift work. Individuals who work the night shift often experience this problem, especially those people who switch to a normal sleep schedule on days off. Also, people who work rotating shifts experience this problem because of the changing sleep-wake schedules they experience. The disruptions caused by shift work result in inconsistent circadian schedules and an inability to adjust to the changes consistently.

The unspecified type of circadian rhythm sleep disorder is characterized by a pattern of sleep-wake disturbance and circadian mismatch that is not due to the causes of the other three types. Examples of other causes include irregular sleep-wake patterns and non-24-hour sleep-wake patterns. If an individual's sleep-wake pattern is based on a period of time of slightly more than 24 hours, their circadian rhythm can become progressively delayed.

Symptoms

Individuals with the delayed sleep phase type of the disorder exhibit habitually late sleep hours and an inability to change their sleeping schedule consistently. They often show sleepiness during the desired wake period of their days. Their actual phase of sleep is normal. Once they fall asleep, they stay asleep for a normal period of time, albeit a period of time that starts and stops at an abnormally late time.

Individuals with the jet lag type of circadian rhythm sleep disorder demonstrate sleepiness during the desired wake portion of the day due to the change in time zone. They have difficulty sleeping during the desired sleep portion of the day. They also have difficulty altering their sleep-wake schedule to one appropriate to the new time zone.

Individuals with the shift work type of the disorder feel sleepy or fall asleep during the desired wake period, which includes the time spent at work. People with rotating shift schedules, especially schedules that gradually change, exhibit sleep disturbance and wake period sleepiness. Insufficient sleep time, family and social expectations, and alcohol use worsen this problem.

Individuals with the unspecified type of circadian rhythm sleep disorder also exhibit daytime and evening sleepiness or insomnia, especially those people who have a non-24-hour sleep pattern. People with irregular sleep patterns have difficulty knowing when they will fall asleep and wake up.

Demographics

The delayed sleep phase type of the disorder usually begins during adolescence and can continue without treatment through adulthood. People with this type may have a family history of delayed sleep phase. The delayed sleep phase type of the disorder is thought to impact up to 4% of adults and up to 7% of adolescents.

The shift work and jet lag types of the disorder often result in more severe symptoms for late-middle-aged and elderly people. It is estimated that up to 60% of night shift workers have the shift work type of circadian rhythm sleep disorder.

Diagnosis

In order to diagnose circadian rhythm sleep disorder, patients are often asked for records of their sleep and wake times in order to determine if a **diagnosis** is warranted. Interviews and direct observation in a sleep lab may also be utilized. A diagnosis requires a pattern of sleep disruption caused by a mismatch between a person's circadian sleep-wake pattern and the pattern required by that person's environment. The disruption can be persistent or recurrent and leads to impaired functioning, often in a social or occupational context.

To differentiate circadian rhythm sleep disorder from other diagnoses, the sleep disruption must not occur exclusively during the cause of another sleep disorder or other disorder. The disturbance in sleep must not be due

to the direct physiological effects of a substance, whether used for medication or abuse, or to a general medical condition.

The delayed sleep phase type of the disorder requires a persistent pattern of delayed sleeping and awakening and an inability to change the pattern. The jet lag type requires sleepiness and wakefulness at inappropriate times relative to the local time zone; there must be repeated travel more than one time zone away. The shift work type requires excessive sleepiness during the desired wake period and an inability to sleep during the desired sleep period, both due to changing shift work or night shift work.

Diagnosis of any type of circadian rhythm sleep disorder must be distinguished from normal adjustments a person makes in reaction to a schedule change. The sleep disruptions must be persistent and recurring and lead to social or occupational problems. People who prefer unusually late or early sleep schedules or people adjusting to a new sleep schedule should not receive this diagnosis unless they meet the other criteria.

Treatments

Treatment of the delayed sleep phase type depends on the severity of the case. Mild cases may be addressed by an individual simply adhering to strict sleep and wake times. Severe cases may require incremental changes in sleep time, where a person sleeps 15 to 30 minutes earlier each day until an appropriate pattern is reached. Other methods of altering delayed sleep patterns include prescribing a night of sleep deprivation or the use of chronotherapy, a method in which sleep is delayed for three hours each night until the sleep pattern is rotated around the clock.

Often, treatment is ignored for persons with the jet lag type because people eventually return to their regular time zone and normal sleep-wake cycle and no longer exhibit symptoms. For people who travel often, it is preferable to adjust to the new time zone by sleeping at times appropriate to that zone if they intend to be there for one week or longer. **Diets** that target jet lag are also effective for some people, and **light therapy**, which involves exposure to a lighted device to simulate daytime, may be helpful to some people to adjust to new time zones.

People with the shift work type of the disorder benefit most from a non-changing work schedule. If rotating or changing shifts are unavoidable, rotations that occur in a clockwise direction, where shifts get progressively later and later, are preferable to those in a counter-clockwise direction. Also, when attempting to sleep, it is a good idea to create a comfortable sleeping environment by eliminating daytime noise and light.

Prognosis

Individuals with delayed sleep phase type often have great difficulty changing their sleep patterns and when they are able to change their circadian cycle, they have difficulty maintaining the changes.

People with jet lag type or shift work type can reduce symptoms often by simply decreasing the amount of travel or returning to a normal work schedule. When these changes are not possible, these individuals have trouble making the constant adjustments required to sleep and wake. People with the shift work type often report a reversal of symptoms two weeks after returning to a normal work and sleep schedule.

Prevention

Because circadian rhythm sleep disorder is usually related to environmental stressors, avoidance of these stressors (such as long-distance travel, shift work, and sleep-disrupting lifestyles) can prevent the disorder from beginning or continuing. People who are able to adhere strictly to a normal sleep-wake schedule can also offset circadian rhythm-related problems.

See also Breathing-related sleep disorder; Sleep disorders

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ORGANIZATIONS

American Sleep Disorders Association. 6301 Bandel Road NW, Suite 101, Rochester, MN 55901. <http://www.asda.org>.

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Citalopram

Definition

Citalopram is a selective serotonin reuptake inhibitor (SSRI) antidepressant drug that is sold in the United States under brand name Celexa.

Purpose

Citalopram is approved by the United States Food and Drug Administration (FDA) for the treatment of depression. It appears to be very effective in the treatment of **panic disorder** and is being evaluated for the treatment of **obsessive-compulsive disorder**, alcohol abuse, headache, **post-traumatic stress disorder**, and premenstrual syndrome.

Description

Serotonin is a **brain** chemical that carries nerve impulses from one nerve cell to another. Researchers think that depression and certain other mental disorders may be caused, in part, because there is not enough serotonin being released and transmitted in the brain. Like the other SSRI antidepressants, **fluoxetine** (Prozac), **sertraline** (Zoloft), and **paroxetine** (Paxil), citalopram increases the level of brain serotonin (also known as 5-HT). Increased serotonin levels in the brain may be beneficial in patients with obsessive-compulsive dirder, alcoholism, certain types of headaches, post-traumatic stress disorder (PTSD), premenstrual tension and mood swings, and panic disorder.

Citalopram is available in 20-mg, 40-mg, and 60-mg tablets.

Recommended dosage

The daily dosage of citalopram for depression ranges from 20–60 mg. The initial dosage is usually 20 mg per day. This dosage may then be increased to 40 mg per day at an interval of no less than one week. Most patients experience relief from depression at this dosage and do not require more than 40 mg per day. The dosage is taken once daily, either in the morning or in the evening.

Patients who are being treated for panic disorder receive doses ranging from 20–60 mg daily. A dosage of 20–30 mg daily appears to be optimal for the treatment of most panic disorders.

KEY TERMS

Obsessive-compulsive disorder—Disorder in which the affected individual has an obsession (such as a fear of contamination, or thoughts he or she doesn't like to have and can't control) and feels compelled to perform a certain act to neutralize the obsession (such as repeated handwashing).

Panic disorder—An anxiety disorder in which an individual experiences sudden, debilitating attacks of intense fear.

Post-traumatic stress disorder—A disorder caused by an extremely stressful or traumatic event (such as rape, act of war, or natural disaster) in which the trauma victim is haunted by flashbacks. In the flashbacks, the event is re-experienced in the present. Other symptoms include nightmares and feelings of anxiety.

Premenstrual syndrome—A severe change in mood that occurs in women immediately prior to, and during, their menstrual period.

Precautions

Patients who are allergic to citalopram, any other SSRI drug, or any component of the preparation should not take citalopram.

Patients with liver problems and elderly patients (over age 65) need to take smaller amounts of the drug. Dosage for these patients should start at 20 mg but can be increased to 40 mg daily if needed. Patients with kidney problems do not need dosage adjustments. Patients with history of mania, **suicide** attempts, or seizure disorders should start citalopram with caution and only under close physician supervision. There is no clinical data available on the use of citalopram in children and adolescents.

Side effects

More than 15% of patients develop **insomnia** while taking citalopram. Nausea and dry mouth occur in about 20% patients being treated with citalopram. Patients also experience tremor, anxiety, agitation, yawning, headaches, dizziness, restlessness, and sedation with citalopram therapy. These side effects usually diminish or disappear with continued use of the drug, although it may take up to four weeks for this to occur.

A drop in blood pressure and increased heart rate have been associated with citalopram use. In general,

patients do not experience weight gain or loss after starting citalopram.

Sexual dysfunction, which includes decreased sex drive in women and difficulty ejaculating in men, is also associated with the use of citalopram. In some patients, it may take up to 12 weeks for these side effects to disappear. In some patients these sexual side effects never resolve. If sexual side effects continue, the dose of citalopram may be reduced, patients can also have drug holidays where the weekend dose is either decreased or skipped, or they can discuss with their physician the risks and benefits of switching to another antidepressant.

Interactions

Citlopram interacts with a long list of other medications. Anyone starting this drug should review the other medications they are taking with their physician and pharmacist for possible interactions. Patients should always inform all their health care providers, including dentists, that they are taking citalopram.

Certain antifungal medications such as itraconazole, fluconazole, ketoconazole, as well as the antibiotic erythromycin, can increase the levels of citalopram in the body. This can cause increased side effects. Levomethadyl, a medication used to treat opioid dependence, may cause toxicity to the heart if used together with citalopram.

Serious side effects called serotonin syndrome have resulted from the combination of antidepressants such as citalopram and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Serotonin syndrome usually consists of at least three of the following symptoms: diarrhea, fever, sweatiness, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking. Because of this, citalopram should never be taken in combination with MAO inhibitors. MAO inhibitors include isocarboxazid, nialamide, pargyline, selegiline, **phenelzine**, procarbazine, iproniazid, and clorgyline. Patient taking any MAO inhibitors, should stop the MAO inhibitor then wait at least 14 days before starting citalopram or any other antidepressant. The same holds true when discontinuing citalopram and starting an MAO inhibitor.

Buspirone, an anti-anxiety medication, should not be used together with citalopram. **Ginkgo biloba** and **St. John's Wort**, herbal supplements that are common in the United States, should not be taken together with citalopram.

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Client-centered therapy *see* **Person-centered therapy**

Clinical Assessment Scales for the Elderly

Definition

The Clinical Assessment Scales for the Elderly, often abbreviated as CASE, is a diagnostic tool used to determine the presence of mental disorders and other conditions in elderly adults.

Purpose

The CASE is used to determine the presence of mental disorders in an elderly person as defined by the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (2000), which is also called *DSM-IV-TR*. The *DSM-IV-TR* is the basic reference work consulted by mental health professionals when making a **diagnosis**. The CASE, which is used with adults between the ages of 55 and 90, consists of a self-report form in which the person answers questions about himself or herself related to various scales. If the elderly adult is unable to complete the form because of cognitive or physical deficiencies, an other-rating form is provided for use by a knowledgeable caregiver, such as a spouse, child, or health care worker.

The CASE is not always used specifically for diagnosing mental disorders. It may be administered simply as a general assessment tool to gain insight about an elderly person. It may serve as a neurological screening tool to rule out other problems. The test makers also claim that it can be used as an early screening tool for **dementia** and thus allow elderly adults to receive medications to slow the progress of **Alzheimer's disease**.

KEY TERMS

Alzheimer's disease—An incurable dementia marked by the loss of cognitive ability and memory over a period of 10–15 years. Usually affects elderly people.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Reliability—The ability of a test to yield consistent, repeatable results.

Standardization—The administration of a test to a sample group of people for the purpose of establishing test norms.

Validity—The ability of a test to measure accurately what it claims to measure.

Description

The Clinical Assessment Scales for the Elderly were written by Cecil Reynolds and Erin Bigler. The most recent version of the test was published in 2001. The CASE consists of 10 clinical scales that measure the following: Anxiety; Cognitive Competence; Depression; Fear of Aging; Obsessive-Compulsiveness; **Paranoia**; Psychoticism; Somatization; Mania; and Substance Abuse. The degree to which an elderly person exhibits symptoms in these areas can help a mental health professional with the process of differential diagnosis for a mental disorder.

The CASE also includes three validity scales. These are helpful in evaluating the consistency of a person's responses and whether the person is faking his or her answers.

The person who is completing the CASE, whether they are using the self-rating or the other-rating form, responds to the test's written items. The test usually takes between 20–40 minutes to finish, but it is not timed. People are generally given as much time as they need to complete it.

A shorter version of the test, called the Clinical Assessment Scales for the Elderly-Short Form (CASE-SF) is also available. The CASE-SF takes about 20 minutes to complete and includes all 10 of the clinical scales.

Results

Scoring for the CASE is relatively simple. Scores are calculated for each scale and then compared to age-appropriate scores to determine the presence or severity of symptoms. For example, if a person scores high on the Depression scale, this information could be used as part of an overall diagnosis for a *DSM-IV* depressive disorder. A person scoring high in Psychoticism may have a psychotic disorder. For any specific *DSM-IV* diagnosis to be made, however, all of the required criteria for that disorder must be met. The results from the CASE may satisfy only some of the requirements.

The Fear of Aging scale assesses the person's degree of apprehension or concern about the aging process. It is not necessarily related to a particular *DSM-IV* disorder. Information about a person's fear of aging, however, may be helpful during the diagnostic process. It may also be useful information for a psychotherapist or other counselor, to understand the patient's concerns or to measure progress in therapy.

The CASE was standardized using a sample of 2000 adults in the United States, 1000 for each of the two test forms. The test has been shown to have good reliability and validity. For example, scores from the CASE Depression scale have been shown to correlate very well with scores on the widely used **Beck Depression Inventory**, or BDI.

See also Figure drawings; House-Tree-Person Test

Resources

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Ali Fahmy, Ph.D.

Clomipramine

Definition

Clomipramine is an antidepressant drug used primarily to alleviate obsessions and compulsions in patients with **obsessive-compulsive disorder**. Clomipramine is also used in the treatment of depressive disorders and in

a number of other psychiatric and medical conditions. In the United States, the drug has also been known by the brand name Anafranil.

Purpose

Clomipramine is principally used in the treatment of the obsessions and compulsions of obsessive-compulsive disorder (OCD), when these symptoms greatly disrupt the patient's daily activities. Obsessions are repetitive thoughts and impulses, and compulsions are repetitive behaviors. Patients with OCD find these experiences inappropriate, distressing, and time-consuming.

Clomipramine may also be used in the treatment of depressive disorders, especially when associated with obsessions and compulsions, in **panic disorder**, pain management, sleep attacks (**narcolepsy** and cataplexy), and **anorexia nervosa**. The drug may help to reduce compulsive behaviors in a variety of disorders with such symptoms, including **trichotillomania** (hair-pulling), onychophagia (nail-biting), Tourette's disorder (tics and vocalizations), and childhood **autism**.

Description

Clomipramine is one of the tricyclic antidepressants, so-called because of the three-ring chemical structure common to these drugs. In the 1940s and 1950s, pharmaceutical researchers synthesized a number of new compounds for possible medical use as antihistamines and sedatives. After testing in animal experiments, a few of these substances were selected for human study. One potential drug, a tricyclic compound called **imipramine**, was not useful in calming agitation, but it had a striking effect in improving the mood of certain patients with depression.

Since the discovery of imipramine, many other tricyclic antidepressants have been developed with somewhat differing pharmacological activities and side effect profiles. Within this group of drugs, clomipramine is exceptionally potent in affecting levels of serotonin in the **brain**. In this action, it is similar to serotonin-selective antidepressant drugs, like **fluoxetine** (Prozac), which act specifically on serotonin levels and are effective in OCD. Serotonin is a messenger chemical (neurotransmitter) involved in transmitting signals between nerve cells. Clomipramine reduces the effects on serotonin neurotransmission in depression and OCD symptoms.

Recommended dosage

For adults, clomipramine is administered in dosages up to a maximum of 250 mg per day. Starting with a dose of 25 mg, the dosage is increased during the first two weeks to 100 mg per day. If needed, it is further increased gradually over the next several weeks. The initial dose is low to avoid side effects, and it is increased slowly to permit the patient to develop tolerance or adapt to side effects that may occur.

Older patients (over age 65), children, and adolescents are more sensitive to the side effects and toxicities of tricyclic antidepressants such as clomipramine. The maximum daily dose is usually lower for elderly patients than younger adults. For children and adolescents, the maximum recommended daily dose is the lesser of 100 mg or 3 mg per kg of body weight.

KEY TERMS

Autonomic—The part of the nervous system that governs the heart, involuntary muscles, and glands.

Cataplexy—A symptom of narcolepsy marked by a sudden episode of muscle weakness triggered by strong emotions. The muscle weakness may cause the person's knees to buckle, or the head to drop. In severe cases, the patient may become paralyzed for a few seconds to minutes.

Epilepsy—A neurological disorder characterized by the onset of seizures. Seizures are caused by a disturbance in the electrical activity in the brain and can cause loss of consciousness, muscle spasms, rhythmic movements, abnormal sensory experiences, or altered mental states.

Kilogram—A metric unit of weight. It equals 2.2 lbs.

Monoamine oxidase (MAO) inhibitors—A group of antidepressant drugs that decreases the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Precautions

Epileptic **seizures** are the most important risk associated with clomipramine. Among patients taking the drug for six months or more, more than 1% may experience seizures. The risk of seizure increases with larger doses, and seizures have been reported to occur following abrupt discontinuation of the medication. Caution and physician supervision is required if the patient has a history of epilepsy or some other condition associated with seizures, such as brain damage or alcoholism.

Clomipramine and other tricyclic antidepressants often cause drowsiness. Activities requiring alertness, such as driving, should be avoided until patients understand how the drug affects them. Dizziness or lightheadedness may occur on arising from a seated position, due to sudden decreases in blood pressure. Fainting may also occur. Some patients, especially men with prostate enlargement, may experience difficulty urinating. Glaucoma may be worsened. Sensitivity to ultraviolet light may increase, and sunburns may occur more easily.

Tricyclic antidepressants, including clomipramine, should be used with caution and physician supervision in patients with heart disease, because of the possibility of adverse effects on heart rhythm. Adverse effects on the heart occur frequently when tricyclics are taken in overdose. Only small quantities of these drugs should be given to patients who may be suicidal.

Tricyclic antidepressants may cause dry mouth, due to decreased saliva, possibly contributing to the development of tooth decay, gum disease, and mouth infections. Patients should avoid sweets, sugary beverages, and chewing gum containing sugar.

It has not been determined whether clomipramine is safe to take during pregnancy, and the patient's need for this medicine should be balanced against the possibility of harm to the fetus. Tricyclic antidepressants may be secreted in breast milk and may cause sedation and depress breathing of a nursing infant.

Side effects

Clomipramine may cause many side effects. Initially, the side effects of tricyclic drugs may be more pronounced, but sensitivity often decreases with continued treatment.

The following more common side effects are grouped by the body system affected:

- Cardiovascular: decreases of blood pressure on arising, which may cause dizziness or fainting, increases of blood pressure, rapid heart rate, pounding heart, altered heart rhythm.
- Nervous system: sedation, dizziness, headache, confusion, nervousness, restlessness, sleep difficulties, numbness, tingling sensations, tremors, twitches, increased seizure tendency.
- Digestive system: dry mouth, nausea, loss of appetite, indigestion, and constipation.
- Autonomic: blurred vision, increased sweating.
- Genital/urinary: difficulty urinating, menstrual pain, ejaculatory difficulty, impotence, decreased sex drive.
- Skin: rashes, sensitivity to sunlight.
- Body as a whole: **fatigue**, weight gain, flushing.

Less commonly, tricyclic drugs may cause adverse effects on almost any organ or system of the body, particularly the blood, hormones, kidney, and liver. Patients should consult their physicians if symptoms develop or bodily changes appear.

Interactions

Tricyclic antidepressants, such as clomipramine, may interact with many other drugs. Patients should inform their physicians about all other drugs they are taking before starting treatment.

Clomipramine may intensify the effects of other drugs that act on serotonin levels, possibly producing serotonin syndrome, a rare but dangerous condition with fever, sweating, tremors, and changes in mental state. Drugs that may interact this way include other antidepressants, especially selective serotonin re-uptake inhibitor (SSRI) drugs and monoamine oxidase (MAO) inhibitors. These drugs should not be taken within two weeks of taking clomipramine. Other drugs to avoid include lithium, **alprazolam** (Xanax), fenfluramine (Pondimin), amphetamine, dextromethorphan (used in cough suppressants), meperidine (Demerol), and tramadol (Ultram).

Tricyclic drugs may intensify the effects of other drugs causing sedation, including alcohol, **barbiturates**, narcotic pain medications, minor tranquilizers, and antihistamines. Tricyclics may cause excessive reductions of blood pressure in patients taking blood pressure medicine, especially on arising or standing up. Conversely, these drugs may interfere with the pressure-reducing effects of certain other blood pressure medicines and may necessitate an adjustment in dosage. Tricyclics may interact with thyroid medications to produce abnormalities of heart rhythm. Concurrent use of tricyclic antidepressants with other psychiatric medicines may result in intensification of certain side effects.

Certain drugs may interfere with the elimination of tricyclic antidepressants from the body, causing higher blood levels and increased side effects. This effect may occur with cimetidine (Tagamet), other antidepressants, **methylphenidate** (Ritalin, Concerta), and some antipsychotic medications.

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Richard Kapit, M.D.

Clonazepam

Definition

Clonazepam belongs to a group of drugs called benzodiazepines. Benzodiazepines are medications that help relieve nervousness, tension, symptoms of anxiety, and some types of **seizures** by slowing the central nervous system. In the United States, clonazepam is sold under brand name Klonopin.

Purpose

Although clonazepam is approved by the United States Food and Drug Administration (FDA) for the treatment of **panic disorder** and some types of epilepsy, it is also used to treat **social phobia**, mania, and **post-traumatic stress disorder**.

KEY TERMS

Anorexia—Loss of appetite or unwillingness to eat. Can be caused by medications, depression, or many other factors.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Description

Clonazepam belongs to a group of drugs called benzodiazepines. Benzodiazepines are sedative-hypnotic drugs that help to relieve nervousness, tension, anxiety symptoms, and seizures by slowing the central nervous system. To do this, they block the effects of a specific chemical involved in the transmission of nerve impulses in the **brain**, decreasing the excitement level of the nerve cells.

When clonazepam is used to treat panic disorder, it is more sedating than **alprazolam**, another benzodiazepine drug used to treat panic disorder. However, unlike alprazolam, clonazepam may trigger depressive episodes in patients with a previous history of depression. In people who experience social phobia, treatment with clonazepam reduces the rate of depression. The use of clonazepam for social phobia is considered off-label use—a use that is legal, but not specifically approved by the FDA.

Clonazepam comes in 0.5 mg-, 1 mg-, and 2 mg-tablets.

Recommended dosage

For panic disorder, the initial recommended dose is 0.25 mg twice daily. This dose can be increased every three days in increments of 0.125–0.25 mg twice daily. The target dose for panic disorder is 1.0 mg per day, although some people benefit from doses up to a maximum of 4 mg per day. When a person stops taking clonazepam, the drug should be gradually discontinued by decreasing the dose by 0.125 mg twice daily every three days.

Although clonazepam is not FDA-approved for the treatment of post-traumatic stress disorder, doses in the range of 0.25–3 mg daily appears to help treat symptoms of this disorder. Daily dosages for the treatment of social

phobia range from 1.0–2.5 mg, while the dosage to control mania may be as high as 10 mg daily.

Precautions

Women who are pregnant should not use clonazepam, because it may harm the developing fetus. Clonazepam should never be taken by people who have had an allergic reaction to it or another benzodiazepine drug such as **diazepam** (Valium). People with narrow-angle glaucoma or severe liver disease should not take clonazepam. People who have kidney disease may need to take a reduced dosage of the drug. Saliva production may increase while taking clonazepam. Because of this, people with respiratory disease or an impaired gag reflex should use clonazepam with close physician supervision.

Because clonazepam is a nervous system depressant, it should not be taken with other such depressants, such as alcohol, other sedatives, sleeping pills, or tranquilizers. People taking clonazepam may feel unusually drowsy and mentally sluggish when they first start taking the drug. They should not drive, operate dangerous machinery, or engage in hazardous activities that require mental alertness until they see how clonazepam affects them. This excessive sedation usually goes away after a short time on the drug.

People who have underlying depression should be closely monitored while taking clonazepam, especially if they are at risk for attempting **suicide**.

Side effects

The main side effects of clonazepam are sedation, dizziness, impaired coordination, depression, and **fatigue**. Some people experience decreased sex drive while taking clonazepam.

A small number of people develop sinus problems and upper respiratory tract infections while taking clonazepam. One of the side effects of clonazepam may be increased salivation. This may cause some people to start coughing while taking clonazepam. Clonazepam may also cause anorexia and dry mouth. It may cause either constipation or diarrhea. There are a few reports of clonazepam causing menstrual irregularities or blurred vision.

Interactions

Clonazepam may increase the sedative effects of other drugs that depress the central nervous system such as certain pain strong medicines (opiates such as codeine, oxycodone, hydromorphone) and antihistamines (found in many cold and allergy medications). The sedative effect is also increased if clonazepam is taken with alcohol.

Disulfiram (Antabuse), a medication used to treat alcohol dependence, increases the effect of clonazepam. Medications that make clonazepam ineffective include phenobarbital, phenytoin, **carbamazepine**, theophylline, rifampin, and rifabutin.

Resources

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Clonidine

Definition

Clonidine belongs to a class of drugs called central alpha-adrenergic agonists. In the United States, clonidine tablets are sold under the brand name Catapres and clonidine skin patches are sold under the brand name Catapres-TTS. The tablets are also available generically. There is also an injectable form that is administered directly into the spinal cord for the treatment of postoperative pain.

Purpose

Clonidine tablets and patches are approved by the United States Food and Drug Administration (FDA) for the treatment of high blood pressure. However, clonidine has been found to be useful in the treatment of alcohol, opiate, and nicotine withdrawal syndromes, **attention-deficit/hyperactivity disorder** (ADHD), and Tourette's syndrome, one of the **tic disorders**.

Description

Clonidine was synthesized in 1960s and was initially tested as a nasal decongestant. In the United States, clonidine was first used to treat hypertension although it has also been investigated for treatment of different neuropsychiatric disorders. Clonidine works on specific nerve cells in the **brain** that are responsible for lowering blood pressure, slowing heart rate, and decreasing the body's reaction to the withdrawal of chemicals like alco-

hol, opiates, cocaine, and nicotine. Because of this, clonidine is often used to treat the symptoms of drug, alcohol, and nicotine withdrawal.

Clonidine is beneficial in opiate withdrawal because it treats symptoms that are commonly associated with that condition (watery eyes and nose, diarrhea, irritability). For this condition, clonidine is often used alone. For the treatment of alcohol withdrawal, clonidine is usually combined with benzodiazepine tranquilizers such as Librium, Valium, Xanax, or Ativan.

Several studies of treatment for smoking cessation showed patients treated with clonidine had decreased nicotine craving. Clonidine skin patches appear to be more effective than tablets in this condition. Both dermal patches and tablets are effective in the treatment of Tourette's syndrome and ADHD.

Clonidine tablets are available in 0.1-mg, 0.2-mg, and 0.3-mg strengths. Clonidine skin patches are available in 0.1-mg, 0.2-mg, and 0.3-mg per day patches. Each patch lasts seven days.

Recommended dosage

Dosages of 0.4–0.6 mg have been used for the treatment of alcohol withdrawal. Total daily dosage for the treatment of opiate withdrawal range between 0.5 and 1.4 mg, depending on the stage as well as the severity of withdrawal symptoms. If the clonidine patch is used to treat nicotine withdrawal symptoms, dosages that deliver 0.1–.2 mg daily are used. For oral therapy (tablets), a total dosage of 0.2–0.4 mg daily is taken in divided doses.

Pediatric doses of clonidine are calculated based on the child's body weight. Clonidine dosage for ADHD in children is 5 micrograms per kilogram of body weight per day orally in four divided doses. Children who require a daily dosage of 0.2 mg usually can use the 0.3 mg dermal patch. If ADHD is associated with sleep disturbances, low to moderate doses of clonidine can be taken at bedtime. Oral doses in children with Tourette's syndrome range from 3 to 6 micrograms per kilogram of body weight per day divided into two to four even doses.

Precautions

Clonidine should not be used by people who have a known allergy to this drug. If a person has underlying depression, clonidine should be used with caution and under close physician supervision.

Clonidine should not be abruptly withdrawn but rather, slowly decreased over several days to avoid withdrawal symptoms. Withdrawal symptoms include increases in blood pressure, irritability, nervousness, **insomnia**, and headache. Because of the possibility of withdrawal, clonidine should not be used in patients who are unwilling or unable to follow the prescribing information.

Clonidine should be used only with caution and close physician supervision in patients with chronic renal failure, coronary artery disease, and in patients with preexisting eye problems. Often people with kidney disease should take a reduced dosage. Clonidine should not be used by pregnant women, except in the rare case where the benefits of taking clonidine outweigh the risks to the developing fetus.

Side effects

The most common side effect associated with clonidine is dizziness associated with sudden changes in position such as standing up rapidly. In order to avoid this, patients should stand up slowly. People using the dermal patch may develop rash, hair loss, a burning sensation on the skin, or other skin irritations where the patch is applied. Switching to tablets may not completely eliminate these skin problems, however.

Clonidine can cause dry mouth, constipation, nausea, daytime sleepiness, weakness, and lethargy. These side effects may take several weeks to disappear. In some cases, these side effects can be eliminated with dosage readjustment. In addition, clonidine may cause eye dryness, loss of sex drive, and decreased sexual activity.

If patients experience weight gain in the beginning of therapy, they can expect this side effect to decline over a period of several days to weeks.

KEY TERMS

Attention-deficit/hyperactivity disorder (ADHD)—A learning and behavioral disorder characterized by difficulty in sustaining attention, impulsive behavior, and excessive activity.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Tourette syndrome—Neurological disorder characterized by multiple involuntary movements and uncontrollable vocalizations called tics that come and go over years, usually beginning in childhood and becoming chronic. Sometimes the tics include inappropriate language.

Interactions

Clonidine's blood pressure-lowering effects may be enhanced by other drugs that lower blood pressure. Conversely, the blood pressure-lowering effects of clonidine may be negated by many antidepressants.

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Clorazepate

Definition

Clorazepate is a medication that belongs to a family of drugs called benzodiazepines—a group of pharmacologically active compounds used to produce a calming effect by relieving anxiety and tension. In the United States, clorazepate is sold under brand names Tranxene and Gen-XENE.

Purpose

Clorazepate is used for the treatment of anxiety and alcohol withdrawal. Moreover, clorazepate is an adjunct in the management of partial **seizures**.

Description

Clorazepate binds to different sites in the **brain**, causing them to shift into a state that is less excitable. It is very effective in treating **anxiety and anxiety disorders**. Moreover, anxiety associated with undergoing surgical procedures is controlled with clorazepate. Clorazepate alone is not efficacious in treating seizures; however, if used along with other standard seizure medications, such as phenobarbital, primidone, phenytoin, **carbamazepine**, and **valproic acid**, better seizure control may be achieved. Convulsions and anxiety associated with alcohol withdrawal are controlled with clorazepate.

KEY TERMS

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Clorazepate is available in two different formulations. Clorazepate tablets come in 3.75-, 7.5-, and 15-mg doses, while slow-release tablets, administered once daily, are available in 11.25- and 22.5-mg strengths. Capsules are available in 3.75-, 7.5-, and 15-mg strengths.

Recommended dosage

If used for anxiety, the dose of clorazepate usually ranges anywhere from 15 mg to 60 mg daily in divided dose intervals. Usually, however, the average dose is 30 mg daily given in two to four doses. If slow-release formulation is used, the dose of either 11.25 mg or 22.5 mg is usually administered at bedtime. Slow-release products should not be used to initiate therapy.

Doses of clorazepate for the management of seizures differ in adult and pediatric populations. Patients who are nine to 12 years of age should be started on 3.75–7.5 mg twice daily. This dose should be increased by no more than 3.75 mg weekly. The maximum dose per day is 60 mg administered in two to three divided doses. Children older than 12 and adults should receive 7.5 mg two to three times daily. This can be increased to a higher dose by adding 7.5 mg at weekly intervals. The total daily dose should not exceed 90 mg daily administered in two to three doses. In patients undergoing alcohol withdrawal, the first dose is 30 mg. Treatment is continued with 15 mg two to four times daily for the maximum dose of 90 mg in one day. Once maximum dose is achieved, the dose is gradually decreased over subsequent days.

Precautions

Pregnant women should not take clorazepate. Patients who have narrow-angle glaucoma should not take clorazepate, as this may worsen their condition. Clorazepate should not be used in patients younger than nine years of age.

If depression coexists with anxiety, clorazepate should be used with caution as suicidal tendencies may be present. (One of the side effects with this medication is depression; if a patient has an underlying problem with depression, that problem can be exacerbated with clorazepate.) Patients should be cautioned against engaging in hazardous occupations requiring mental alertness, since clorazepate causes drowsiness and dizziness. Abrupt discontinuation of clorazepate has been associated with withdrawal symptoms and seizures. Hence, doses of clorazepate should be slowly decreased in patients who have been taking clorazepate continuously over several weeks. Other withdrawal symptoms may include nervousness, **insomnia**, irritability, diarrhea, and muscle aches. The doses for elderly patients, as well as patients with liver or kidney problems, may need to be decreased.

Side effects

The most common side effects include drowsiness, dizziness, and confusion. There are a few reports about behavioral changes associated with the use of clorazepate and they include rage, depression, irritability, and aggression.

Other side effects include vision disturbances—such as blurred and double vision—decreased libido, nausea, vomiting, either decreased or increased appetite, and diarrhea or constipation. In a few cases, clorazepate has been associated with liver toxicity where patients developed jaundice or fever. It is also known to cause a rash.

Interactions

Simultaneous use of clorazepate and dong quai, a Chinese herb, has been associated with excessive muscle relaxation and central nervous system depression. Other herbs that should not be used with clorazepate include **ginkgo biloba** and **kava kava**.

Omeprazole, a medication used to treat heartburn, should not be used together with clorazepate. Medicines to treat disorders associated with increased acid secretions—such as ranitidine, sucralfate, and pantoprazole—are not contraindicated with clorazepate. **Valerian**, an herb used as a sleep aid, binds to the same receptors in the brain as clorazepate; thus, the desired effects of clorazepate may not be seen in patients taking it and valerian at the same time.

Clorazepate may increase the effects of other drugs that cause drowsiness,. These drugs include antihistamines (such as Benadryl), sedatives (usually used to treat insomnia), pain relievers, anxiety and seizure medicines, and muscle relaxants. Alcohol combined with clorazepate also causes excessive drowsiness.

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Clozapine

Definition

Clozapine is an antipsychotic drug used to alleviate the symptoms and signs of schizophrenia—a form of severe mental illness— which is characterized by loss of contact with reality, **hallucinations**, **delusions**, and unusual behavior. In the United States, the drug is also known by the brand name Clozaril.

Purpose

Clozapine is principally used to reduce the signs and symptoms of severe schizophrenic illness. The drug is intended for use in patients with severe **schizophrenia** who have not responded to any other antipsychotic drug. Clozapine is also used in patients with severe schizophrenia when other antipsychotic medications have caused intolerable side effects.

Description

Clozapine is considered an atypical antipsychotic drug. Atypical antipsychotics differ from typical antipsychotics in their effectiveness in schizophrenia and their profile of side effects. Clozapine may reduce the signs and symptoms of schizophrenia in a large proportion of treatment-resistant schizophrenic patients who do not respond to typical antipsychotics. Moreover, the drug is less likely than typical antipsychotics to cause **tardive dyskinesia** and other extrapyramidal side effects. Tardive dyskinesia is a syndrome of involuntary, uncoordinated movements that may not disappear or may only partially improve after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage. It

KEY TERMS

Agranulocytosis—A blood disorder characterized by a reduction in the number of circulating white blood cells (granulocytes). White blood cells defend the body against infections. Agranulocytosis is a potential side effect of some of the newer antipsychotic medications used to treat schizophrenia.

Antipsychotic drug—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Autonomic—The part of the nervous system that governs the heart, involuntary muscles, and glands.

Epilepsy—A neurological disorder characterized by the onset of seizures. Seizures are caused by a disturbance in the electrical activity in the brain and can cause loss of consciousness, muscle spasms, rhythmic movements, abnormal sensory experiences, or altered mental states.

Extrapyramidal side effects—A group of neurological side effects including muscle spasms, involuntary movements, and symptoms that resemble Parkinson's disease (also called drug-induced Parkinsonism).

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

may also appear after use of the antipsychotic has stopped. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for this syndrome, although gradual (but rarely complete) improvement may occur over a long period.

Clozapine was the first atypical antipsychotic drug to be developed. In the late 1980s, clozapine was tested in severely ill schizophrenic patients who had been treated with a typical antipsychotic drug but had not shown much improvement. A significant proportion of these patients improved as a result of treatment with clozapine.

The superiority of clozapine in treatment-resistant patients is considered an important advance, but the drug is not without problems. Clozapine is generally considered the most toxic of the antipsychotic drugs. It causes agranulocytosis, a life-threatening depletion of white blood cells, in 1-2% of patients. It also causes epileptic **seizures** and adverse effects on the heart and blood pressure more frequently than other antipsychotic medicines. Clozapine is usually reserved for the most severely ill schizophrenic patients who have not responded to other treatments. Other atypical antipsychotic drugs have been developed in recent years, and they are considered safer to use than clozapine.

The mechanisms of action of antipsychotic drugs are not completely understood. The effect of clozapine is believed to be related to its actions in blocking neurotransmission due to the **neurotransmitters** dopamine and serotonin in a region of the **brain** called the limbic system, which is involved with emotions and motivation. The actions of clozapine may target the limbic system more specifically than those of typical antipsychotic drugs.

Clozapine is available as Clozaril, the only brand, as 25- and 100-mg tablets.

Recommended dosage

The usual dosage of clozapine is 300–600 mg per day; however, some patients may require daily dosages of up to 900 mg. To minimize side effects, the initial dose of clozapine is 12.5 mg (one-half tablet) twice a day, and the dose is increased by 25–50 mg each day, until the dose reaches 300–450 mg per day. The daily dosage of the drug is then determined based on the individual patient's response, but increases should not exceed 100 mg once or twice a week.

Precautions

Clozapine may cause agranulocytosis, a life-threatening depletion of white blood cells. The blood cells affected by clozapine defend the body against infections by bacteria and other microorganisms, and patients with agranulocytosis are subject to severe infections. Clozapine treatment is reserved for the most severely ill schizophrenic patients who have not responded to other treatments. Clozapine is available only through a distribution system that assures close monitoring of white blood cells. Patients must have white blood cell counts determined before starting treatment, once every week for the first six months, once every other week after that,

and once a week for the first month after clozapine treatment is stopped.

Clozapine may cause epileptic seizures in about 5% of patients. The frequency of seizures goes up as the dose of the drug is increased. Patients who experience seizures on clozapine should usually have the drug discontinued or the dose reduced. Neuroleptic malignant syndrome (NMS), a dangerous condition with high fever, muscular rigidity, rapid pulse, sweating, and altered mental state, may occur with all antipsychotic medications, including clozapine. NMS requires immediate medical treatment.

Clozapine frequently causes sedation and may interfere with driving and other tasks requiring alertness. The drug may increase the effects of alcohol and sedatives. Clozapine may cause low blood pressure and sudden drops in blood pressure on standing up, which may cause dizziness or fainting. Elevated heart rate may occur in 25% of patients; this effect may be a serious risk for patients with heart disease. Clozapine-induced fever, unrelated to any illness, may occur. The fever usually subsides within a few days, but it may require stopping the drug.

The safety and effectiveness of clozapine in children under 16 years old have not been established. Elderly patients may be particularly sensitive to sedation, low blood pressure, and other side effects. The drug should be used with caution in older patients. Clozapine should be used in pregnant women only when strictly necessary. The drug has not been adequately studied in pregnancy. In animal studies, however, clozapine has not produced harmful effects on the fetus. Clozapine may be secreted in breast milk, and breast-feeding may not be advisable.

Side effects

Clozapine may cause many side effects. The following side effects are grouped by the body system affected:

- Cardiovascular: decreases of blood pressure, especially on arising from a seated or lying position, which may cause dizziness or fainting; rapid heart rate, changes in heart rhythm and electrocardiogram.
- Nervous system: sedation, increased seizure tendency.
- Digestive system: increased appetite, excessive salivation, nausea, constipation, abnormal liver tests, elevated blood sugar.
- Autonomic: blurred vision, exacerbation of glaucoma, dry mouth, nasal congestion, decreased sweating; difficulty urinating, particularly in men with enlarged prostate.
- Skin: rashes.
- Body as a whole: weight gain, fever.

Interactions

Clozapine may interact with many other drugs. Patients should inform their physicians about all other drugs they are taking before starting treatment. Because of the risk of agranulocytosis, clozapine should not be given along with medications that suppress production of blood cells.

Clozapine may intensify the effects of drugs causing sedation, including alcohol, **barbiturates**, narcotic pain medications, minor tranquilizers, and antihistamines. Similarly, clozapine may cause excessive reductions of blood pressure in patients taking other medicines that lower blood pressure. Clozapine may also intensify side effects of drugs that cause blurred vision, dry mouth, diminished sweating in hot weather, and constipation. Many other antipsychotics and antidepressants cause such side effects.

Clozapine may potentiate (increase) the effects of other medications that also lower seizure threshold (make it more likely to have seizure), such as steroid drugs, the asthma medication theophylline, and many other psychiatric drugs. Patients with epilepsy may require adjustment in their dosage of anti-seizure medications. Lithium may increase the risk of seizures and other nervous system adverse effects when given with clozapine.

Certain drugs that are eliminated by the liver may interfere with the elimination of clozapine from the body, causing higher blood levels and increased side effects. Conversely, clozapine may interfere with the elimination of other drugs that are eliminated by the liver. Antidepressants that affect brain serotonin levels may increase blood levels of clozapine, possibly causing increased side effects.

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Richard Kapit, M.D.

Clozaril *see* **Clozapine**

Cocaine and related disorders

Definition

Cocaine is extracted from the coca plant, which grows in Central and South America. The substance is processed into many forms for use as an illegal drug of abuse. Cocaine is dangerously addictive, and users of the drug experience a "high"—a feeling of euphoria or intense happiness, along with hypervigilance, increased sensitivity, irritablity or anger, impaired judgment, and anxiety.

Forms of the drug

In its most common form, cocaine is a whitish crystalline powder that produces feelings of euphoria when ingested. In powder form, cocaine is known by such street names as "coke," "blow," "C," "flake," "snow" and "toot." It is most commonly inhaled or "snorted." It may also be dissolved in water and injected.

Crack is a form of cocaine that can be smoked and that produces an immediate, more intense, and more short-lived high. It comes in off-white chunks or chips called "rocks."

In addition to their stand-alone use, both cocaine and crack are often mixed with other substances. Cocaine may be mixed with methcathinone to create a "wildcat." Cigars may be hollowed out and filled with a mixture of crack and marijuana. Either cocaine or crack used in conjunction with heroin is called a "speedball." Cocaine used together with alcohol represents the most common fatal two-drug combination.

Description

Cocaine-related disorders is a very broad topic. According to the mental health clinician's handbook, ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revised (also known as the *DSM-IV-TR*), the broad category of cocaine-related disorders can be subdivided into two categories: cocaine use disorders and cocaine-induced disorders. Cocaine use disorders include cocaine dependence and cocaine abuse. Cocaine-induced disorders include:

- cocaine intoxication
- cocaine withdrawal
- cocaine intoxication **delirium**
- cocaine-induced psychotic disorder, with **delusions**
- cocaine-induced psychotic disorder, with **hallucinations**
- cocaine-induced mood disorder
- cocaine-induced anxiety disorder
- cocaine-induced sexual dysfunction
- cocaine-induced sleep disorder
- cocaine-related disorder not otherwise specified

Cocaine use disorders

COCAINE ABUSE. For the cocaine abuser, the use of the substance leads to maladaptive behavior over a 12-month period. The person may fail to meet responsibilities at school, work, or home. The cocaine abuse impairs the affected person's judgment, and he or she puts him- or herself in physical danger to use the substance. For example, the individual may use cocaine in an unsafe environment. The person who abuses cocaine may be arrested or charged with possession of the substance, yet will continue to use cocaine despite all of the personal and legal problems that may result.

COCAINE DEPENDENCE. Cocaine dependence is even more serious than cocaine abuse. Dependence is a maladaptive behavior that, over a three-month period, has caused the affected individual to experience tolerance for and withdrawal symptoms from cocaine. Tolerance is the need to increase the amount of cocaine intake to achieve the same desired effect. In other words, someone who is dependent on cocaine needs more cocaine to produce the same "high" that a lesser amount produced in the past. The dependent person also experiences cocaine withdrawal. Withdrawal symptoms develop within hours or days after cocaine use that has been heavy and prolonged and then abruptly stopped. The symptoms include irritable mood and two or more of the following symptoms: **fatigue**, nightmares, difficulty sleeping or too much sleep, elevated appetite, agitation (restlessness), or slowed physical movements. The onset of withdrawal symptoms can cause a person to use more cocaine to avoid these painful and uncomfortable symptoms. The dependent person uses larger amounts of cocaine for longer periods of time than intended. He or she cannot cut back on the use of the substance, often has a difficult time resisting cocaine

when it is available, and may abandon work or school to spend more time acquiring and planning to acquire more cocaine. The individual continues to use the cocaine despite the negative effects it has on family life, work, and school.

KEY TERMS

Amygdala—An almond-shaped brain structure in the limbic system that is activated in acute stress situations to trigger the emotion of fear.

Coca plant—The plant that is the source of cocaine.

Crack—A slang term for a form of cocaine that is smokable.

Craving—An overwhelming need to seek out more drugs.

Physical dependence—A maladaptive behavior that over a three-month period has caused the individual to experience tolerance and withdrawal symptoms.

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

Cocaine-induced disorders

COCAINE INTOXICATION. Cocaine intoxication occurs after recent cocaine use. The person experiences a feeling of intense happiness, hypervigilance, increased sensitivity, irritability or anger, with impaired judgment, and anxiety. The intoxication impairs the person's ability to function at work, school, or in social situations. Two or more of the following symptoms are present immediately after the use of the cocaine:

- enlarged pupils
- elevated heart rate
- elevated or lowered blood pressure
- chills and increased sweating
- nausea or vomiting
- weight loss
- agitation or slowed movements
- weak muscles
- chest pain
- coma
- confusion
- irregular heartbeat
- depressed respiration
- seizures
- odd postures
- odd movements

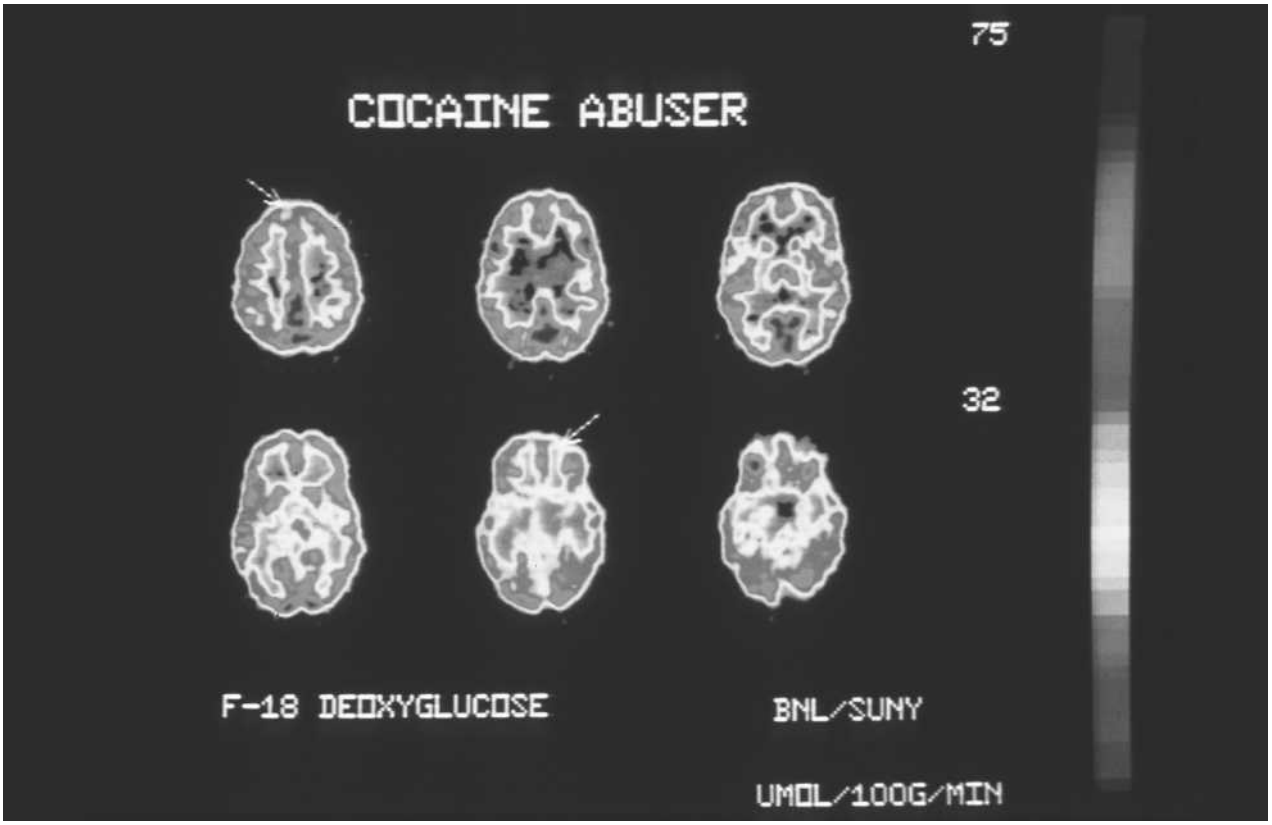
COCAINE WITHDRAWAL. As mentioned, withdrawal symptoms develop within hours or days after cocaine use that has been heavy and prolonged and then abruptly stopped. The symptoms include irritable mood and two or more of the following symptoms: fatigue, nightmares, difficulty sleeping or too much sleep, elevated appetite, agitation (restlessness), or slowed physical movements.

COCAINE-INDUCED DELIRIUM. According to the *DSM-IV-TR*, several criteria must be met in order for a health care professional to establish the **diagnosis** of cocaine-induced delirium. Patients have a disturbance of their level of consciousness or awareness, evidenced by drowsiness or an inability to concentrate or pay attention. Patients also experience a change in their cognition (ability to think) evidenced by a deficit in their language or their memory. For example, these patients may forget where they have placed an item, or their speech is confusing. These symptoms have rapid onset within hours or days of using cocaine and the symptoms fluctuate throughout the course of the day. These findings cannot be explained by **dementia** (state of impaired thought processes and memory that can be caused by various diseases and conditions) and the doctor must not be able to recognize some other physical reason that can account for the symptoms other than cocaine intoxication.

COCAINE-INDUCED PSYCHOTIC DISORDER, WITH DELUSIONS. The person suffering from this disorder has experienced intoxication or withdrawal from cocaine within a month from the time he or she begins to experience delusions (beliefs that the person continues to maintain, despite evidence to the contrary). In order for this state to be considered cocaine-induced psychotic disorder, these symptoms cannot be due to another condition or substance.

COCAINE-INDUCED PSYCHOTIC DISORDER, WITH HALLUCINATIONS. This condition is the same as cocaine-induced psychotic disorder with delusions, except that this affected individual experiences hallucinations instead of delusions. Hallucinations can be described as hearing and seeing things that are not real.

COCAINE-INDUCED MOOD DISORDER. The person suffering from this disorder has experienced intoxication or withdrawal from cocaine within a month from the time



Colored positron emission tomography (PET) brain scans of a cocaine user. *(Photo Researchers, Inc. Reproduced by permission.) See color insert for color version of photo.*

he or she begins to experience depressed, elevated, or irritable mood with **apathy** (lack of empathy for others, and lack of showing a broad range of appropriate emotions).

COCAINE-INDUCED ANXIETY DISORDER. The person suffering from this disorder has experienced intoxication or withdrawal from cocaine within a month from the time he or she begins to experience anxiety, panic attacks, obsessions, or compulsions. Panic attacks are discrete episodes of intense anxiety. Persons affected with panic attacks may experience accelerated heart rate, shaking or trembling, sweating, shortness of breath, or fear of going crazy or losing control, as well as other symptoms. An **obsession** is an unwelcome, uncontrollable, persistent idea, thought, image, or emotion that a person cannot help thinking even though it creates significant distress or anxiety. A **compulsion** is a repetitive, excessive, meaningless activity or mental exercise which a person performs in an attempt to avoid distress or worry.

COCAINE-INDUCED SEXUAL DYSFUNCTION. The person suffering from this disorder has experienced intoxication or withdrawal from cocaine within a month from the time he or she begins to experience sexual difficulties, and these difficulties are deemed by the clinician to be due directly to the cocaine use. Substance-induced sexual difficulties can range from impaired desire, impaired arousal, impaired orgasm, or sexual pain.

COCAINE-INDUCED SLEEP DISORDER. This disorder is characterized by difficulty sleeping (**insomnia**) during intoxication or increased sleep duration when patients are in withdrawal.

COCAINE-RELATED DISORDER NOT OTHERWISE SPECIFIED. This classification is reserved for clinicians to use when a cocaine disorder that the clinician sees does not fit into any of the above categories.

Causes and symptoms

Causes

BIOCHEMICAL/PHYSIOLOGICAL CAUSES. Twin studies have demonstrated that there is a higher rate of cocaine abuse in identical twins as compared to fraternal twins. This indicates that genetic factors contribute to the development of cocaine abuse. This finding also indicates, however, that unique environmental factors contribute to the development of cocaine abuse, as well. (If genes alone determined who would develop cocaine dependence,

100% of the identical twins with the predisposing genes would develop the disorder. However, because the results show only a relationship, or a correlation, between genetics and cocaine use among twins, these results indicate that other factors must be at work, as well.) Studies have also shown that disorders like **attention-deficit/hyperactivity disorder** (ADHD), **conduct disorder**, and **antisocial personality disorder** all have genetic components, and since patients who abuse cocaine have a high incidence of these diagnoses, they may also be genetically predisposed to abusing cocaine.

REINFORCEMENT. Learning and conditioning also play a unique role in the perpetuation of cocaine abuse. Each inhalation and injection of cocaine causes pleasurable feelings that reinforce the drug-taking procedure. In addition, the patient's environment also plays a role in cueing and reinforcing the experience in the patient's mind. The association between cocaine and environment is so strong that many people recovering from cocaine **addiction** report that being in an area where they used drugs brings back memories of the experience and makes them crave drugs. Specific areas of the **brain** are thought to be involved in cocaine craving, including the amygdala (a part of the brain that controls aggression and emotional reactivity), and the prefrontal cortex (a part of the brain that regulates anger, aggression, and the brain's assessment of fear, threats, and danger).

Symptoms

The following list is a summary of the acute (short-term) physical and psychological effects of cocaine on the body:

- blood vessels constrict
- elevated heart rate
- elevated blood pressure
- a feeling of intense happiness
- elevated energy level
- a state of increased alertness and sensory sensitivity
- elevated anxiety
- panic attacks
- elevated self-esteem
- diminished appetite
- spontaneous ejaculation and heightened sexual arousal
- psychosis (loss of contact with reality)

The following list is a summary of the chronic (long-term) physical and psychological effects of cocaine on the body:

- depressed mood
- irritability
- physical agitation
- decreased motivation
- difficulty sleeping
- hypervigilance
- elevated anxiety
- panic attacks
- hallucinations
- psychosis

Demographics

The patterns of cocaine abuse in the United States have changed much over the past thirty years. The patterns have also been changing in other parts of the world as well, including South America and Western Europe. In the United States, several studies have attempted to track drug abuse in many different populations. The studies include: the Monitoring the Future Study (MTF); the National Household Survey on Drug Abuse (NHSDA); the Drug Abuse Warning Network (DAWN), which gets reports from Emergency Rooms and medical examiners' offices on drug-related cases and deaths; and Arrestee Drug Abuse Monitoring (ADAM), which gets information on urine samples obtained from people who have been arrested.

In the annual MTF study, cocaine use among high school seniors had declined from 13.1% in 1985 to 3.1% in 1992—the lowest it had been since 1975 when the survey was first implemented. The rate of cocaine use began to rise again and peaked at 5.5% in 1997. The NHSDA found that the levels of cocaine use declined over the same time period. The decline in the rates has been thought to be due in part to education about the risks of cocaine abuse.

The incidence of new crack cocaine users has also decreased. There was a minimal decline in the numbers of excessive cocaine users between the years 1985 and 1997. The Epidemiologic Catchment Area (ECA) studies done in the early 1980s combined cocaine dependence with cocaine abuse and found that one-month to six-month prevalence rates for cocaine abuse and dependence were low or could not be measured. The lifetime rate of cocaine abuse was 0.2%.

A 1997 study from The National Institute on Drug Abuse indicates that among outpatients who abuse substances, 55% abuse cocaine.

Cocaine abuse affects both genders and many different populations across the United States. Males are one-

and-a-half to two times more likely to abuse cocaine than females. Cocaine began as a drug of the upper classes in the 1970s; now the socioeconomic status of cocaine users has shifted. Cocaine is more likely to be abused by the economically disadvantaged because it is easy for them to get, and it is inexpensive ($10 for a small bag of crack cocaine). These factors have led to increased violence (because people who are cocaine dependent often will become involved in illegal activity, such as drug dealing, in order to acquire funds for their habit) and higher rates of acquired immune deficiency (AIDS) among disadvantaged populations.

Diagnosis

If a mental health clinician suspects cocaine use, he or she may ask the patient specifically about swallowing, injecting, or smoking the substance. Urine and blood testing will also be conducted to determine the presence of the substance. Doctors may also talk to friends or relatives concerning the patient's drug use, especially for cases in which the physician suspects that the patient is not being entirely honest about substance use. The clinician may also investigate a patient's legal history for drug arrests that may give clues to periods of substance abuse to which the patient will not admit.

Differential diagnosis

Differential diagnosis is the process of distinguishing one condition from other, similar conditions. The cocaine abuse disorder is easily confused with other substance abuse disorders and various forms of mental illness.

The symptoms of cocaine intoxication, such as increased talkativeness, poor sleep, and the intense feelings of happiness are similar to the symptoms for **bipolar disorder**, so the urine toxification screening test may play a key role in the diagnosis. Patients with cocaine intoxication with hallucinations and delusions can be mistaken for schizophrenic patients instead, further emphasizing the importance of the urine and blood screens. As part of establishing the diagnosis, the physician must also rule out PCP (phencyclidine) intoxication and Cushing's disease (an endocrine disorder of excessive cortisol production). Withdrawal symptoms are similar to those of the patient with major depression. For this reason, the clinician may ask the patient about his or her mood during times of abstinence from drug use to discern if any true mood disorders are present. If cocaine use is causing depression, the depression should resolve within a couple of weeks of stopping drug use.

Laboratory testing

The breakdown products of cocaine remain in the urine. The length of time that they remain depends on the dose of cocaine, but most doses would not remain in the urine longer than a few days. Cocaine can also be found in other bodily fluids such as blood, saliva, sweat, and hair, and these provide better estimates as to recent cocaine use. The hair can hold evidence that a patient has been using drugs for weeks to months. **Positron emission tomography** (PET) and single-photon emission **computed tomography** (SPECT) are different kinds of **imaging studies**. Both kinds of scans look at the amount of blood that is flowing to the brain. When these images are taken of the brains of people who abuse cocaine, the resulting scans have revealed abnormalities in certain sections of the brain. The brains of people addicted to cocaine shrink, or atrophy.

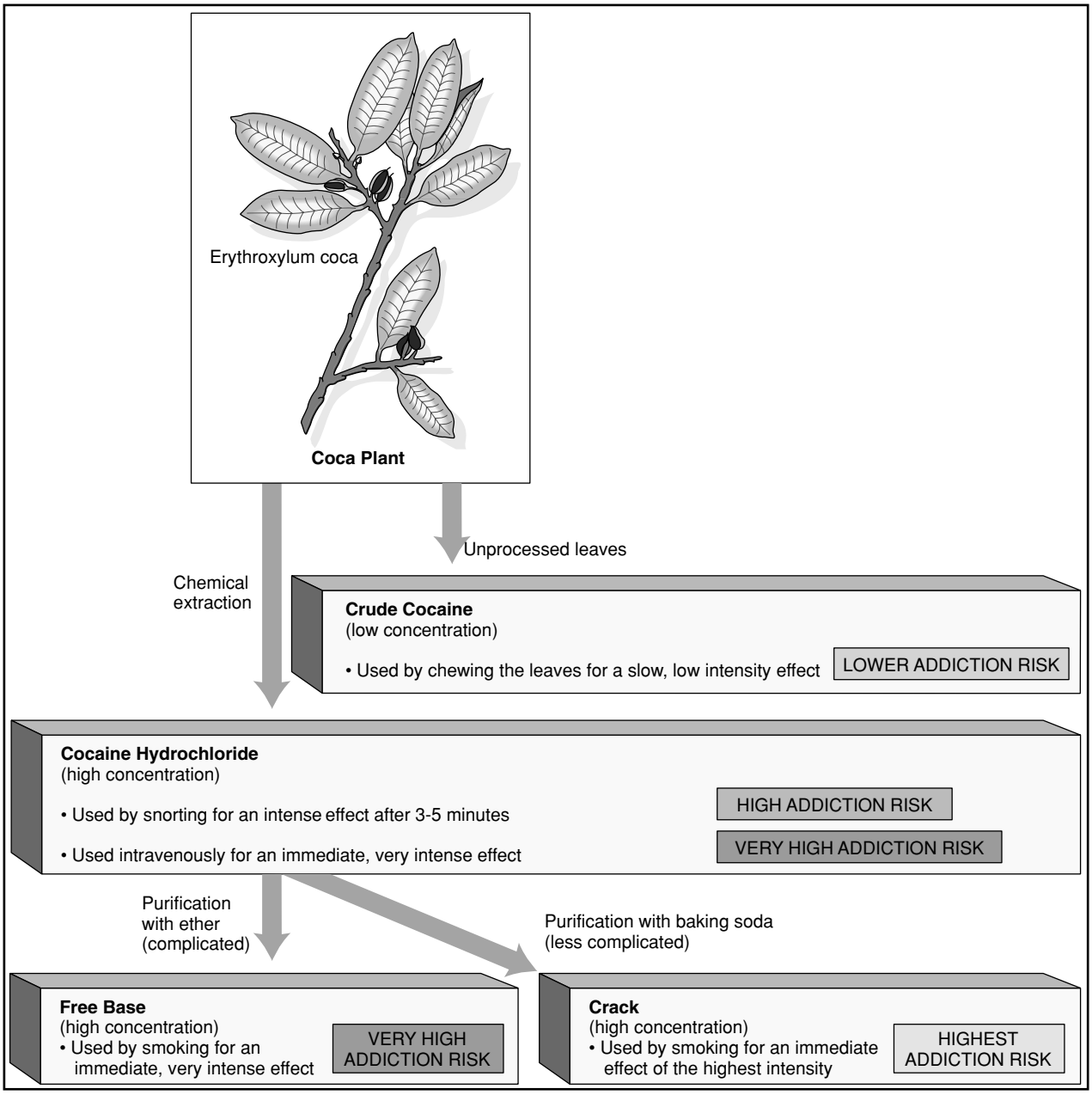
Neuropsychological assessment

Neuropsychological testing is also an important tool for examining the effects of toxic substances on brain functioning. Some physicians may use neuropsychological assessments to reveal patients' cognitive and physical impairment after cocaine use. Neuropsychological testing assesses brain functioning through structured and systematic behavioral observation. Neuropsychological tests are designed to examine a variety of cognitive abilities, including speed of information processing, attention, memory, and language. An example of a task that a physician might ask the patient to complete as part of a neuropsychological examination is to name as many words beginning with a particular letter as the patient can in one minute. Patients who abuse cocaine often have difficulty completing tasks, such as the one described, that require concentration and memory.

Treatments

Psychological and social interventions

TREATMENT SETTINGS. Not all patients who abuse cocaine need to resort to long-term treatment. Treatment length varies with the degree that a person is dependent on the substance. If the patient has other psychiatric conditions such as major depression or **schizophrenia** or has significant medical complications of cocaine abuse, then he or she is more likely to require higher-intensity treatment. Residential programs/therapeutic communities may be helpful, particularly in more severe cases. Patients typically spend six to 12 months in such programs, which may also include vocational training and other features. The availability of such treatment, as well



Various forms of cocaine and the addiction risks associated with them.

as medical insurance's ability to cover treatment, are all issues that affect the patient's access to treatment.

PSYCHOTHERAPY. A wide range of behavioral interventions have been successfully used to treat cocaine addiction. The approach used must be tailored to the specific needs of each individual patient, however.

Contingency management rewards drug abstinence (confirmed by urine testing) with points or vouchers which patients can exchange for such things as an evening out or membership in a gym. **Cognitive-behavioral therapy** helps users learn to recognize and avoid situations most likely to lead to cocaine use and to develop healthier ways to cope with stressful situations.

Supportive therapy helps patients to modify their behavior by preventing relapse by taking actions such as staying away from drug-using friends and from neighborhoods or situations where cocaine is abundant.

Self-help groups like Narcotics Anonymous (NA) or Cocaine Anonymous (CA) are helpful for many recovering substance abusers. CA is a twelve-step program for

cocaine abusers modeled after Alcoholics Anonymous (AA). **Support groups** and **group therapy** led by a therapist can be helpful because other addicts can share coping and relapse-prevention strategies. The group's support can help patients face devastating changes and life issues. Some experts recommend that patients be cocaine-free for at least two weeks before participating in a group, but other experts argue that a two-week waiting period is unnecessary and counterproductive. Group counseling sessions led by drug counselors who are in recovery themselves are also useful for some people overcoming their addictions. These group counseling sessions differ from group therapy in that the people in a counseling group are constantly changing.

The National Institute of Drug Abuse conducted a study comparing different forms of **psychotherapy**: patients who had both group drug counseling and individual drug counseling had improved outcomes. Patients who had cognitive-behavioral therapy stayed in treatment longer.

Medications

Many medications—greater than twenty—have been tested but none have been found to reduce the intensity of withdrawal. Dopamine agonists like **amantadine** and bromocriptine and tricyclic antidepressants such as **desipramine** have failed in studies to help treat symptoms of cocaine withdrawal or intoxication.

Alternative therapy

Alternative techniques, such as **acupuncture**, EEG **biofeedback**, and visualization, may be useful in treating addiction when combined with conventional treatment approaches.

Prognosis

Not all cocaine abusers become dependent on the drug. However, even someone who only uses occasionally can experience the harmful effects (interpersonal relationship conflicts, work or school difficulties, etc.) of using cocaine, and even occasional use is enough to addict. In the course of a person's battle with cocaine abuse, he or she may vary the forms of the drug that he or she uses. A person may use the inhaled form at one time and the injected form at another, for example.

Many studies of short-term outpatient treatment over a six-month to two-year period indicate that people addicted to cocaine have a better chance of recovering than people who are addicted to heroin. A study of veterans who participated in an inpatient or day hospital treatment program that lasted 28 days, revealed that about 60% of people who were abstinent at four months were able to maintain their abstinence at seven months.

Having a good social support network greatly improves the prognosis for recovery from cocaine abuse and dependence.

Prevention

Efforts to prevent cocaine abuse, as well as any substance abuse, begin with prevention programs that are based in schools, in the workplace, heath care clinics, criminal justice systems, and public housing. Programs such as Students Taught Awareness (STAR) are cost effective and have reduced the rates of substance abuse in the schools. These school-based programs also foster parental involvement and education about substance abuse issues. The juvenile justice system also implements drug prevention programs. Even many workplaces provide drug screening and treatment and counseling for those who test positive. Employers may also provide workshops on substance abuse prevention. The United States Department of Housing and Urban Development (HUD) also sponsors drug prevention programs.

See also Addiction; Detoxification; Disease concept of chemical dependency

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Cocaine Anonymous World Services (CAWS). 3740 Overland Ave. Ste. C, Los Angeles, CA, 90034. (310) 559-5833. <http://www.ca.org>.

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Cogentin *see* **Benztropine**

Cognex *see* **Tacrine**

Cognistat

Definition

The Cognistat is a standardized neurobehavioral screening test. It is a test that examines neurological (**brain** and central nervous system) health in relation to a person's behavior.

Purpose

As a screening test, the Cognistat may be administered to identify basic strengths and weaknesses so that further tests (if necessary) can be selected, and the data provided by the Cognistat can then be used as preliminary data against which scores from other tests given may be compared. Cognistat results have been used in a number of arenas, most notably in behavioral medicine. For example, Cognistat results may be useful to track cognitive decline (decreased thinking and reasoning abilities) in patients with organic brain disorders, to develop helpful strategies for cognitive problems associated with **schizophrenia**, and to help distinguish among terminally ill cancer patients those with depression and anxiety versus those with cognitive impairment.

KEY TERMS

Screening test—A test given as a preliminary tool, that helps to target a later, more thorough analysis.

Standardized test—A test that follows a regimented structure, and each individual's scores may be compared with those of groups of people. In the case of the Cognistat, test takers' scores can be compared to groups of young adults, middle-aged adults, the geriatric, and people who have undergone neurosurgery.

Precautions

The Cognistat is more sensitive than many similar tests, but considers a limited sample of behavior at a brief point in time. Thus, its results are not generalizable and should not be viewed as conclusive indicators of the areas being assessed. It is important that the examiner be properly trained in the use of the test. Test takers may be affected by test-related discomfort or performance anxiety. This may be particularly true when prior to testing, the examinee was not fully aware of his or her deficits, especially deficits that become more apparent as testing progresses. The test's reliability has not been fully documented. Further research and standardization data is needed.

Description

The Cognistat usually takes less than 45 minutes to take, and the test explores, quantifies, and describes performance in central areas of brain-behavior relations: level of consciousness, orientation, attention, language, constructional ability, memory, calculations and reasoning. The sub-areas of language are spontaneous speech, comprehension, repetition and naming. The sub-areas of reasoning are similarities and judgment. Exploration occurs through interactive behavioral tasks that rely on perception, cognitive processing, and motor skills. The test is more quickly administered to higher than lower functioning individuals by providing a difficult screening item at the beginning of each section. Only when a screening item is missed are the metric, or more remedial, items applied, usually from easiest to most difficult within that section.

The test begins with the examiner asking general questions of the test taker (name, address, age, etc.), and while these questions are being answered, the examiner is subjectively assessing the test taker's level of consciousness. Then, the examiner asks general questions to confirm the test taker's level of orientation, meaning that

the test taker is correctly oriented to place and time— he or she knows what day it is and where he or she is. To test the examinee's attention and memory, the test taker will be asked to repeat a series of digits and the first part of a verbal memory task will be given. (This task will be asked about again later in the test.)

The language section begins with a sample of spontaneous speech derived by asking for a description of a detailed line drawing. The language comprehension section requires responses to simple commands that involve manipulation of common objects placed before the examinee. In the language repetition subtest, the test taker is asked to repeat short phrases and simple sentences. In naming, the last of the language subtests, the screening item differs in form from the metric (easier) items. In the screening item, the examiner holds up an object and asks the test taker to name its four major parts, as the examiner points to them one after another. If the test taker fails, he or she is asked to name eight separate objects, one after another represented by line drawings.

In the next section, constructional ability, the screening item is a visual memory task wherein a stimulus sheet is presented for ten seconds, and the examinee is asked to draw the stimuli from memory. The test taker is then asked to assemble plastic tiles into designs, one after another, as each is shown on a card. Faster completion yields greater points. After the constructional items, the test taker is asked to recall the verbal memory items presented earlier. For items he or she cannot recall, the examiner provides prompts, or clues.

The calculations section is composed of simple verbal mathematics, and is followed by the reasoning section, which includes two subtests. The first consists of associative thinking items known as similarities. In similarities items, the examinee is asked to explain how two concepts are alike. Greater points are awarded if their concept is abstract rather than concrete. The final subtest on the Cognistat is the judgment subtest of the reasoning section. In the judgment subtest, the examinee is asked to answer questions that demonstrate practical judgment in solving basic problem scenarios. Scores for this subtest are weighted based on their appropriateness. There is only one fully appropriate response to each item.

The test booklet provides space for listing medications, and for noting comments about any physical deficits and the examinee's impression of his or her own performance.

Results

When test administration is complete, the examiner tallies the points earned in each section, and plots them on the cognitive status profile located on the front of the test booklet. On the profile, numerical scores are described to fall within the normal or impaired range. The impaired range is broken down into mild, moderate and severe. An individual's scores can also be compared to standardization group data, and their profile may be compared to five case study profiles presented in the test guide. The few items that do not allow for quantitative analysis —the sample of spontaneous speech, for example— are factored into the interpretation of results by the examiner. There is no mechanism for transforming raw scores into percentiles or standard scores, and the test is not designed to generate one main score.

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Cognitive problem-solving skills training

Definition

Cognitive problem-solving skills training (CPSST) attempts to decrease a child's inappropriate or disruptive behaviors by teaching the child new skills for approaching situations that previously provoked negative behavior. Using both cognitive and behavioral techniques and focusing on the child more than on the parents or the family unit, CPSST helps the child gain the ability to self-manage thoughts and feelings and interact appropriately with others by developing new perspectives and solutions. The basis of the treatment is the underlying principle that children lacking constructive ways to address the environment have problematic behaviors; teaching these children ways to positively problem-solve and challenge dysfunctional thoughts improves functioning.

Purpose

The goal of CPSST is to reduce or terminate inappropriate, dysfunctional behaviors by expanding the *behavioral repertoire* (including ways of cognitive processing). The behavioral repertoire is the range of ways of behaving that an individual possesses. In children with **conduct disorder**, **intermittent explosive disorder**, **oppositional-defiant disorder**, antisocial behaviors, aggressive acting-out, or **attention-deficit/hyperactivity disorder** with disruptive behavior, the number of ways of interpreting reality and responding to the world are limited and involve negative responses. Although CPSST originally focused on children with problem behaviors or poor relationships with others, it has generalized to a variety of different disorders in children and adults (this treatment has the most research supporting its use in children).

Description

The therapist conducts individual CPSST sessions with the child, once a week for 45 minutes to an hour, typically for several months to a year. The cognitive portion of the treatment involves changing faulty or narrow views of daily situations, confronting irrational interpretations of others' actions, challenging unhelpful assumptions that typically underlie the individual's problem behaviors, and generating alternative solutions to problems. For example, meeting with a child who has received a school suspension for becoming physically enraged at a teacher, the therapist starts by exploring the situation with the child, asking what thoughts and feelings were experienced. The child might state, "My teacher hates me. I'm always getting sent to the principal and she yells at me all the time." The therapist helps the child see some faulty ways of thinking by asking what the child has seen or experienced in the classroom previous to this incident, thus exploring the supporting evidence for the "my teacher hates me" notion. Questions would be ones that could confirm or disconfirm the assumptions, or that identify the precipitants of the teacher disciplining the child. The therapist tries to help the child shift his or her perceptions so that, instead of seeing the student-teacher negative interactions as something external to the self, the child comes to see his or her part in the problem. This discussion also helps the child to discern opportunities to influence the outcome of the interactions. When the child makes a global, stable, and negative attribution about why the interactions with the teacher are negative—where the attitude of the teacher is the cause of the problems—the child loses the sense of having any efficacy and is liable to show poorer behavior. By changing the child's perceptions and examining different options for the child's-responses in that situation, however, the child can identify ways that changing his or her own behavior could improve the outcome.

KEY TERMS

Behavior modification—An approach to therapy based on the principles of operant conditioning. Behavior modification seeks to replace undesirable behaviors with preferable behaviors through the use of positive or negative reinforcement.

Cognitive-behavioral therapy—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

Response-contingent learning—A principle that posits that the consequences of a behavior determine whether it will increase or decrease in frequency. Behaviors that bring about desired responses tend to decrease, while those that either remove the chance to obtain a desirable outcome, or those that cause some unpleasant or painful consequence, tend to decrease.

Social learning—Learning by observing others' responses and acquiring those responses through imitation of the role model(s).

The behavioral aspect of CPSST involves **modeling** of more positive behaviors; role-playing challenging situations; and rewarding improvement in behavior, providing corrective feedback on alternative (and more appropriate) ways of handling situations when undesirable behavior occurs. In each session, the child is coached on problem-solving techniques including brainstorming a number of possible solutions to difficulties, evaluating solutions, and planning the steps involving in gaining a desired goal (also called *means-end thinking*). For instance, if the child in the above example felt that the teacher's accusations were unfair, the therapist would help to come up with some options for the child to use in the event of a similar situation (such as visualizing a calming scene, using a mediator to work out the conflict, or avoiding the behaviors that precipitate a trip to the principal's office). The options generated would be discussed and evaluated as to how practical they are and how to implement them.

The child is given therapy homework of implementing these newer ways of thinking and behaving in specif-

ic types of problematic situations in school, with peers, or at home. The child might be asked to keep track of negative, externalizing thoughts by keeping a log of them for several days. The therapist would ask the child to conduct an experiment—try one of the new options and compare the results. Typically, the between-sessions work begins with the conditions that appear the easiest in which to successfully use the updated ways of thinking and behaving, gradually progressing to more complex or challenging circumstances. The child would get rewarded for trying the new techniques with praise, hugs, or earning points towards something desired.

Although the bulk of the sessions involve the individual child and the therapist, the parents are brought into the therapy for a portion of the work. The parents observe the therapist and the child as they practice the new skills and are educated on how to assist the child outside the sessions. Parents learn how to remind the child correctly to use the CPSST techniques for problem-solving in daily living and assist the child with the steps involved in applying these skills. Parents are also coached on how to promote the positive behaviors by rewarding their occurrence with praise, extra attention, points toward obtaining a reward desired by the child, stickers or other small indicators of positive behavior, additional privileges, or hugs (and other affectionate gestures). The scientific term for the rewarding of desired behavior is *positive reinforcement*, referring to consequences that cause the desired target behavior to increase.

In research studies of outcomes, CPSST has been found to be effective in changing children's behavior. Changes in behavior have been shown to persist long-term after completion of treatment. Success in altering undesirable behaviors is enhanced when CPSST is combined with **parent management training**. Parent management training is the in-depth education of parents or other primary caretakers in applying behavioral techniques such as positive **reinforcement** or time away from reinforcement opportunities in their parenting.

Risks

Inappropriate or inept application of cognitive-behavioral techniques such as those used in CPSST may intensify the problem. CPSST should be undertaken with a behavioral health professional (**psychologist**, **psychiatrist**, or clinical social worker) with experience in CPSST. Parents should seek therapists with good credentials, skills, and training.

Results

While individual results vary, problematic behaviors are reduced or eliminated in many children.

See also Behavior modification; Token economy system

Resources

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Association for the Advancement of Behavior Therapy. 305 Seventh Avenue, 16th Floor, New York, NY 10001-60008. (212) 647-1890.

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Cognitive remediation

Definition

Cognitive remediation is a teaching process that targets areas of neuropsychological functioning involved in learning and basic day-to-day functioning.

Purpose

The goals of cognitive remediation are to bolster specific cognitive capacities that are weak and also to teach compensatory strategies. Cognitive remediation is used primarily with two groups of people: those who have suffered from a traumatic **brain** injury (a **stroke**, tumor, or a head injury); and those who have learning disabilities. For people with brain injury, remediation typically targets the following neuropsychological func-

tions: attention and concentration; memory; planning; monitoring one's work or behavior; and making adjustments based on feedback. Remediation is also used to help children and adults cope with learning disabilities. Learning disabilities can interfere with progress in reading; in understanding and communicating through spoken language; in writing; in arithmetic; in understanding such nonverbal information as telling time or understanding visual information; and in comprehending social interactions and cues. Difficulties with concentration, problem-solving, organization, identifying errors, and using feedback effectively are also areas that can be treated with cognitive remediation.

KEY TERMS

Auditory—Pertaining to the sense of hearing.

Cognitive—Pertaining to the mental processes of memory, perception, judgment, and reasoning.

Compensatory—Counterbalancing or offsetting. A compensatory strategy is one that makes up for or balances a weakness in some area of functioning.

Modality—One of the primary forms of sensation, as vision, touch, or hearing.

Socialization—An ongoing process in which a person learns and internalizes the values and behavior patterns of his or her culture and social group.

Description

Individuals who have suffered a traumatic brain injury will work with a remediator using computer programs that target one area at a time, such as attention. The individual is then helped to generalize what is learned from the program to real life. This **intervention** is usually done at a hospital, although it is not limited to clinical settings. Remediation for this group of people is considered helpful but not curative. It is typically practiced by a neuropsychologist.

Remediation for individuals with learning disabilities aims to bolster a particular area of learning or adaptation, such as in academics or socialization. Although the intervention varies according to the disability and the individual's profile of strengths and weaknesses, the remediator will make use of the person's stronger capacities to bolster the weaker ones. For example, the person might need help with written language because he frequently omits words from his sentences. Once it has been determined that the person's oral language (both receptive and expressive) is adequate and that the motor aspect of writing is intact, the remediator has an idea of the person's strengths and weaknesses in the area of writing. In this case, the remediator makes use of the person's stronger auditory (hearing-related) skills to build up the capacity to translate spoken language into written (visual) language. Specifically, the remediator might read aloud a sentence written by the student (with omissions) and ask the student to identify the mistakes that he hears. The person identifies an omission that he hears and then is shown on paper the place where the word is missing. In this way, he can learn to identify errors visually that he can already identify through the auditory modality of listening. This particular exercise fosters visual awareness of errors, which is a symptom or outcome of the deeper problem of translating language from oral to visual form.

The process then continues with diminishing degrees of assistance. Specifically, after the student becomes more skillful in matching visual omissions with the auditory ones read by the remediator, the person himself begins to read the sentences aloud and identify the words that are missing from the sentences on the page. In the next step, he would begin to read his work silently with the same kind of scrutiny as in the previous exercise. In this manner, remediation fosters both learning and internalizing a cognitive capacity.

Cognitive remediation sessions for learning disabilities usually take place twice a week. This type of intervention is practiced by psychologists, neuropsychologists, special educators, and learning specialists. The depth and breadth of the intervention will vary according to the remediator's professional training and his or her particular area of expertise. Some professionals specialize in working with certain types of learning disabilities; some, like psychologists, may incorporate their understanding of emotional difficulties within their work as a cognitive remediator.

Preparation

Before remediation can begin, the person being treated must receive a neuropsychological or in-depth psychological evaluation in order to identify the underlying neuropsychological capacities (i.e., language, memory, attention, visual perception, visual spatial abilities, motor abilities) that are interfering with acquiring the skills that are needed. The evaluation is also intended to rule out emotional difficulties as the primary cause of learning problems. Children with learning disabilities frequently experience feelings of inadequacy and low self-esteem that need to be addressed. If psychological difficulties, however, are the main reason for a person's

academic struggles, he or she should be treated with **psychotherapy** rather than cognitive remediation.

Normal results

When remediation is targeting the problem area accurately, and the individual is actively engaging in the process, then progress should be evident in the skill area targeted; in the person's awareness of his or her area of difficulty; and his or her awareness of some techniques and strategies that are helpful.

See also Learning disorders

Resources

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Cognitive retraining

Definition

Cognitive retraining is a therapeutic strategy that seeks to improve or restore a person's skills in the areas of paying attention, remembering, organizing, reasoning and understanding, problem-solving, decision making, and higher level cognitive abilities. These skills are all interrelated. Cognitive retraining is one aspect of cognitive rehabilitation, a comprehensive approach to restoring such skills after **brain** injury or other disability.

Purpose

The purpose of cognitive retraining is the reduction of cognitive problems associated with brain injury, other disabilities or disorders, and/or aging. The overall purpose of the therapy is to decrease the everyday problems faced by individuals with cognitive difficulties, thereby improving the quality of their lives.

Precautions

The extent to which a person with a brain injury can recover from or compensate for cognitive problems related to the injury requires more information about the person and about their injury. Therapy must be tailored to each individual's needs and abilities. Some cognitive retraining techniques require higher levels of skill, and therefore would be more suitable for persons who have made some progress in their recovery. Moreover, a person's moods and emotions have an effect on their cognitive skills. Someone who is depressed, for example, may need **psychotherapy** and/or medication before he or she can engage in and benefit from cognitive retraining. Some persons with brain injuries may find it difficult to transfer a skill learned in one setting, such as a clinic, to another setting, such as their home. Although a specific individual may show some improvement on training tasks, his or her cognitive skills may not be considered improved or restored unless there is some evidence that the skills have been transferred to everyday settings and can be maintained over time.

Description

Professionals from a variety of fields, such as psychology, psychiatry, occupational therapy, and **speech-language pathology**, may be involved in cognitive retraining. The techniques of cognitive retraining are best known for their use with persons who have suffered a brain injury. Cognitive retraining has also been used to treat **dementia**, **schizophrenia**, attention-deficit disorder, learning disabilities, and cognitive changes associated with aging.

Cognitive retraining includes a considerable amount of repetitive practice that targets the skills of interest. In fact, repetition is essential for the newly retrained skills to become automatic. Regular feedback is another important element of cognitive retraining, as is the use of such rewards as money. Retraining usually begins with simpler skills and proceeds to more complicated skills. The therapist may address cognitive skills while the person is practicing real-life tasks, in an effort to improve their performance of these tasks. In fact, practicing skills in the ways and settings they will be used in real life is critical to the success of retraining efforts. The length of time for cognitive training varies according to the type and extent of the injury and the type of retraining skills used. For example, retraining memory may take months or years. In comparison, it may take only a few days or weeks to retrain someone to organize his or her home or workplace. The use of computers for cognitive retraining has become an increasingly common practice.

Types of cognitive retraining

- Attention and concentration retraining. This type of cognitive retraining aims to improve several abilities, including focusing attention; dividing attention; maintaining attention while reducing the effects of boredom and **fatigue**; and resisting distraction. Attention has been considered the foundation of other more complicated cognitive skills, and therefore an important skill for cognitive retraining. This area of cognitive retraining has been widely researched, and has been shown to improve patients' abilities in various tasks related to attention.
- Memory retraining. Memory retraining involves teaching the patient several strategies that can be used to recall certain types of information. For example, rhymes may be used as a memory aid. A series of numbers, such as a phone number with an area code, may be broken down into smaller groups. A person may be taught to go through each letter of the alphabet until he or she remembers someone's name. Both memory and organization problems are common and disabling after head injury.
- Organizational skills retraining. This approach is used when the person has difficulty keeping track of or finding items, doing tasks in a set order, and/or doing something in a timely manner. Strategies may include having one identified place for an item ("a place for everything and everything in its place"). In addition, the person can be taught to keep the items that are used most frequently closer to him or her (the front or the lower shelves of a cabinet, drawer, closet, or desk, for example). Items that are often used together (such as comb and brush, toothbrush and toothpaste) are placed beside each other. Items may be put into categories (Christmas decorations, Easter decorations, for example). These strategies help individuals function better in their home or work environment.
- Reasoning. Reasoning refers to the ability to connect and organize information in a logical, rational way. Reasoning retraining techniques include: listing the facts or reality of a situation; excluding irrelevant facts or details; putting the steps to solve a problem in a logical order; and avoiding irrational thinking, such as jumping to conclusions based on incomplete information, or focusing on the negative aspects of the situation and ignoring the positive. When the person can connect relevant information in a logical way, they are better able to understand or comprehend it.
- Problem solving. Problem-solving retraining aims to help people define a problem; come up with possible solutions to it; discuss the solution(s) with others and listen to their advice; review the various possible solutions from many perspectives; and evaluate whether the problem was solved after going through these steps. This sequence may be repeated several times until the problem is solved. This process is referred to as "SOLVE," from the first letter of the name of each step: Specify; Options; Listen; Vary; and Evaluate. The "SOLVE" technique is more appropriate for use with individuals at a higher level of functioning.
- Decision making. Decision-making retraining is used when a person must choose among a number of options. The goal of this retraining is to help him or her consider the decision thoroughly before taking any action. The considerations may range from such practical matters as money, people, rules and policies, to personality issues.
- Executive skills. Executive skills retraining refers to teaching individuals how to monitor themselves, control their thinking and actions, think in advance, set goals, manage time, act in socially acceptable ways, and transfer skills to new situations. These are higher-level cognitive skills. Charts and videotapes may be used to monitor behavior, and a variety of questions, tasks, and games may be used in retraining these skills.

KEY TERMS

Cognitive—Pertaining to the mental processes of memory, perception, judgment, and reasoning.

Executive skills—Higher-level cognitive skills that are used when a person makes and carries out plans, organizes and performs complex tasks, etc.

Preparation

Cognitive retraining usually takes place in a quiet room without distractions. It is also important for the person to feel relaxed and calm while they are being retrained in cognitive skills. Engaging in cognitive retraining is not recommended when someone is emotional distressed; for example, if they have recently lost a loved one. The therapist usually evaluates the person's level of cognitive skills and the extent of their cognitive problems before retraining begins. This evaluation provides a way to monitor improvement by comparing the patient's skill levels during and after retraining to his or her skill levels before retraining. Cognitive retraining requires patience and persistence from everyone involved.

Aftercare

The therapist will try to promote the transfer of skills learned using cognitive retraining techniques to the patient's everyday life settings and demands. Training may be continued until the patient's skills are improved, transferred to, and maintained in real world activities.

Risks

It is important for the therapist, patient, and the patient's friends or family members not to assume that improvement on training exercises and tests automatically leads to transfer of the skills to real-life settings.

Normal results

Cognitive retraining may be considered successful if performance on a behavior related to a particular cognitive skill has improved. It is ultimately successful if it helps the injured person improve his or her functioning and meet his or her needs in real-life situations and settings.

See also Attention-deficit/hyperactivity disorder; Dementia; Learning disabilities; Schizophrenia

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Cognitive self-regulation *see* **Self-control strategies**

Cognitive therapy *see* **Cognitive-behavioral therapy**

Cognitive-behavioral therapy

Definition

Cognitive therapy is a psychosocial (both psychological and social) therapy that assumes that faulty thought patterns (called cognitive patterns) cause maladaptive behavior and emotional responses. The treatment focuses on changing thoughts in order to solve psychological and personality problems. Behavior therapy is also a goal-oriented, therapeutic approach, and it treats emotional and behavioral disorders as maladaptive learned responses that can be replaced by healthier ones with appropriate training. Cognitive-behavioral therapy (CBT) integrates features of **behavior modification** into the traditional cognitive restructuring approach.

Purpose

Cognitive-behavioral therapy attempts to change clients' unhealthy behavior through cognitive restructuring (examining assumptions behind the thought patterns) and through the use of behavior therapy techniques.

Cognitive-behavioral therapy is a treatment option for a number of mental disorders, including depression, **dissociative identity disorder**, eating disorders, **generalized anxiety disorder**, **hypochondriasis**, **insomnia**, **obsessive-compulsive disorder**, and **panic disorder** without **agoraphobia**.

Precautions

Cognitive-behavioral therapy may not be appropriate for all patients. Patients with significant cognitive impairments (patients with traumatic **brain** injury or organic brain disease, for example) and individuals who are not willing to take an active role in the treatment process are not usually good candidates.

Description

Origins of the two approaches

Psychologist Aaron Beck developed cognitive therapy in the 1960s. The treatment is based on the principle that maladaptive behavior (ineffective, self-defeating behavior) is triggered by inappropriate or irrational thinking patterns, called automatic thoughts. Instead of reacting to the reality of a situation, an individual automatically reacts to his or her own distorted view of the situation. Cognitive therapy strives to change these thought patterns (also known as cognitive distortions), by examining the rationality and validity of the assumptions behind them. This process is termed cognitive restructuring.

Behavior therapy focuses on observable behavior and its modification in the present, in sharp contrast to the psychoanalytic method of Sigmund Freud (1856-1939), which focuses on unconscious mental processes and their roots in the past. Behavior therapy was developed during

the 1950s by researchers and therapists who were critical of the prevailing psychodynamic treatment methods. The therapy drew on a variety of theories and research, including the classical conditioning principles of the Russian physiologist Ivan Pavlov (1849-1936), the work of American B. F. Skinner (1904-1990), and the work of **psychiatrist** Joesph Wolpe (1915-1997). Pavlov became famous for experiments in which dogs were trained to salivate at the sound of a bell, and Skinner pioneered the concept of operant conditioning, in which behavior is modified by changing the response it elicits. Wolpe is probably best known for his work in the areas of desensitization and **assertiveness training**. By the 1970s, behavior therapy enjoyed widespread popularity as a treatment approach. Since the 1980s, many therapists have begun to use cognitive-behavioral therapy to change clients' unhealthy behavior by replacing negative or self-defeating thought patterns with more positive ones.

The combined approach

In cognitive-behavioral therapy, the therapist works with the patient to identify the thoughts that are causing distress, and employs behavioral therapy techniques to alter the resulting behavior. Patients may have certain fundamental core beliefs, known as schemas, that are flawed and are having a negative impact on the patient's behavior and functioning.

For example, a patient suffering from depression may develop a **social phobia** because he is convinced that he is uninteresting and impossible to love. A cognitive-behavioral therapist would test this assumption by asking the patient to name family and friends who care for him and enjoy his company. By showing the patient that others value him, the therapist exposes the irrationality of the patient's assumption and also provides a new model of thought for the patient to change his previous behavior pattern (i.e., I am an interesting and likeable person, therefore I should not have any problem making new social acquaintances). Additional behavioral techniques such as conditioning (the use of positive and/or negative reinforcements to encourage desired behavior) and **systematic desensitization** (gradual exposure to anxiety-producing situations in order to extinguish the fear response) may then be used to gradually reintroduce the patient to social situations.

Cognitive-behavioral therapy is usually administered in an outpatient setting (clinic or doctor's office) by a specially trained therapist. Therapy may be in either individual or group sessions. Therapists are psychologists (Ph.D., Psy.D., Ed.D., or M.A. degree), clinical **social workers** (M.S.W., D.S.W., or L.S.W. degree), counselors (M.A. or M.S. degree), or psychiatrists (M.D. trained in psychiatry).

Techniques

Therapists use several different techniques in the course of cognitive-behavioral therapy to help patients examine and change thoughts and behaviors. These include:

- *Validity testing.* The therapist asks the patient to defend his or her thoughts and beliefs. If the patient cannot produce objective evidence supporting his or her assumptions, the invalidity, or faulty nature, is exposed.
- *Cognitive rehearsal.* The patient is asked to imagine a difficult situation he or she has encountered in the past, and then works with the therapist to practice how to cope successfully with the problem. When the patient is confronted with a similar situation again, the rehearsed behavior will be drawn on to manage it.
- *Guided discovery.* The therapist asks the patient a series of questions designed to guide the patient towards the discovery of his or her cognitive distortions.
- *Writing in a journal.* Patients keep a detailed written diary of situations that arise in everyday life, the thoughts and emotions surrounding them, and the behaviors that accompany them. The therapist and patient then review the journal together to discover maladaptive thought patterns and how these thoughts impact behavior.
- *Homework.* In order to encourage self-discovery and reinforce insights made in therapy, the therapist may ask the patient to do homework assignments. These may include note-taking during the session, journaling, review of an audiotape of the patient session, or reading books or articles appropriate to the therapy. They may also be more behaviorally focused, applying a newly learned strategy or coping mechanism to a situation, and then recording the results for the next therapy session.
- *Modeling.* Role-playing exercises allow the therapist to act out appropriate reactions to different situations. The patient can then model this behavior.
- *Systematic positive reinforcement.* Human behavior is routinely motivated and rewarded by positive **reinforcement**, and a more specialized version of this phenomenon (systematic positive reinforcement) is used by behavior-oriented therapists. Rules are established that specify particular behaviors that are to be reinforced, and a reward system is set up. With children, this sometimes takes the form of tokens that may be accumulated and later exchanged for certain privileges. Just as providing reinforcement strengthens behaviors,

withholding it weakens them. Eradicating undesirable behavior by deliberately withholding reinforcement is another popular treatment method called extinction. For example, a child who habitually shouts to attract attention may be ignored unless he or she speaks in a conversational tone.

- *Aversive conditioning*. This technique employs the principles of classical conditioning to lessen the appeal of a behavior that is difficult to change because it is either very habitual or temporarily rewarding. The client is exposed to an unpleasant stimulus while engaged in or thinking about the behavior in question. Eventually the behavior itself becomes associated with unpleasant rather than pleasant feelings. One treatment method used with alcoholics is the administration of a nausea-inducing drug together with an alcoholic beverage to produce an aversion to the taste and smell of alcohol by having it become associated with nausea. In counterconditioning, a maladaptive response is weakened by the strengthening of a response that is incompatible with it. A well-known type of counterconditioning is **systematic desensitization**, which counteracts the anxiety connected with a particular behavior or situation by inducing a relaxed response to it instead. This method is often used in the treatment of people who are afraid of flying.

Preparation

Because cognitive-behavioral therapy is a collaborative effort between therapist and patient, a comfortable working relationship is critical to successful treatment. Individuals interested in CBT should schedule a consultation session with their prospective therapist before starting treatment. The consultation session is similar to an interview session, and it allows both patient and therapist to get to know one another. During the consultation, the therapist gathers information to make an initial assessment of the patient and to recommend both direction and goals for treatment. The patient has the opportunity to learn about the therapist's professional credentials, his/her approach to treatment, and other relevant issues.

In some managed-care settings, an intake interview is required before a patient can meet with a therapist. The intake interview is typically performed by a psychiatric nurse, counselor, or social worker, either face-to-face or over the phone. It is used to gather a brief background on treatment history and make a preliminary evaluation of the patient before assigning them to a therapist.

Results

Because cognitive-behavioral therapy is employed for such a broad spectrum of illnesses, and is often used in conjunction with medications and other treatment interventions, it is difficult to measure overall success rates for the therapy. However, several studies have indicated that CBT:

- may reduce the rate of rehospitalization and improve social and occupational functioning for **bipolar disorder** patients, when combined with pharmacotherapy (treatment with medication)
- is an effective treatment for patients with bulimia nervosa
- can help generalized anxiety patients manage their worry, when combined with relaxation exercises
- is helpful in treating hypochondriasis
- may be effective for treating depression, especially when combined with pharmacotherapy, and may also prevent depression in at-risk children
- is one of the first-line treatments for obsessive-compulsive disorder
- that focuses on education and provides some exposure and coping skills is effective for treating panic disorder without agoraphobia
- is effective for helping to treat insomnia, and its effects may be sustained longer than the effects of medications alone

See also Aversion therapy; Behavior modification; Cognitive problem-solving skills training; Cognitive retraining techniques; Covert sensitization; Exposure treatment; Rational emotive therapy

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Communication skills and disorders

Definition

Communication skills are the skills needed to use language (spoken, written, signed, or otherwise communicated) to interact with others, and communication disorders are problems related to the development of these skills.

Description

Language employs symbols—words, gestures, or spoken sounds—to represent objects and ideas. Communication of language begins with spoken sounds combined with gestures, relying on two different types of skills. Children first acquire the skills to receive communications, that is, the ability to listen and understand what they hear (supported by accompanying gestures). Next, they will begin experimenting with expressing themselves through speaking and gesturing. Speaking will begin as repetitive syllables, followed by words, phrases, and sentences. Later, children will acquire the skills of reading and writing—the written forms of communication. Although milestones are discussed for development of these skills, many children begin speaking significantly earlier or later than the milestone date. Parents should refrain from attaching too much significance to either deviation from the average. When a child's deviation from the average does cause the parents concern, they may contact a pediatrician or other professional for advice.

Spoken language problems are referred to by a number of labels, including language delay, language disability, or a specific type of language disability. In general, experts distinguish between those people who seem to be slow in developing spoken language (language delay) and those who seem to have difficulty achieving a milestone of spoken language (language disorders). Language disorders include **stuttering**; articulation disorders, such as substituting one sound for another (tandy for candy), omitting a sound (canny for candy), or distorting a sound (shlip for sip); and voice disorders, such as inappropriate pitch, volume, or quality. Causes can be related to hearing, nerve/muscle disorders, head injury, viral diseases, **mental retardation**, drug abuse, or cleft lip or palate.

The ***Diagnostic and Statistical Manual of Mental Disorders*** (also known as the *DSM-IV-TR*), published by the American Psychiatric Association, lists the following disorders as communication disorders:

- **Expressive language disorder:** Disorder characterized by impairment in expressive language development.
- **Mixed receptive-expressive language disorder**: Impairment in both receptive and expressive language development. The affected child has a more difficult time understanding and expressing language as compared to peers.
- **Phonological disorder**: Inability to use expected speech sounds appropriate for the child's age and dialect.
- Stuttering: Unexpected disturbances in the normal patterns and flow of speech.
- Communication disorder not otherwise specified: This may be diagnosed when a child has an irregularity in speech or a difficulty (in voice or pitch, etc.) but the child's symptoms do not exactly match any of the specific categories of impairment that the *DSM* recognizes.

See also Speech-language pathology

Resources

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National Institute on Deafness and Other Communication Disorders. National Institutes of Health, Bethesda, MD, 20892. <http://www.nidcd.nih.gov/>.

Community mental health

Definition

Community mental health is a decentralized pattern of mental health, mental health care, or other services for people with mental illnesses. Community-based care is designed to supplement and decrease the need for more costly inpatient mental health care delivered in hospitals. Community mental health care may be more accessible and responsive to local needs because it is based in a variety of community settings rather than aggregating and isolating patients and patient care in central hospitals. Community mental health assessment, which has grown into a science called psychiatric epidemiology, is a field of research measuring rates of mental disorder upon which mental health care systems can be developed and evaluated.

KEY TERMS

Catchment—In mental health, a term that refers to a particular geographical area served by a particular mental health agency.

Psychiatric epidemiology—A field of research for establishing the incidence, distribution or prevalence, and control of mental disorders in a population, including the sum of the factors controlling the presence of mental disorders.

Community mental health centers

In the United States, a modern increase in community mental health care delivery began in the 1960s when President John F. Kennedy signed the 1963 Community Mental Health Centers (CMHC) Act (Public Law #88-164). Growing community mental health capacities were intended to complement and mirror trends toward fewer hospital stays and shorter visits for mental illness (see **Deinstitutionalization**). This restructuring of mental health service delivery has occurred in the context of evolving fiscal responsibilities, however. The goals and practices of community mental health have been complicated and revised by economic and political changes.

The National Institute of Mental Health (NIMH) initially developed a CMHC program in the 1960s. CMHCs were designed to provide comprehensive services for people with mental illness, locate these services closer to home, and provide an umbrella of integrated services for a catchment area of 125,000-250,000 people. CMHCs were designed to provide prevention, early treatment, and continuity of care in communities, promoting social integration of people with mental health needs.

Competing public interests

At the outset, CMHCs were providing outpatient care to people with less severe, episodic, or acute mental health problems. In the 1980s, more people with serious mental illness began using CMHCs, due in part to deinstitutionalization, and following the redirection and capping of federal funds for local mental health care. With growing awareness of the homeless mentally ill, state-funded CMHCs faced new challenges, and their work became fragmented according to catchment areas of responsibility, leaving some urban centers overburdened, while others maintained locally funded operations, limiting responsibility for their area only.

The growth of local community mental health centers was an example of competing governmental interests and authorities. Growing numbers of CMHCs were mandated federally and to be funded by local communities, bypassing state control. This growth in outpatient capacity was later used to complement decreases in inpatient hospital care, or deinstitutionalization, which reduced the costs of diminishing and state-funded mental hospitals.

Policies to improve public mental health care

Community mental health centers were the first of several programmatic attempts to improve mental health care in the latter part of the twentieth century. A second was when the federal government recommended Community Support Programs (CSPs) in 1977-78 in response to problems associated with deinstitutionalization. CSPs focused on providing direct care and rehabilitation for the chronically mentally ill. However, federal support for mental health care and CMHCs in particular was reduced in 1980-81, with the repeal of the Mental Health Systems Act and the federal budgeting actions that cut funding and provided it through block grants to states.

A third initiative has been to expand the national capacity for children's mental health care under the Child and Adolescent Service System Program (CASSP), beginning in the 1980s. Principles for this system of care included a continuum of services, including mental health. The expansion of mental health classification systems and the ***Diagnostic and Statistical Manual of Mental Disorders*** has helped identify and treat a growing number of children and youth. A fourth initiative was a joint effort by the Robert Wood Johnson Foundation and the department of Housing and Urban Development. Their Program on Chronic Mental Illness (PCMI) promoted the integration of regional mental health authorities in nine cities. Coordinated local mental health systems run by local mental health authorities remain an important goal of mental health policy.

Finally, many private and public health systems have moved towards managed mental health care, which has become also known as behavioral health care. This form of cost containment is a constellation of organizational reforms, financing systems, and regulatory techniques. **Managed care** expanded throughout health care in the 1990s, providing new challenges to mental health care policy. While federal health policy and medical assistance provide reimbursement for mental health care and for people with mental illness, the regulation of these systems has grown increasingly complex.

While the ideals of community mental health were supplemented with new ideals in the years following the CMHC Act, they were not forgotten. Thanks to the work of NIMH, Medicare and Medicaid legislation (1965), and Supplemental Security Income legislation (1972), com-

munities were able to provide mental health care for growing populations in need. National epidemiological studies in the 1980s and 1990s reinforced the large-scale need for mental health care, as CMHCs and subsequent organizational forms provided services to the nation.

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Michael Polgar, Ph.D.

Compliance

Definition

Compliance with appropriate, recommended, and prescribed mental health treatments simply means that a person is following a doctor's orders. Compliance is more likely when there is agreement and confidence regarding the medical **diagnosis** and prognosis. Compliance is complicated by uncertainty about the nature of an illness and/or the effects of certain treatments, particularly medications.

In everyday usage, the term compliance means deference and obedience, elevating the authority of medical expertise. Alternatively, adherence to medical advice refers to a somewhat more informed and equitable decision by a consumer to stick with appropriate medical treatment. In any case, a mental health treatment cannot be effective or even evaluated if a consumer does not follow a doctor's orders. A mental health treatment that is effective for one disorder may not be beneficial for other disorders, and diagnoses may evolve over time, complicating the issue of compliance.

Health providers and consumers

From a health provider's viewpoint, in order for effective medical treatments to have their desired effects, complying or conforming to treatments is absolutely necessary. The concept of medication management reflects this idea that the provider is responsible and in control, while the consumer is a docile body who is incapacitated by disease or condition. From the perspective of health consumers, adherence to medical treatment is enhanced when there is a good health care relationship and when consumers openly share their health beliefs and experience of illness with their provider.

Problems with compliance

In mental health care, uncertainty about compliance is a challenging source of variation in the effectiveness of treatments. Noncompliance can represent a significant risk and cost to the medical system. For providers, partial compliance or discontinuation of medications represents the difficulty of maintaining treatment successes over time. Problems with compliance are often attributed to the consumer, but may also reflect the appropriateness of a medication or treatment.

Compliance rates

Rates of compliance with mental health appointments are the greatest challenge (estimated in one hospital at 91%), while medication noncompliance is the second most challenging problem in the treatment of persons with mental illness. Mental health medication compliance can be determined by questioning patients, counting pills or prescriptions, and through drug monitoring with urine, blood, or other test measures. Overall, recent research estimates compliance to be 58%. Patients who report lower rates are often considered unreliable indicators of compliance, while physicians report higher rates. Compliance with antidepressant medications is higher on average (65%). Mental health medication compliance rates are only somewhat lower than medication compliance in other types of health care, which have been estimated at 76%.

Explaining variation in compliance

Research in psychiatry, psychology, and sociology provides many explanations for variations in compliance. In psychiatry, clinical problems such as drug or alcohol abuse are sometimes used to explain noncompliance. Patients may also discontinue taking medications because of unwanted side effects. Health beliefs and patient-provider relationships are also recognized. In psychology and sociology, health beliefs and behaviors (in context of family, work, etc.) may enhance or limit compliance. If an individual's family member supports medication compliance, and the individual believes in the medicine's benefits, compliance may be enhanced. If an individual discontinues a medicine because it makes him or her drowsy and affects work, compliance may be reduced. People who have limited access to or trust in doctors or medical science, and people whose faith precludes them from certain types of medical care, are less likely to comply with treatment recommendations.

To a large extent, patient compliance is a direct reflection of the quality of the doctor-patient relationship. When provider and consumer achieve a successful treatment alliance, and when the treatment is practical and beneficial for both the provider and the consumer, cooperation reduces concerns about treatment for both parties. When consumers are empowered and motivated to improve their health with the help of a doctor, compliance or adherence to treatment is higher. When there is distrust, disagreement, or misunderstanding involved, as when mental health status is uncertain or treatment side effects are unwelcome, compliance is lower. One British study found that patients with mental disorders were likely to prefer the form of treatment recommended by psychiatrists with whom they had good relationships, even if the treatment itself was painful. Some patients preferred **electroconvulsive therapy** (ECT) to tranquilizers for depression because they had built up trusting relationships with the doctors who used ECT, and perceived the doctors who recommended medications as bullying and condescending. Since noncompliant consumers are less likely to continue in care, they are also less likely to find helpful providers or successful treatments. Thus, noncompliance with treatment may become a self-fulfilling cycle.

Compliance is higher when treatments, including medications, help consumers feel better, when a family supports the treatment, and when taking medication prevents relapse of symptoms. However, as mentioned, people may be distressed by potential side effects of any medication, including those psychiatric medications that limit functioning. Limited functioning through drowsiness, also a problem of the older generation of antihistamines, is the best example. It is an effect of many medicines, particularly those for mental disorders. Other unwelcome side effects of various psychiatric medications include weight gain, involuntary movements such as muscle twitching, and impaired coordination. Consumers may feel embarrassed about taking medication, may have difficulty getting a prescription for medication, and may have financial problems paying for treatment or medication. In some cases, when a patient is non-compliant or perceived to be at odds with treatment recommendations, they may risk losing autonomy over medical decisions. When at risk to self or others, people who are medication noncompliant may be pressured or forced to take medication at the risk of being involuntarily hospitalized.

Multiple challenges in mental health care

Compliance rates reflect the proportion of individuals in treatment who have the highest possibility of successful treatment. Noncompliance rates reflect those individuals who have either discontinued or avoided treatment, and thus have lower probabilities of treatment success. Sometimes patients do not want to get rid of their symptoms (mania, for example), or patients may not consider their experiences (called symptoms) to be indicative of a disorder. In addition, successful mental health care is hampered by the fact that many people with mental health problems either do not use or lack access to mental health care.

The National Co-morbidity Survey shows that only 40% of individuals with serious mental illness receive any treatment in a given year, and 39% of this group receives minimally adequate care. This means that merely 15% of all people in need receive minimally adequate care. Therefore, compliance with treatment is part of a larger national challenge to provide quality mental health care and to use it well.

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Compulsion

Definition

A compulsion is a repetitive, excessive, meaningless activity or mental exercise that a person performs in an attempt to avoid distress or worry.

Description

Compulsions are not voluntary activities and are not performed for pleasure. Instead, a person with a compulsion feels the need to engage in a particular behavior to relieve the **stress** and discomfort which would become overwhelming if the activity were not performed in a specific, repeated manner. Examples of compulsive motor activities are washing hands until raw, repeatedly checking the security of a locked door, and arranging and rearranging items in a set order. Some examples of compulsory mental acts are counting or silently repeating specific words. If a person troubled by compulsions is unable to perform such activities, stress and discomfort increase. The performance of the acts relieves distress but only temporarily.

Often, compulsions are not acts that could logically be expected to relieve or prevent the fears that inspire them. For example, a person might feel compelled to count numbers in a certain order to "undo" the perceived damage or threat that follows a thought or behavior. Or a person might check to make sure a door is locked every few minutes. Compulsions, in some cases, are attempts to undo obsessions and are usually not successful.

See also Obsession; Obsessive-compulsive disorder

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Compulsive gambling *see* **Pathological gambling disorder**

Computed tomography

Definition

Computed tomography scanning, also called CT scan, CAT scan, or computerized axial tomography, is a diagnostic tool that provides views of internal body structures using x rays. In the field of mental health, a CT scan may be used when a patient seeks medical help for symptoms that could possibly be caused by a **brain** tumor. These symptoms may include headaches, emotional abnormalities, or intellectual or memory problems. In these cases, a CT scan may be performed to "rule out" a tumor, so that other tests can be performed in order to establish an accurate **diagnosis**.

Purpose

CT scans are used to image bone, soft tissues, and air. Since the 1990s, CT equipment has become more affordable and available. CT scans have become the imaging exam of choice for the diagnoses of most solid tumors. Because the computerized image is sharp, focused, and three-dimensional, many structures can be better differentiated (visualized) when compared with standard x rays.

Common indications for CT scans include:

- Sinus studies. The CT scan can show details of sinusitis, bone fractures, and the presence of bony tumor involvement. Physicians may order a CT scan of the sinuses to provide an accurate map for surgery.

KEY TERMS

Aneurysm—A symptomless bulging of a weak arterial wall that can rupture, leading to stroke.

Cerebral ateriography—a procedure that allows a wire to be inserted in blood vessels in the brain which generates an image of diseases in these arteries.

Contrast (agent, medium)—A substance injected into the body that illuminates certain structures that would otherwise be hard to see on the radiograph (film).

Gantry—A name for the couch or table used in a CT scan. The patient lies on the gantry while it slides into the x-ray scanner.

Hematoma—An accumulation of blood, often clotted, in a body tissue or organ, usually caused by a break or tear in a blood vessel.

Metastasis—Secondary cancer, or cancer that has spread from one body organ or tissue to another.

Osteoporosis—A loss of bone minerals.

Radiologist—A medical doctor specially trained in radiology (x ray) interpretation and its use in the diagnosis of disease and injury.

Spiral CT—Also referred to as helical CT, this method allows for continuous 360-degree x-ray image capture.

Thoracic—Refers to the chest area. The thorax runs between the abdomen and neck and is encased in the ribs.

- Brain studies. Brain CT scans can detect hematomas (blood clotted mass), tumors, strokes, aneurysms (a blood vessel that ruptures), and degenerative or infected brain tissue. The introduction of CT scanning, especially spiral CT, has helped reduce the need for more invasive procedures such as cerebral angiography (inserting a wire through an artery to where it will reach brain vessels for visualization in real time).
- Body scans. CT scans of the chest, abdomen, spine, and extremities can detect the presence of tumors, enlarged lymph nodes, abnormal collection of fluid, and vertebral disc disease. These scans can also be helpful in evaluating the extent of bone breakdown in osteoporosis.
- Heart and aorta scans. CT scans can focus on the thoracic (chest) or abdominal aorta to locate aneurysms and other possible aortic diseases. A newer type of CT scan, called electron beam CT, can be used to detect calcium buildup in arteries. Because it is a new technology, it is not yet widely used and its indications are not yet well-defined.
- Chest scans. CT scans of the chest are useful in distinguishing tumors and in detailing accumulation of fluid in chest infections.

Precautions

Pregnant women or those who could possibly be pregnant should not have a CT scan, particularly a full body or abdominal scan, unless the diagnostic benefits outweigh the risks. If the exam is necessary for obstetric purposes, technologists are instructed not to repeat films if there are errors. Pregnant patients receiving a CT scan or any x ray exam away from the abdominal area may be protected by a lead apron; most radiation, known as scatter, travels through the body, however, and is not totally blocked by the apron.

Contrast agents are often used in CT exams, though some types of tumors are better seen without it. Patients should discuss the use of contrast agents with their doctor, and should be asked to sign a consent form prior to the administration of contrast. One of the common contrast agents, iodine, can cause allergic reactions. Patients who are known to be allergic to iodine or shellfish should inform the physician prior to the CT scan; a combination of medications can be given to such patients before the scan to prevent or minimize the reaction. Contrast agents may also put patients with diabetes at risk of kidney failure, particularly those taking the medication glucophage.

Description

Computed tomography, is a combination of focused x-ray beams and the computerized production of an image. Introduced in the early 1970s, this radiologic procedure has advanced rapidly and is now widely used, sometimes in the place of standard x rays.

CT equipment

A CT scan may be performed in a hospital or outpatient imaging center. Although the equipment looks large and intimidating, it is very sophisticated and fairly comfortable. The patient is asked to lie on a gantry, or narrow table, that slides into the center of the scanner. The scanner looks like a doughnut and is round in the middle, which allows the x-ray beam to rotate around the patient. The scanner section may also be tilted slightly to allow for certain cross-sectional angles.

CT procedure

The gantry moves very slightly as the precise adjustments for each sectional image are made. A technologist watches the procedure from a window and views the images on a computer screen. Generally, patients are alone during the procedure, though exceptions are sometimes made for pediatric patients. Communication is possible via an intercom system.

It is essential that the patient lie very still during the procedure to prevent motion blurring. In some studies, such as chest CTs, the patient will be asked to hold his or her breath during image capture.

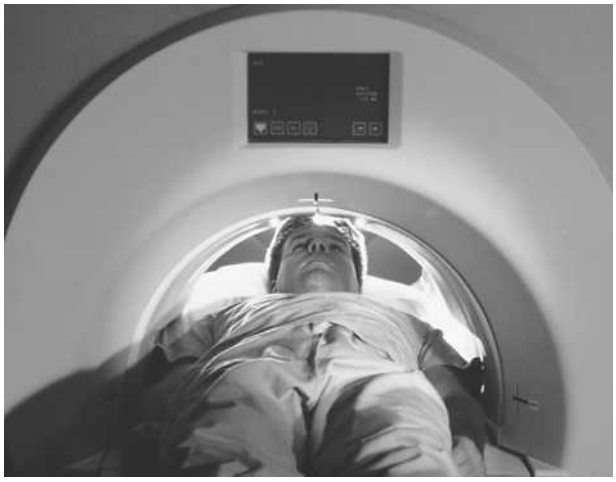
Following the procedure, films of the images are usually printed for the radiologist and referring physician to review. A radiologist can also interpret CT exams on the computer screen. The procedure time will vary in length depending on the area being imaged. Average study times are from 30 to 60 minutes. Some patients may be concerned about claustrophobia (a feeling of being "closed in") but the width of the "doughnut" portion of the scanner is such that many patients can be reassured of openness. Doctors may consider giving sedatives to patients who have severe claustrophobia or difficulty lying still (such as small children).

The CT image

While traditional x-ray machines image organs in two dimensions, often resulting in organs in the front of the body being superimposed over those in the back, CT scans allow for a more three-dimensional effect. CT images can be likened to slices in a loaf of bread. Precise sections of the body can be located and imaged as cross-sectional views. The screen before the technologist shows a computer's analysis of each section detected by the x-ray beam. Thus, various densities of tissue can be easily distinguished.

Contrast agents

Contrast agents are often used in CT exams and in other radiology procedures to illuminate certain details of anatomy more clearly. Some contrasts are natural, such as air or water. A water-based contrast agent is sometimes administered for specific diagnostic purposes. Barium sulfate is commonly used in gastroenterology procedures. The patient may drink this contrast or receive it in an enema. Oral or rectal contrast is usually given when examining the abdomen or cells, but not when scanning the brain or chest. Iodine is the most widely used intravenous contrast agent and is given through an intravenous needle.



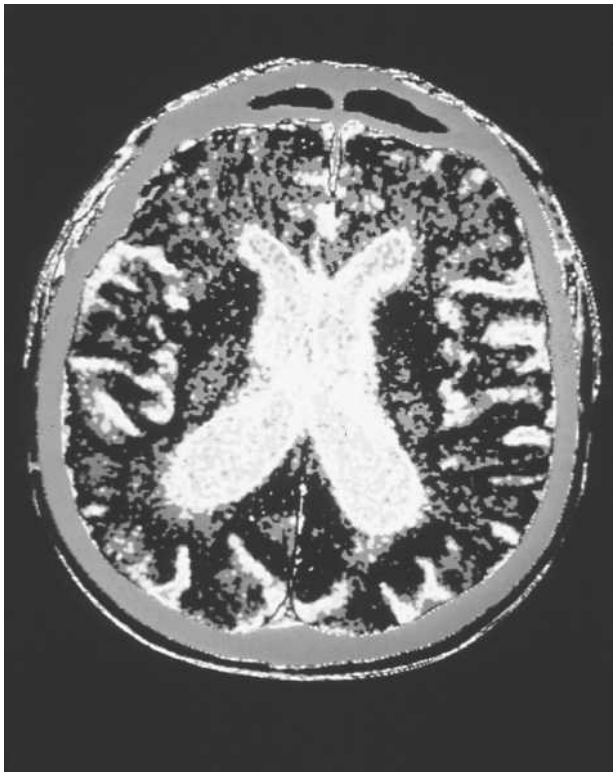
Patient lying on mobile table, entering a CT (computed tomography or CAT) scanner. *(Volker Steger/Science Photo Library, Science Source/Photo Researchers, Inc. Reproduced by permission.)*

If contrast agents are used in the CT exam, these will be administered several minutes before the study begins. Patients undergoing abdominal CT may be asked to drink a contrast medium. Some patients may experience a salty taste, flushing of the face, warmth or slight nausea, or hives from an intravenous contrast injection. Technologists and radiologists have the equipment and training to help patients through these minor reactions and to handle more severe reactions. Severe reactions to contrast are rare, but do occur.

Newer types of CT scans

The spiral CT scan, also called a helical CT, is a newer version of CT. This type of scan is continuous in motion and allows for the continuous re-creation of images. For example, traditional CT allows the technologist to take slices at very small and precise intervals one after the other. Spiral CT allows for a continuous flow of images, without stopping the scanner to move to the next image slice. A major advantage of spiral CT is the ability to reconstruct images anywhere along the length of the study area. Because the procedure is faster, patients are required to lie still for shorter periods of time. The ability to image contrast more rapidly after it is injected, when it is at its highest level, is another advantage of spiral CT's high speed.

Electron beam CT scans are another newer type of CT technology that can be used to detect calcium buildup in arteries. These calcium deposits are potential risk factors for coronary artery disease. Electron beam CT scans take pictures much more quickly than conventional CTs, and are therefore better able to produce clear images of the



Computerized axial tomography (CAT) scan of a human brain with Parkinson's disease showing atrophy. *(GJLP/CNRI/Phototake. Reproduced by permission.) See color insert for color version of photo.*

heart as it pumps blood. Because it is a newer and expensive test, electron beam CT scanning is not widely used.

Some facilities will have spiral, electron, and conventional CT available. Although spiral is more advantageous for many applications, conventional CT is still a superior and precise method for imaging many tissues and structures. The physician will evaluate which type of CT works best for the specific exam purpose.

Preparation

If a contrast medium is administered, the patient may be asked to fast for about four to six hours prior to the procedure. Patients will usually be given a gown (like a typical hospital gown) to be worn during the procedure. All metal and jewelry should be removed to avoid artifacts on the film. Depending on the type of study, patients may also be required to remove dentures.

Aftercare

Generally, no aftercare is required following a CT scan. Immediately following the exam, the technologist will continue to watch the patient for possible adverse contrast reactions. Patients are instructed to advise the technologist of any symptoms, particularly respiratory difficulty. The site of contrast injection will be bandaged and may feel tender following the exam.

Risks

Radiation exposure from a CT scan is similar to, though higher than, that of a conventional x ray. Although this is a risk to pregnant women, the risk for other adults is minimal and should produce no effects. Severe contrast reactions are rare, but they are a risk of many CT procedures.

Normal results

Normal findings on a CT exam show bone, the most dense tissue, as white areas. Tissues and fat will show as various shades of gray, and fluids will be gray or black. Air will also look black. Intravenous, oral, and rectal contrast appear as white areas. The radiologist can determine if tissues and organs appear normal by the sensitivity of the gray shadows.

Abnormal results

Abnormal results may show different characteristics of tissues within organs. Accumulations of blood or other fluids where they do not belong may be detected. Radiologists can differentiate among types of tumors throughout the body by viewing details of their makeup.

Sinus studies

The increasing availability and lowered cost of CT scanning has led to its increased use in sinus studies, either as a replacement for a sinus x ray or as a follow-up to an abnormal sinus radiograph. The sensitivity of CT allows for the location of areas of sinus infection, particularly chronic infection. Sinus tumors will show as shades of gray indicating the difference in their density from that of normal tissues in the area.

Brain studies

The precise differences in density allowed by CT scan can clearly show tumors, strokes, or lesions in the brain area as altered densities. These lighter or darker areas on the image may indicate a tumor or hematoma within the brain and skull area. Different types of tumors can be identified by the presence of edema (fluid), by the tissue's density, or by studying blood vessel location and activity. The speed and convenience of CT often allows for detection of hemorrhage (bleeding) before symptoms even occur.

Body scans

The body CT scan can identify abnormal body structures and organs. A CT scan may indicate tumors or cysts, enlarged lymph nodes, abnormal collections of fluids, blood, fat, or cancer metastasis. Tumors resulting from metastasis (movement of the cancer from the primary site of cancer growth to a distant site) are different in makeup than primary (original) tumors.

Chest scans

In addition to those findings which may indicate aortic aneurysms (rupture of the largest artery in the body), chest CT studies can show other problems in the heart and lungs, and distinguish between an aortic aneurysm and a tumor adjacent to the aorta. CT will not only show differences between air, water, tissues and bone, but will also assign numerical values to the various densities. Coin-sized lesions in the lungs may be indicative of tuberculosis or tumors. CT will help distinguish among the two. Enlarged lymph nodes in the chest area may indicate Hodgkin's disease (a blood disorder).

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Conduct disorder

Definition

Conduct disorder is a childhood behavior disorder characterized by aggressive and destructive activities that cause disruptions in the child's natural environments such as home, school, church, or the neighborhood. The overriding feature of conduct disorder is the repetitive and persistent pattern of behaviors that violate societal norms and the rights of other people. It is one of the most prevalent categories of mental health problems of children in the United States, with rates estimated at 9% for males and 2% for females.

Description

The specific behaviors used to produce a **diagnosis** of conduct disorder fall into four groups: aggressive conduct that causes or threatens physical harm to other people or animals, nonaggressive behavior that causes property loss or damage, deceitfulness or theft, and serious violations of rules. Two subtypes of conduct disorder can be delineated based on the age that symptoms first appear. Childhood-onset type is appropriate for children showing at least one of the behaviors in question before the age of 10. Adolescent onset type is defined by the absence of any conduct disorder criteria before the age of 10. Severity may be described as mild, moderate or severe, depending on the number of problems exhibited and their impact on other people.

Youngsters who show symptoms (most often aggression) before age 10 may also exhibit oppositional behavior and peer relationship problems. When they also show persistent conduct disorder and then develop adult **antisocial personality disorder**, they should be distinguished from individuals who had no symptoms of conduct disorder before age 10. The childhood type is more highly associated with heightened aggression, male gender, **oppositional defiant disorder**, and a family history of antisocial behavior.

The individual behaviors that can be observed when conduct disorder is diagnosed may be both common, problematic, and chronic. They tend to occur frequently and are distressingly consistent across time, settings, and families. Not surprisingly, these children function poorly in a variety of places. In fact, the behaviors clustered within the term "conduct disorder" account for a majority of clinical referrals, classroom detentions or other sanctions, being asked to stop participating in numerous activities, and can be extremely difficult (even impossible) for parents to manage.

The negative consequences of conduct disorder, particularly childhood onset, may include illicit drug use, dropping out of school, violent behavior, severe family conflict, and frequent delinquent acts. Such behaviors often result in the child's eventual placement out of the home, in special education and/or the juvenile justice system. There is evidence that the rates of disruptive behavior disorders may be as high as 50% in youth in public sectors of care such as juvenile justice, alcohol and drug

The overriding feature of conduct disorder is the repetitive and persistent pattern of behaviors that violate societal norms and the rights of other people. Youngsters with conduct disorder often exhibit aggressive behavior to other people (bullying, starting fights, etc.) or to animals. They may also damage others' property. *(Carolyn A. McKeone. Photo Researchers, Inc. Reproduced by permission.)*

services, schools for youths with serious emotional disturbances, child welfare, and mental health.

The financial costs of crime and correction for repeated juvenile offenses by youth with conduct disorder are extensive. The social costs include citizens' fear of such behavior, loss of a sense of safety, and disruptions in classrooms that interfere with other children's opportunity to learn. The costs to the child and his or her family are enormous in terms of the emotional and other resources needed to address the consequences of the constellation of symptoms that define conduct disorder.

Causes and symptoms

There is no known cause for conduct disorder. The frustrating behavior of youngsters with conduct disorder frequently leads to blaming, labeling, and other unproductive activities. Children who are "acting out" do not inspire sympathy or the benefit of the doubt. They are often ostracized by other children. Parents of such children are often blamed as poor disciplinarians or bad parents. As a result, parents of children with conduct disorder may be reluctant to engage with schools or other authorities. At the same time, there is a strong correlation between children diagnosed with conduct disorder and a significant level of family dysfunction, poor parenting practices, an overemphasis on coercion and hostile communication patterns, verbal and physical aggression and a history of maltreatment.

There is a suggestion of an, as yet, unidentified genetic component to what has generally been viewed as a behavioral disorder. One study with adopted children in the mid-1990s looked at the relationship between birth parents with antisocial personality disorder, and adverse adoptive home environments. When these two adverse conditions occurred, there was significantly increased aggressiveness and conduct disorder in the adopted children. That was not the case if there was no indication of antisocial personality disorder in the birth parents. This finding has important implications for prevention and **intervention** of conduct disorders and its associated conditions of substance abuse and aggressiveness.

Symptoms

The ***Diagnostic and Statistical Manual of Mental Disorders*** (also known as the *DSM-IV-TR*) indicates that for conduct disorder to be diagnosed, the patient has repeatedly violated rules, age-appropriate social norms and the rights of others for a period of at least twelve months. This is shown by three or more of the following behaviors, with at least one having taken place in the previous six months: aggression to people or animals, property destruction, lying or theft, and serious rule violations.

Aggression to people or animals includes:

- engaging in frequent bullying or threatening
- often starting fights
- using a weapon that could cause serious injury (gun, knife, club, broken glass)
- showing physical cruelty to people
- showing physical cruelty to animals
- engaging in theft with confrontation (armed robbery, extortion, mugging, purse snatching)
- forcing sex upon someone

Property destruction includes:

- deliberately setting fires to cause serious damage
- deliberately destroying the property of others by means other than fire setting

Lying or theft includes:

- breaking into building, car, or house belonging to someone else
- frequently lying or breaking promises for gain or to avoid obligations (called "conning")
- stealing valuables without confrontation (burglary, forgery, shop lifting)

Serious rule violations include:

- beginning before age 13, frequently staying out at night against parents' wishes

- running away from parents overnight twice or more or once if for an extended period
- engaging in frequent truancy beginning before the age of 13

Mild severity would mean there are few problems with conduct beyond those needed to make a diagnosis AND all of the problems cause little harm to other people. Moderate severity means the number and effect of the conduct problems is between the extremes of mild and severe. Severe is indicated if there are many more conduct symptoms than are needed to make the diagnosis (more than three in the previous twelve months or more than one in the previous six months), or, the behaviors cause other people considerable harm.

Diagnosis

Conduct disorder is generally diagnosed when somebody, often a child in school, comes to the attention of authorities (school, law enforcement, and others) most often because of behavior. The person might then be referred to a **psychiatrist** or **psychologist** for **assessment and diagnosis**. It is unlikely that any sort of specific test is given; rather, the individual would have to meet the criteria in the *DSM-IV-TR*. Usually there is a history of acting out in school, neighborhood, home, and other social settings. Court-ordered treatment would likely occur if the person comes to the attention of the police and if a crime is involved. A judge might order treatment as an alternative to jail, or before a sentence is served.

Treatments

Earlier treatments of youth with conduct disorder relied on legal processes to declare a child in need of supervision or treatment and thus able to be placed in residential settings established for this purpose. While residential placements may still be used, recent treatment models have relied less on such restrictive procedures. The increased visibility and sophistication of the consumer movement, comprised of families of children and youth with mental health disorders, is bringing pressure to bear on treatment providers to stop blaming families, stop removing children from their families for services, focus instead on strengths and assets in both the child and his or her family, and to use community-based interventions in several domains in which the child and family live.

Community-based interventions are sometimes called wrap-around services to describe the intention that they will be brought to the child's natural environment in a comprehensive and flexible way. The idea is to target a range of child, parent, family and social system factors associated with a child's behavioral problems. This approach has been successful in modifying antisocial behavior, rates of restrictive placement, and in reducing the cost of services.

Another treatment that has been used with some success is the *Child Cognitive Behavioral Treatment and Skills Training* which trains children with conduct disorder in anger-coping, peer coping, and problem-solving skills.

Parent Management Training and **family therapy** are also used to treat conduct disorder. Parents learn to apply behavioral principles effectively, how to play with their children, and how to teach and coach the child to use new skills.

Medication is sometimes used and may be effective in controlling aggression. Generally, a variety of treatment modes are used to address such a complex disorder. Severe antisocial behavior on the part of the child and adverse parenting practices may suggest that the family will stop treatment before it can be effective, or before meaningful change can result.

Prognosis

Early identification and appropriate and innovative treatment will improve the course of conduct disorder and possibly prevent a host of negative outcomes that are often a consequence of the behaviors associated with it. Unfortunately, the **stigma** of treatment and the undiagnosed problems of many parents are still significant enough that families whose children could benefit from treatment never find their way to a treatment setting. Instead their children come into contact with the juvenile and criminal justice system.

Prevention

Prognosis may best be improved by prevention of conduct disorder before it becomes so resistant to treatment. Research is being conducted on what early interventions hold the greatest promise. The research incorporates several components such as child tutoring, classroom intervention, peer training, social-cognitive skills training, parent training, and family problem-solving.

Other studies have included early parent or family interventions, school-based interventions and community interventions. Again, these include a variety of elements as suggested before, including parent training that includes education about normal child development, child problem-solving, and family communication skills training. Research is still needed to determine where and when to target specific preventive interventions.

See also Cognitive-behavioral therapy; Cognitive problem-solving skills training

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American Academy of Child and Adolescent Psychiatry. 3615 Wisconsin Ave. NW, Washington, DC 20016. <http://www.aacap.org>.

Federation of Families for Children's Mental Health. 1101 King St., Suite 420, Alexandria, VA 22314. <http://www.ffcmh.org>.

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KEY TERMS

Normed—Describes a process used in the developmental stages of a test instrument. The new test is first given to a cross-section of a population for which it is designed. The scores, placements, rankings, etc., of these persons then become the source for all future comparisons (norm group). When a new subject takes the test, his/her score, placement, ranking, etc., is determined based upon comparison with or deviation from the norm group.

Psychosomatic—Physical disorder originating in, or aggravated by, the psychic or emotional processes of the individual.

Conners' Rating Scales-Revised

Definition

Developed by C. Keith Conners, Ph.D., the Conners' Rating Scales-Revised (CRS-R) are paper and pencil screening questionnaires designed to be completed by parents and teachers to assist in evaluating children for **attention-deficit/hyperactivity disorder** (ADHD).

Purpose

The CSR-R is used as part of a comprehensive examination and are designed to be easily administered and scored. Both the long and short versions are tools to assist in determining whether children between the ages of three and 17 years might suffer from ADHD.

Precautions

Those who administer the CRS-R should have a good understanding of psychological testing and its limitations. Although the CRS-R can be readily administered and scored by a nonprofessional, the ultimate responsibility for interpretation lies with a seasoned professional. As with all psychological evaluation instruments, the CRS-R is not perfect. One runs the risk of obtaining false positives (incorrectly diagnosing the disorder) or false negatives (failing to identify the disorder). Therefore, the information obtained from completed forms should not be used in isolation. It should be one piece of a complex evaluation that includes a clinical interview with the child, other diagnostic measures such as a computerized continuous performance test, and patient self-report—for those old enough and with sufficient reading ability to do so.

Previous versions of the Conners' scales were criticized by those claiming disparity between results obtained in different ethnic groups. The most recent version should dispel this concern, since they were "normed" using data from more than 8,000 subjects crossing all cultural and ethnic boundaries. The technical

manual for CRS-R even contains separate normative information for specific ethnic groups. However, when age and sex are taken into account there were either no differences or insignificant differences. Statistically, a difference of two or three T-score points would be insignificant.

Description

The CRS-Rs are available in long and short versions for both parents and teachers. The long version for parents contains 80 items while the long version for teachers contains 59 items. The parents' short version contains 27 items and the teachers' short version has 28. The forms are multi-paged, and numbers circled on the front or back page are automatically transferred to a middle section for use by the clinician. The clinician transfers the circled scores into appropriate scales on the middle form and totals each scale at the bottom of the page. The parent version contains scales A through N. The teacher version is similar but lacks scale G (psychosomatic) contained on the parent version.

Results

After transferring the raw scores to the various scales and totaling them, the total of each scale (A–N) is transferred to another form designed to portray the results graphically. The clinician must be careful to transpose the raw scores to the correct age group column within each major scale. For example, column 1 is used for ages three to five, column 2 for ages six to eight, column 3 for ages nine to 11, etc. Each of these column scores can then be converted to a T-score. T-scores are standardized scores with a mean of 50 and a standard deviation of 10. These can be further converted to percentile scores as needed.

As a rule, T-scores above 60 are cause for concern and have interpretive value. Interpretable scores range from a low T-score of 61 (mildly atypical) to above 70 (markedly atypical). However, again, this information should not be used in isolation to make a **diagnosis**.

See also Attention-deficit/hyperactivity disorder

Resources

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Conners' Rating Scales-Revised Technical Manual. North Tonawanda, New York: Multi Health Systems, 2000.

ORGANIZATIONS

Center for Mental Health Services. Office of Consumer, Family, and Public Information, 5600 Fishers Lane, Room 15-105 Rockville, MD 20857. (301) 443-2792.

Children and Adults with Attention Deficit Disorders (CH.A.D.D.). 499 NW 70th Avenue, Suite 109, Plantation, FL 33317. (305) 587-3700. (800) 233-4050 <www.chadd.org>.

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Conversion disorder

Definition

Conversion disorder is defined by ***Diagnostic and Statistical Manual of Mental Disorders***, 4th Edition, Text Revision, also known as the *DSM-IV-TR,* as a mental disorder whose central feature is the appearance of symptoms affecting the patient's senses or voluntary movements that suggest a neurological or general medical disease or condition. Somatoform disorders are marked by persistent physical symptoms that cannot be fully explained by a medical condition, substance abuse, or other mental disorder, and seem to stem from psychological issues or conflicts. The *DSM-IV-TR* classifies conversion disorder as one of the somatoform disorders, first classified as a group of mental disorders by the *DSM III* in 1980. Other terms that are sometimes used for conversion disorder include pseudoneurologic syndrome, hysterical **neurosis**, and psychogenic disorder.

Conversion disorder is a major reason for visits to primary care practitioners. One study of health care utilization estimates that 25–72% of office visits to primary care doctors involve psychological distress that takes the form of somatic (physical) symptoms. Another study estimates that at least 10% of all medical treatments and diagnostic services are ordered for patients with no evidence of organic disease. Conversion disorder carries a high economic price tag. Patients who convert their emotional problems into physical symptoms spend nine times as much for health care as people who do not somatosize; and 82% of adults with conversion disorder stop working because of their symptoms. The annual bill for conversion disorder in the United States comes to $20 billion, not counting absenteeism from work and disability payments.

Description

Conversion disorder has a complicated history that helps to explain the number of different names for it. Two eminent neurologists of the nineteenth century, Jean-Martin Charcot in Paris and Josef Breuer in Vienna were investigating what was then called hysteria, a disorder primarily affecting women (the term "hysteria" comes from the Greek word for uterus or womb). Women diagnosed with hysteria had frequent emotional outbursts and

KEY TERMS

Aphonia—Inability to speak caused by a functional disturbance of the voice box or vocal cords.

(la) Belle indifférence—A psychiatric symptom sometimes found in patients with conversion disorder, in which the patient shows a surprising lack of concern about the nature or implications of his/her physical symptom.

Conversion—In psychiatry, a process in which a repressed feeling, impulse, thought, or memory emerges in the form of a bodily symptom.

Diplopia—A disorder of vision in which a single object appears double. Diplopia is sometimes called double vision.

Dyskinesia—Difficulty in performing voluntary muscular movements.

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

Electroencephalogram—(EEG) A test that measures the electrical activity of the brain by means of electrodes placed on the scalp or on or in the brain itself.

Factitious disorder—A type of mental disturbance in which patients intentionally act physically or mentally ill without obvious benefits. It is distinguished from malingering by the absence of an obvious motive, and from conversion disorder by intentional production of symptoms.

Hysteria—In nineteenth-century psychiatric use, a neurotic disorder characterized by violent emotional outbursts and disturbances of the sensory and motor (movement-related) functions. The term "hysterical neurosis" is still used by some psychiatrists as a synonym for conversion disorder.

Malingering—Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

Myoclonus—An abrupt spasm or twitching in a muscle or group of muscles.

Narcotherapy—A form of psychotherapy that involves the administration of a drug that makes the patient drowsy.

Primary gain—In psychiatry, the principal psychological reason for the development of a patient's symptoms. In conversion disorder, the primary gain from the symptom is the reduction of anxiety and the exclusion of an inner conflict from conscious awareness.

Pseudoseizure—A fit that resembles an epileptic seizure but is not associated with abnormal electrical discharges in the patient's brain.

Psychogenic—Originating in the mind, or in a mental process or condition. The term "psychogenic" is sometimes used as a synonym for "conversion."

Ptosis—Drooping of the upper eyelid.

Secondary gain—A term that refers to other benefits that a patient obtains from a conversion symptom. For example, a patient's loss of function in an arm might require other family members to do the patient's share of household chores; or they might give the patient more attention and sympathy than he or she usually receives.

Shaman—In certain indigenous tribes or groups, a person who acts as an intermediary between the natural and supernatural worlds. Shamans are regarded as having the power or ability to cure illnesses.

Social modeling—A process of learning behavioral and emotional response patterns from observing one's parents or other adults. Some researchers think that social modeling plays a part in the development of conversion disorder in children.

Somatoform disorders—A group of psychiatric disorders in the *DSM-IV-TR* classification that are characterized by external physical symptoms or complaints that are related to psychological problems rather than organic illness. Conversion disorder is classified as a somatoform disorder.

Stressor—A stimulus or event that provokes a stress response in an organism. Stressors can be categorized as acute or chronic, and as external or internal to the organism.

a variety of neurologic symptoms, including paralysis, fainting spells, convulsions, and temporary loss of sight or hearing. Pierre Janet (one of Charcot's students), and Breuer independently came to the same conclusion about the cause of hysteria—that it resulted from psychological trauma. Janet, in fact, coined the term "dissociation" to describe the altered state of consciousness experienced by many patients who were diagnosed with hysteria.

The next stage in the study of conversion disorder was research into the causes of "combat neurosis" in World War I (1914-1918) and World War II (1939-1945). Many of the symptoms observed in "shell-shocked" soldiers were identical to those of "hysterical" women. Two of the techniques still used in the treatment of conversion disorder—hypnosis and narcotherapy—were introduced as therapies for combat veterans. The various terms used by successive editions of the *DSM* and the *ICD* (the European equivalent of *DSM*) for conversion disorder reflect its association with hysteria and dissociation. The first edition of the *DSM* (1952) used the term "conversion reaction." *DSM-II* (1968) called the disorder "hysterical neurosis (conversion type)," *DSM-III* (1980), *DSM-III-R* (1987), and *DSM-IV* (1994) have all used the term "conversion disorder." ICD-10 refers to it as "dissociative (conversion) disorder."

DSM-IV-TR (2000) specifies six criteria for the **diagnosis** of conversion disorder. They are:

- The patient has one or more symptoms or deficits affecting the senses or voluntary movement that suggest a neurological or general medical disorder.
- The onset or worsening of the symptoms was preceded by conflicts or stressors in the patient's life.
- The symptom is not faked or produced intentionally.
- The symptom cannot be fully explained as the result of a general medical disorder, substance intake, or a behavior related to the patient's culture.
- The symptom is severe enough to interfere with the patient's schooling, employment, or social relationships, or is serious enough to require a medical evaluation.
- The symptom is not limited to pain or sexual dysfunction, does not occur only in the context of **somatization disorder**, and is not better accounted for by another mental disorder.

DSM-IV lists four subtypes of conversion disorder: conversion disorder with motor symptom or deficit; with sensory symptom or deficit; with **seizures** or convulsions; and with mixed presentation.

Although conversion disorder is most commonly found in individuals, it sometimes occurs in groups. One such instance occurred in 1997 in a group of three young men and six adolescent women of the Embera, an indigenous tribe in Colombia. The young people believed that they had been put under a spell or curse, and developed dissociative symptoms that were not helped by antipsychotic medications or traditional herbal remedies. They were cured when shamans from their ethnic group came to visit them. The episode was attributed to psychological **stress** resulting from rapid cultural change.

Another example of group conversion disorder occurred in Iran in 1992. Ten girls out of a classroom of 26 became unable to walk or move normally following tetanus inoculations. Although the local physicians were able to treat the girls successfully, public health programs to immunize people against tetanus suffered an immediate negative impact. One explanation of group conversion disorder is that an individual who is susceptible to the disorder is typically more affected by suggestion and easier to hypnotize than the average person.

Causes and symptoms

Causes

The immediate cause of conversion disorder is a stressful event or situation that leads the patient to develop bodily symptoms as symbolic expressions of a long-standing psychological conflict or problem. One **psychiatrist** has defined the symptoms as "a code that conceals the message from the sender as well as from the receiver.".

Two terms that are used in connection with the causes of conversion disorder are primary gain and secondary gain. Primary gain refers to the lessening of the anxiety and communication of the unconscious wish that the patient derives from the symptom(s). Secondary gain refers to the interference with daily tasks, removal from the uncomfortable situation, or increased attention from significant others that the patient obtains as a result of the symptom(s).

Physical, emotional, or sexual abuse can be a contributing cause of conversion disorder in both adults and children. In a study of 34 children who developed pseudoseizures, 32% had a history of depression or sexual abuse, and 44% had recently experienced a parental divorce, death, or violent quarrel. In the adult population, conversion disorder may be associated with mobbing, a term that originated among European psychiatrists and industrial psychologists to describe psychological abuse in the workplace. One American woman who quit her job because of mobbing was unable to walk for several months. Adult males sometimes develop conversion disorder during military basic training. Conversion disorder may also develop in adults as a long-delayed after-effect of childhood abuse. A team of surgeons reported on the case of a patient who went into a psychogenic coma following a throat operation. The surgeons found that she had been repeatedly raped as a child by her father, who stifled her cries by smothering her with a pillow.

Symptoms

In general, symptoms of conversion disorder are not under the patient's conscious control, and are fre-

quently mysterious and frightening to the patient. The symptoms usually have an acute onset, but sometimes worsen gradually.

The most frequent forms of conversion disorder in Western countries include:

- Pseudoparalysis. In pseudoparalysis, the patient loses the use of half of his/her body or of a single limb. The weakness does not follow anatomical patterns and is often inconsistent upon repeat examination.
- Pseudosensory syndromes. Patients with these syndromes often complain of numbness or lack of sensation in various parts of their bodies. The loss of sensation typically follows the patient's notion of their anatomy, rather than known characteristics of the human nervous system.
- Pseudoseizures. These are the most difficult symptoms of conversion disorder to distinguish from their organic equivalents. Between 5% and 35% of patients with pseudoseizures also have epilepsy. Electroencephalograms (EEGs) or measurement of serum prolactin levels, are useful in distinguishing pseudoseizures from epileptic seizures.
- Pseudocoma. Pseudocoma is also difficult to diagnose. Because true coma may indicate a life-threatening condition, patients must be given standard treatments for coma until the diagnosis can be established.
- Psychogenic **movement disorders**. These can mimic myoclonus, parkinsonism, dystonia, dyskinesia, and tremor. Doctors sometimes give patients with suspected psychogenic movement disorders a placebo medication to determine whether the movements are psychogenic or the result of an organic disorder.
- Pseudoblindness. Pseudoblindness is one of the most common forms of conversion disorder related to vision. Placing a mirror in front of the patient and tilting it from side to side can often be used to determine pseudoblindness, because humans tend to follow the reflection of their eyes.
- Pseudodiplopia. Pseudodiplopia, or seeing double, can usually be diagnosed by examining the patient's eyes.
- Pseudoptosis. Ptosis, or drooping of the upper eyelid, is a common symptom of myasthenia gravis and a few other disorders. Some people can cause their eyelids to droop voluntarily with practice. The diagnosis can be made on the basis of the eyebrow; in true ptosis, the eyebrows are lifted, whereas in pseudoptosis they are lowered.
- Hysterical aphonia. Aphonia refers to loss of the ability to produce sounds. In hysterical aphonia, the patient's cough and whisper are normal, and examination of the throat reveals normal movement of the vocal cords.

Psychiatrists working in various parts of the Middle East and Asia report that the symptoms of conversion disorder as listed by *DSM-IV* and ICD-10 do not fit well with the symptoms of the disorder most frequently encountered in their patient populations.

Demographics

The lifetime prevalence rates of conversion disorder in the general U.S. population are estimated to fall between 11 and 300 per 100,000 people. The differences in the estimates reflect differences in the method of diagnosis as well as some regional population differences. In terms of clinical populations, conversion disorder is diagnosed in 5%–14% of general hospital patients; 1%–3% of outpatient referrals to psychiatrists; and 5%–25% of psychiatric outpatients.

Among adults, women diagnosed with conversion disorder outnumber men by a 2:1 to 10:1 ratio; among children, however, the gender ratio is closer to 1:1. Less educated people and those of lower socioeconomic status are more likely to develop conversion disorder; race by itself does not appear to be a factor. There is, however, a major difference between the populations of developing and developed countries; in developing countries, the prevalence of conversion disorder may run as high as 31%.

Diagnosis

Conversion disorder is one of the few mental disorders that appears to be overdiagnosed, particularly in emergency departments. There are numerous instances of serious neurologic illness that were initially misdiagnosed as conversion disorder. Newer techniques of diagnostic imaging have helped to lower the rate of medical errors.

Diagnostic issues

Diagnosis of conversion disorder is complicated by its coexistence with physical illness in as many as 60% of patients. Alternatively explained, a diagnosis of conversion disorder does not exclude the possibility of a concurrent organic disease. The examining doctor will usually order a mental health evaluation when conversion disorder is suspected, as well as x rays, other **imaging studies** that may be useful, and appropriate laboratory tests. The doctor will also take a thorough patient history that will include the presence of recent stressors in the patient's life, as well as a history of abuse. Children and adolescents are usually asked about their school experiences; one question they are asked is whether a recent

change of school or an experience related to school may have intensified academic pressure.

In addition, there are a number of bedside tests that doctors can use to distinguish between symptoms of conversion disorder and symptoms caused by physical diseases. These may include the drop test, in which a "paralyzed" arm is dropped over the patient's face. In conversion disorder, the arm will not strike the face. Other tests include applying a mildly painful stimulus to a "weak" or "numb" part of the body. The patient's pulse rate will typically rise in cases of conversion disorder, and he or she will usually pull back the limb that is being touched.

Factors suggesting a diagnosis of conversion disorder

The doctor can also use a list of factors known to be associated with conversion disorder to assess the likelihood that a specific patient may have the disorder:

- Age. Conversion disorder is rarely seen in children younger than six years or adults over 35 years.
- Sex. The female to male ratio for the disorder ranges between 2:1 and 10:1. It is thought that higher rates of conversion disorder in women may reflect the greater vulnerability of females to abuse.
- Residence. People who live in rural areas are more likely to develop conversion disorder than those who live in cities.
- Level of education. Conversion disorder occurs less often among sophisticated or highly educated people.
- Family history. Children sometimes develop conversion disorder from observing their parents' reactions to stressors. This process is known as social **modeling**.
- A recent stressful change or event in the patient's life.

An additional feature suggesting conversion disorder is the presence of *la belle indifférence*. The French phrase refers to an attitude of relative unconcern on the patient's part about the symptoms or their implications. *La belle indifférence* is, however, much more common in adults with conversion disorder than in children or adolescents. Patients in these younger age groups are much more likely to react to their symptoms with fear or hopelessness.

Medical conditions that mimic conversion symptoms

It is important for the doctor to rule out serious medical disorders in patients who appear to have conversion symptoms. The following disorders must be considered in the differential diagnosis:

- multiple sclerosis (blindness resulting from optic neuritis)
- myasthenia gravis (muscle weakness)
- periodic paralysis (muscle weakness)
- myopathies (muscle weakness)
- polymyositis (muscle weakness)
- Guillain-Barré syndrome (motor and sensory symptoms)

Treatments

Patients diagnosed with conversion disorder frequently benefit from a team approach to treatment and from a combination of treatment modalities. A team approach is particularly beneficial if the patient has a history of abuse, or if he or she is being treated for a concurrent physical condition or illness.

Medications

While there are no drugs for the direct treatment of conversion disorder, medications are sometimes given to patients to treat the anxiety or depression that may be associated with conversion disorder.

Psychotherapy

Psychodynamic psychotherapy is sometimes used with children and adolescents to help them gain insight into their symptoms. Cognitive behavioral approaches have also been tried, with good results. **Family therapy** is often recommended for younger patients whose symptoms may be related to family dysfunction. **Group therapy** appears to be particularly useful in helping adolescents to learn social skills and coping strategies, and to decrease their dependency on their families.

Inpatient treatment

Hospitalization is sometimes recommended for children with conversion disorders who are not helped by outpatient treatment. Inpatient treatment also allows for a more complete assessment of possible coexisting organic disorders, and for the child to improve his or her level of functioning outside of an abusive or otherwise dysfunctional home environment.

Alternative and complementary therapies

Alternative and complementary therapies that have been shown to be helpful in the treatment of conversion disorder include hypnosis, relaxation techniques, visualization, and **biofeedback**.

Prognosis

The prognosis for recovery from conversion disorder is highly favorable. Patients who have clearly identifiable

stressors in their lives, acute onset of symptoms, and a short interval between symptom onset and treatment, have the best prognosis. Of patients hospitalized for the disorder, over half recover within two weeks. Between 20% and 25% will relapse within a year. The individual symptoms of conversion disorder are usually self-limited and do not lead to lasting disabilities; however, patients with hysterical aphonia, paralysis, or visual disturbances, have better prognoses for full recovery than those with tremor or pseudoseizures.

Prevention

The incidence of conversion disorder in adults is likely to continue to decline with rising levels of formal education and the spread of basic information about human psychology. Prevention of conversion disorder in children and adolescents depends on better strategies for preventing abuse.

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American Academy of Child and Adolescent Psychiatry. 3615 Wisconsin Avenue, NW, Washington, DC 20016-3007. (202) 966-7300. Fax: (202) 966-2891. <www.aacap.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

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Co-occurring disorders *see* **Dual diagnosis**

Couples therapy

Definition

Couples therapy is a form of psychological therapy used to treat relationship distress for both individuals and couples.

Purpose

The purpose of couples therapy is to restore a better level of functioning in couples who experience relationship distress. The reasons for distress can include poor communication skills, incompatibility, or a broad spectrum of psychological disorders that include domestic violence, alcoholism, depression, anxiety, and **schizophrenia**. The focus of couples therapy is to identify the presence of dissatisfaction and distress in the relationship, and to devise and implement a treatment plan with objectives designed to improve or alleviate the presenting symptoms and restore the relationship to a better and healthier level of functioning. Couples therapy can assist persons who are having complaints of intimacy, sexual, and communication difficulties.

Precautions

Couples who seek treatment should consult for services from a mental health practitioner who specializes in this area.

Patients should be advised that honesty, providing all necessary information, cooperation, keeping appointments on time, and a sincere desire for change and improvement are all imperative to increase the chance of successful outcome. Additionally, a willingness to work "towards" and "with" the process of treatment is essential.

Description

Couples therapy sessions differ according to the chosen model, or philosophy behind the therapy. There are several models for treating couples with relationship difficulties. These commonly utilized strategies include psychoanalytic couples therapy, object relations couple therapy, ego analytical couples therapy, behavioral couples therapy, integrative behavioral couples therapy, and cognitive behavioral couples therapy.

Psychoanalytical couples therapy

Psychoanalytic therapy attempts to uncover unresolved childhood conflicts with parental figures and how these behaviors are part of the current relationship problems. The psychoanalytic approach tends to develop an understanding of interpersonal interactions (at present) in connection with early development. The success in development of early stages dictates the future behavior of interpersonal relationships. The essential core of this model deals with the process of separation and individuation (becoming a separate, distinct self) from mother-child interactions during childhood. A critical part of this model is introjection. The process of introjection includes introjects (infant processing versions) of the love object (mother). The developmental process of introjection forms the basis an unconscious representation of others (objects) and is vital for development of a separate and defined sense of self. The psychoanalytic approach analyzes marital relations and mate selection as originating from parent-child relationship during developmental stages of the child.

Object relations couple therapy

The object relations model creates an environment of neutrality and impartiality to understand the distortions and intrapsychic (internalized) conflicts that each partner contributes to the relationship in the form of dysfunctional behaviors. This model proposes that there is a complementary personality fit between couples that is unconscious and fulfills certain needs. This model supports the thought that a "mothering figure" is the central motivation for selection and attachment of a mate. Choosing a "mothering" figure induces further repression (non-development) of portions of personality that were not well-developed (referred to as "lost parts"). This repression causes relationship difficulties.

Ego analytic couples therapy

Ego analytical approaches utilize methods to foster the ability to communicate important feelings in the couple's relationship. This model proposes that dysfunction originates from the patient's incapabilities to recognize intolerance and invalidation of sensitivities and problems in a relationship. According to this model, there are two major categories of problems. The first category of problems relates to dysfunction brought into the relationship from early childhood trauma and experiences. The sec-

The focus of couples therapy is to identify the presence of dissatisfaction and distress in the relationship, and to devise and implement a treatment plan. The objectives of treatment are to improve or alleviate the symptoms and restore the relationship to a healthier level of functioning. *(Aaron Haupt. Photo Researchers, Inc. Reproduced by permission.)*

ond involves the patient's reaction to difficulties and a sense of unentitlement (a personal feeling that one does not deserve something). A patient's shame and guilt are major factors precipitating the thoughts of unentitlement.

Behavioral marital therapy

Behavioral marital therapists tend to improve relationships between a couple by increasing positive exchanges and decreasing the frequency of negative and punishing interactions. This model focuses on the influence that environment has in creating and maintaining relationship behavior. Behavior exchange between partners is flowing continuously and prior histories can affect relationship interactions. Behavior therapy in general is based on the idea that when certain behaviors are rewarded, they are reinforced. The amount of rewards (positive reinforcers) received in relation to the amount of aversive behavior is linked to an individual's sense of relationship dissatisfaction.

Integrative behavioral couples therapy

Integrative behaviorists help couples by improving behavior exchange, communication, and the couples' abilities for problem-solving skills. The integrative behavioral therapy approach examines functioning of the couple and is more flexible and individualized to specific problems in the relationship than behavior marital therapy. This approach examines problems and interactions that are repetitious (acts that are repeatedly done causing relationship problems).

Cognitive behavior marital therapy

The cognitive approach therapist educates and increases awareness concerning perceptions, assumptions, attributions or standards of interaction between the couple. The central theme for understanding marital discourse using cognitive behavioral therapy is based on the behavioral marital therapy model. A couple's emotional and behavioral dysfunction are related to inappropriate information processing (possibly "jumping to conclusions," for example) and negative cognitive appraisals. This models attempts to discover the negative types of thinking that drive negative behaviors that cause relationship distress.

Emotionally focused therapy

Emotionally focused therapy assists patients to acknowledge, assess, and express emotions that are related to distress. This model views emotion and cognition (thinking) as interdependent and that emotion is a primary "driver" of interpersonal expression. The primary theme of emotionally focused therapy is that couple distress stems from unexpressed and unacknowledged emotional needs. The dysfunction arises from negative interactions from emotions that have been withheld from disclosure from each partner.

Structural strategic marital therapy

Structural strategic therapists will challenge existing negative perceptions and present alternative possibilities and behaviors. These alternate behaviors encourage positive perceptions by role-playing. This model views relationship progression in developmental stages. According to this model, the couple's distress reflects difficulties in coping mechanisms related to life changes— either environmental or personal change. Despite relationship dissatisfaction, the couple will tend to resist change, maintaining status quo, and attempting baseline functioning to keep the system going.

Preparation

Couples should be informed that cooperation is vital for the process. Couples should have a desire to modify and/or change dysfunctional behaviors. Honesty and emotional openness is a necessary component for treatment. Results cannot be guaranteed. The psychotherapist would typically provide an extensive assessment process during the initial appointment. This couples assessment process usually includes in-depth information gathering concerning the presenting problem, and assessment of occupations, schooling, employment, childhood development, parental history, sub-

stance abuse, religion, relational, medical, legal, and past psychological history, in the form an interview. The psychotherapist can then devise the best course of treatment planning. Further psychological tests and measurements may be indicated initially or as the need arises during the treatment process.

Aftercare

Treatment usually takes several months or longer. Once the couple has developed adequate skills and has displayed an improved level of functioning that is satisfactory to both, then treatment can be terminated. An awareness of relapse prevention behaviors and relapsing behaviors is important. (Relapsing behaviors refer to the return to the behaviors that the couple is trying to change or eliminate.) Patients are encouraged to return to treatment if relapse symptoms appear. Follow-up visits and long-term psychological therapy can be arranged between parties if this is mutually decided as necessary and beneficial.

Risks

The major risk of couples therapy is lack of improvement or return to dysfunctional behaviors. These tend not to occur unless there is a breakdown in skills learned and developed during treatment, or a person is resistant to long-term change.

Normal results

A normal progression of couples therapy is relief from symptomatic behaviors that cause marital discourse, distress, and difficulties. The couple is restored to healthier interactions and behaviors are adjusted to produce a happier balance of mutually appropriate interactions. Patients who are sincere and reasonable with a willingness to change tend to produce better outcomes. Patients usually develop skills and increased awareness that promotes healthier relationship interactions.

Abnormal results

There are no known abnormal results from couples therapy. At worst, patients do not get better because they cannot break away from self-induced, self-defeating behaviors that precipitate marital dysfunction and distress. The problems are not worsened if treatment is provided by a trained mental health practitioner in this specialty.

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The American Association for Marriage and Family Therapy. 1133 15th Street NW, Suite 30, Washington, DC 20005. Phone: (202) 452-0109. Fax: (202) 223-2329. Web site: <http:/www.aamft.org>.

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Covert sensitization

Definition

Covert sensitization is a form of behavior therapy in which an undesirable behavior is paired with an unpleasant image in order to eliminate that behavior.

Purpose

As with other **behavior modification** therapies, covert sensitization is a treatment grounded in learning theory—one of the basic tenets being that all behavior is learned and that undesirable behaviors can be unlearned under the right circumstances. Covert sensitization is one of a group of behavior therapy procedures classified as covert conditioning, in which an aversive stimulus in the form of a nausea- or anxiety-producing image is paired with an undesirable behavior to change that behavior. It is best understood as a mixture of both the classical and the operant conditioning categories of learning. Based on research begun in the 1960s, psychologists Joseph Cautela and Albert Kearney published the 1986 classic *The Covert Conditioning Handbook*, which remains a definitive treatise on the subject.

The goal of covert sensitization is to directly eliminate the undesirable behavior itself, unlike insight-oriented psychotherapies that focus on uncovering unconscious motives in order to produce change. The behaviors tar-

KEY TERMS

Covert—Concealed, hidden, or disguised.

Operant—Conditioning in which the desired response is reinforced by an introduced stimulus.

Sensitization—To make sensitive or susceptible.

geted for modification are often referred to as "maladaptive approach behaviors," which includes behaviors such as alcohol abuse, drug abuse, and smoking, pathological gambling, overeating, sexual deviations, and sexually based nuisance behaviors such as obscene phone calling. The type of behavior to be changed and the characteristics of the aversive imagery to be used influence the treatment, which is usually administered in an outpatient setting either by itself or as a component of a multimodal program. Self-administered homework assignments are almost always part of the treatment package. Some therapists incorporate covert sensitization with hypnosis in the belief that outcome is enhanced.

Description

The patient being treated with covert sensitization can expect a fairly standard set of procedures. The therapist begins by assessing the problem behavior, and will most likely measure frequency, severity, and the environment in which it occurs. Depending upon the type of behavior to be changed, some therapists may also take treatment measures before, during, and after physiological arousal (such as heart rate) to better assess treatment impact. Although the therapeutic relationship is not the focus of treatment, the behavior therapist believes that good rapport will facilitate a more successful outcome and strives to establish positive but realistic expectations. Also, a positive relationship is necessary to establish patient confidence in the rationale for exposure to the discomfort of unpleasant images.

The therapist will explain the treatment rationale and protocol. Patient understanding and consent are important, since, by intention, he or she will be asked to experience images that arouse unpleasant and uncomfortable physical and psychological associations. The therapist and patient collaborate in creating a list of aversive images uniquely meaningful to the patient that will be applied in the treatment. Standard aversive images include vomiting, snakes, spiders, vermin, and embarrassing social consequences. An aversive image is then selected appropriate to the target problem behavior. Usually, the image with the most powerful aversive response is chosen. The patient is instructed on how to relax—an important precursor to generating intense imagery. The patient is then asked to relax and imagine approaching the situation where the undesirable behavior occurs (for example, purchasing donuts prior to overeating).

If the patient has a difficult time imagining the scene, the image may be presented verbally by the therapist. As the patient imagines getting closer to the situation (donut store), he or she is asked to clearly imagine an unpleasant consequence (such as vomiting) just before indulging in the undesirable behavior (purchasing donuts and overeating). The scene must be imagined with sufficient vividness so that a sense of physiological discomfort or high anxiety is actually experienced. Then the patient imagines leaving the situation and experiencing considerable relief. The patient learns to associate unpleasant sensations (nausea and vomiting) with the undesirable behavior, leading to decreased desire and avoidance of the situation in the future. An alternative behavior incompatible with the problem behavior may be recommended (eat fruit when hungry for a donut).

The patient is given the behavioral homework assignment to practice self-administering the treatment. The patient is told to alternate the aversive scenes with scenes of self-controlled restraint in which he or she rejects the undesirable behavior before indulging in it, thus avoiding the aversive stimulus. The procedure is practiced several times with the therapist in the office, and the patient practices the procedure ten to 20 times during each home session between office sessions. The patient is then asked to practice in the actual situation, imagining the aversive consequences and avoiding the situation. With much variation, and depending upon the nature of the behavior targeted for change, the patient may see the therapist anywhere from five to 20 sessions over a period of a few weeks to several months. The treatment goal is to eliminate the undesirable behavior altogether.

Aftercare

Patients completing covert sensitization treatment are likely to be asked by the therapist to return periodically over the following six to twelve months or longer, for booster sessions to prevent relapse.

Risks

Covert sensitization is comparatively risk-free. This is in contrast to the medical and ethical concerns raised by some other aversive procedures such as **aversion therapy**, in which potent chemical and pharmacological stimulants may be used as aversants.

Normal results

Depending upon the objectives established at the beginning of treatment, patients successfully completing covert sensitization might expect to stop the undesirable behavior. And, if they practice relapse prevention techniques, they can expect to maintain the improvement. Although this treatment may appear to be relatively simple, it has been found to be quite effective for treating many problem behaviors.

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ORGANIZATIONS

Association for Advancement of Behavior Therapy. 305 Seventh Ave.—16th Floor, New York, NY 10001-6008. <http://www.aabt.org>.

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KEY TERMS

Genogram—A family tree diagram that represents the names, birth order, sex, and relationships of the members of a family. Therapists use genograms to detect recurrent patterns in the family history and to help the members understand their problem(s).

Journaling—Involves writing out thoughts and feelings in an unstructured format. A "stream of consciousness" approach (writing whatever comes to mind) is suggested for greatest effectiveness.

Psychodrama—A specific form of role play that focuses on acting out "scripts" of unresolved issues within the family, or helping family members adopt new approaches and understanding of one another.

Role-playing—Involves adopting the role of other family members, oneself, or significant persons within the life of the individual and acting out various life situations in order to explore the relationships of those involved.

Creative therapies

Definition

Creative therapy refers to a group of techniques that are expressive and creative in nature. The aim of creative therapies is to help clients find a form of expression beyond words or traditional therapy, such as cognitive or **psychotherapy**. Therefore, the scope of creative therapy is as limitless as the imagination in finding appropriate modes of expression. The most commonly used and professionally supported approaches include art therapy, writing, sand play, clay, movement therapy, psychodrama, role play, and music therapy.

Purpose

Creative therapy includes techniques that can be used for self-expression and personal growth when the client is unable to participate in traditional "talk therapy," or when that approach has become ineffective. Appropriate clients include children, individuals who are unable to speak due to **stroke** or **dementia**, or people who are dealing with clinical issues that are hidden within the subconscious, beyond the reach of language. The latter often occurs when the focus is on trauma or **abuse** that may have occurred before the client was able to speak, or in families where there is a strict code against talking about feelings or "negative" things. Creative therapy is also effective when used to explore fears around medical issues, such as cancer or HIV.

Precautions

Caution is indicated when strong emotions become overwhelming, thus debilitating the client. Possible indications for caution include the presence of flashbacks, panic attacks, recently revealed trauma or abuse, and vivid and realistic nightmares. Other indications for caution include individual characteristics, such as a tendency toward overly emotional responses, difficulty managing change or surprises, and poor coping skills. Therapists should also take care with patients with **psychosis** or **borderline personality disorder**.

Description

Visually expressive forms of creative therapy include drawing, painting, and modeling with clay. The goal is to provide a medium for expression that bypasses

Writing in a journal can relieve stress and can be practiced on a regular basis by writing down whatever comes to mind, or it can be used for specific problem areas, such as focusing attention on goals or on unresolved feelings of grief or anger. *(Jeff Greenberg. Photo Researchers, Inc. Reproduced by permission.)*

words, thus helping the individual connect with emotions about various personal experiences. The scope of the drawings is limited only by the imagination of the individual and by the creativity of the therapists. This technique can often be continued by clients on their own after beginning the work in session.

Movement and music therapies are often used in conjunction with relaxation approaches. Movement therapy involves dance and the interpretation of feelings or thoughts into movement, and is often set to music. For teens in particular, music and movement are often healthy releases for **stress** and emotions. These therapies can also help people develop appropriate coping skills. Movement and music may be used in nursing homes, gym class, residential treatment centers, a therapist's office, or a home.

Journaling techniques have been studied extensively regarding their health benefits, both physical and emotional. Its application is broad and it can be used in various therapeutic approaches. Journaling can be used on a regular basis for stress relief by writing down whatever comes to mind, or it can be used for specific problem areas, such as focusing attention on goals or on unresolved feelings of **grief** or anger. In journaling, it seems to be more important to focus on emotional aspects, rather than using it to simply record daily events.

Other techniques include sand play, pet therapy, **play therapy**, and horticulture therapy. Sand play is a specialized form of play therapy in which sand is used to form designs or set up stories using play figures. Play therapy is an approach used with children, and is quite extensive in background theory and application. It is a psychological therapy in which the child plays in the therapist's presence. The therapist then uses a child's fantasies and the symbolic meanings of his or her play as a medium for understanding and communicating with the child. Pet therapy and horticulture therapy are often used in hospitals and residential treatment centers. Although these therapies are not expressive in the same way as other approaches, they offer a different experience for the individuals participating in them—helping people feel a sense of joy, connection, or accomplishment that may be missing from their lives.

Preparation

Little preparation is needed for the visually expressive forms. Drawing is often used in a first session with young children. When used with adults, drawing or painting is often helpful, especially at a time of impasse when "talk therapy" is not effective, or when focusing on more emotional aspects of the therapeutic work.

Role-playing requires the review of specific family roles to determine goals for the work. If the family work is focused on communication, each member may be asked to adopt the role of another family member to clarify their perceptions of current roles for themselves and the other family members. The purpose of adopting these roles is to gain insight and understanding about the other person's perspective in terms of their thoughts, feelings, and actions. Taking on the role of another helps to build empathy and provide a mechanism for personal growth and change.

A genogram or diagram of family members is sometimes helpful as a guide in identifying specific roles and directing the drama.

Aftercare

For most of the creative therapy techniques, aftercare will largely be maintained by the individual client, unless the individual is participating in a support group or ongoing therapy. One advantage of creative therapy is the ease of implementation. Little special equipment is needed, and many of the techniques easily lend themselves to use in the home. If an individual is participating in a support group or individual therapy following a **hospitalization**, the techniques can be maintained as part of those activities.

Self-portrait done by a girl while in treatment for anorexia nervosa. *(Susan Rosenberg, Science Photo Library/Photo Researchers, Inc. Reproduced by permission.)*

Risks

Risks occur when the client is exposed to intense emotional material or memories before the necessary preparatory work has been completed in therapy. Such negative reactions may include a psychotic break, or a need for hospitalization, although this is a rare occurrence.

A more likely risk is that of altering existing family relationships. Working through certain issues surrounding trauma or abuse may alter the participant's feelings or thoughts about significant people in his or her life. Conflicted feelings about these individuals may arise as recognition of certain patterns or behavior become apparent to the client. The increased awareness and insight may make it impossible for the client to continue some relationships. The resulting conflict may be uncomfortable for the client.

Normal results

Typical results include increased awareness, the release of suppressed emotions, a general lifting of depressive feelings, increased energy, and the resolution of internal conflict. Ongoing health benefits, such as lowered blood pressure, may result from decreased stress and improved coping skills. A greater sense of self-acceptance and decreased agitation are often experienced by clients.

Abnormal results

Unusual results include increasingly intense feelings of agitation and stress. For some individuals, the techniques may appear to have no benefits. It is recommended that these individuals seek clinical help.

See also Support groups; Grief counseling

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Arts in Therapy Network. <www.artsintherapy.com>.

Deanna Pledge, Ph.D.

Crisis housing

Definition

Crisis housing (or crisis residential services) are supervised short-term residential alternatives to **hospitalization** for adults with serious mental illnesses or children with serious emotional or behavioral disturbances.

Purpose

The course of most serious mental illness (such as **schizophrenia**, **bipolar disorder**, severe depression, and **borderline personality disorder**) is cyclical, typically characterized by periods of relative well-being, interrupted by periods of deterioration or relapse. When relapse occurs, the individual generally exhibits florid symptoms that require immediate psychiatric attention and treatment. More often than not, relapse is caused by the individual's failure to comply with a prescribed medication regimen (not taking medication regularly, not taking the amount or dose prescribed, or not taking it all). Relapse can also be triggered during periods of great **stress** or can even occur spontaneously, without any marked changes in lifestyle or medication regimen. When these crises recur, the goal of treatment is to stabilize the individual as soon as possible, since research suggests that these patients are also more likely to attempt **suicide**.

Description

Over the past 30 years, crisis housing programs have evolved as short-term, less costly, and less restrictive residential alternatives to hospitalization. Intended to divert individuals from emergency rooms, jails, and hospitals into community-based treatment settings, they offer intensive crisis support to individuals and their families. Services include **diagnosis**, assessment, and treatment (including medication stabilization); rehabilitation; and links to community-based services. These programs are intended to stabilize the individual as rapidly as possible—usually between eight and 60 days—so they can return to their home or residence in the community.

Some of the earliest crisis housing programs include Soteria House and La Posada, which began in northern California in the 1970s, and the START (short-term acute residential treatment) program that began in San Diego in 1980. While programs vary from location to location, most offer acute services 24 hours a day in a small non-institutional residential setting. Adequate structure and supervision is provided by an interdisciplinary team of professionals and other trained workers.

Beginning the day they arrive, residents help develop their own plans for recovery and continued care in the community. Patients receive state-of-the art psychopharmacological treatment and other cognitive-behavioral interventions. Residents are encouraged to play an active role in the operation of the household, including meal preparation. The home-like environment is helpful in lessening the **stigma** and sense of failure that often occurs when someone needs to return to an inpatient psychiatric unit.

Similarly, in the case of seriously emotionally disturbed children and adolescents, the goal of crisis housing is to avert visits to the emergency room or hospitalization by stabilizing the individual in as normal a setting as possible. Compared to these services for adults, there is typically greater emphasis placed on involving families and schools in planning for community-based care after discharge.

While the evidence base for crisis housing is comprised primarily of uncontrolled studies, evaluations of several of these programs suggest that they may provide high-quality treatment and care at a lower cost than hospitals. Crisis housing is not currently available in all communities, however.

See also Crisis intervention

Resources

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Irene S. Levine, Ph.D.

Crisis intervention

Definition

Crisis intervention refers to the methods used to offer immediate, short-term help to individuals who experience an event that produces emotional, mental, physical, and behavioral distress or problems. A crisis can refer to any situation in which the individual perceives a sudden loss of his or her ability to use effective problem-solving and coping skills. A number of events or circumstances can be considered a crisis: life-threatening situations, such as natural disasters (such as an earthquake or tornado), sexual assault or other criminal victimization; medical illness; mental illness; thoughts of **suicide** or homicide; and loss or drastic changes in relationships (death of a loved one or divorce, for example).

KEY TERMS

Coping—In psychology, a term that refers to a person's patterns of response to stress.

Critical incident—Also known as a crisis event. An event that is stressful enough to overwhelm the coping skills of a person or group.

Stress—A physical and psychological response that results from being exposed to a demand or pressure.

Stressor—A stimulus or event that provokes a stress response in an organism. Stressors can be categorized as acute or chronic, and as external or internal to the organism.

Purpose

Crisis intervention has several purposes. It aims to reduce the intensity of an individual's emotional, mental, physical and behavioral reactions to a crisis. Another purpose is to help individuals return to their level of functioning before the crisis. Functioning may be improved above and beyond this by developing new coping skills and eliminating ineffective ways of coping, such as withdrawal, isolation, and substance abuse. In this way, the individual is better equipped to cope with future difficulties. Through talking about what happened, and the feelings about what happened, while developing ways to cope and solve problems, crisis intervention aims to assist the individual in recovering from the crisis and to prevent serious long-term problems from developing. Research documents positive outcomes for crisis intervention, such as decreased distress and improved problem solving.

Description

Individuals are more open to receiving help during crises. A person may have experienced the crisis within the last 24 hours or within a few weeks before seeking help. Crisis intervention is conducted in a supportive manner. The length of time for crisis intervention may

Trained adults staff a 24-hour suicide hotline. *(Stock Boston, Inc. Reproduced by permission.)*

range from one session to several weeks, with the average being four weeks. Crisis intervention is not sufficient for individuals with long-standing problems. Session length may range from 20 minutes to two or more hours. Crisis intervention is appropriate for children, adolescents, and younger and older adults. It can take place in a range of settings, such as hospital emergency rooms, crisis centers, counseling centers, mental health clinics, schools, correctional facilities, and other social service agencies. Local and national telephone hotlines are available to address crises related to suicide, domestic violence, sexual assault, and other concerns. They are usually available 24 hours a day, seven days a week.

Responses to crisis

A typical crisis intervention progresses through several phases. It begins with an assessment of what happened during the crisis and the individual's responses to it. There are certain common patterns of response to most crises. An individual's reaction to a crisis can include emotional reactions (fear, anger, guilt, **grief**), mental reactions (difficulty concentrating, confusion, nightmares), physical reactions (headaches, dizziness, **fatigue**, stomach problems), and behavioral reactions (sleep and appetite problems, isolation, restlessness). Assessment of the individual's potential for suicide and/or homicide is also conducted. Also, information about the individual's strengths, coping skills, and social support networks is obtained.

Education

There is an educational component to crisis intervention. It is critical for the individual to be informed about various responses to crisis and informed that he or she is having normal reactions to an abnormal situation. The individual will also be told that the responses are temporary. Although there is not a specific time that a person can expect to recover from a crisis, an individual can help recovery by engaging in the coping and problem-solving skills described below.

Coping and problem solving

Other elements of crisis intervention include helping the individual understand the crisis and their response to it as well as becoming aware of and expressing feelings, such as anger and guilt. A major focus of crisis interven-

tion is exploring coping strategies. Strategies that the individual previously used but that have not been used to deal with the current crisis may be enhanced or bolstered. Also, new coping skills may be developed. Coping skills may include relaxation techniques and exercise to reduce body tension and **stress** as well as putting thoughts and feelings on paper through journal writing instead of keeping them inside. In addition, options for social support or spending time with people who provide a feeling of comfort and caring are addressed. Another central focus of crisis intervention is problem solving. This process involves thoroughly understanding the problem and the desired changes, considering alternatives for solving the problem, discussing the pros and cons of alternative solutions, selecting a solution and developing a plan to try it out, and evaluating the outcome. Cognitive therapy, which is based on the notion that thoughts can influence feelings and behavior, can be used in crisis intervention.

In the final phase of crisis intervention, the professional will review changes the individual made in order to point out that it is possible to cope with difficult life events. Continued use of the effective coping strategies that reduced distress will be encouraged. Also, assistance will be provided in making realistic plans for the future, particularly in terms of dealing with potential future crises. Signs that the individual's condition is getting worse or "red flags" will be discussed. Information will be provided about resources for additional help should the need arise. A telephone follow-up may be arranged at some agreed-upon time in the future.

Suicide intervention

Purpose

Suicidal behavior is the most frequent mental health emergency. The goal of crisis intervention in this case is to keep the individual alive so that a stable state can be reached and alternatives to suicide can be explored. In other words, the goal is to help the individual reduce distress and survive the crisis.

Assessment

Suicide intervention begins with an assessment of how likely it is that the individual will kill himself or herself in the immediate future. This assessment has various components. The professional will evaluate whether or not the individual has a plan for how the act would be committed, how deadly the method is (shooting, overdosing), if means are available (access to weapons), and if the plan is detailed and specific versus vague. The professional will also assess the individual's emotions, such as depression, hopelessness, hostility and anxiety. Past suicide attempts as well as completed suicides among family and friends will be assessed. The nature of any current crisis event or circumstance will be evaluated, such as loss of physical abilities because of illness or accident, unemployment, and loss of an important relationship.

Treatment plan

A written safekeeping contract may be obtained. This is a statement signed by the individual that he or she will not commit suicide, and agrees to various actions, such as notifying their clinician, family, friends, or emergency personnel, should thoughts of committing suicide again arise. This contract may also include coping strategies that the individual agrees to engage in to reduce distress. If the individual states that he or she is not able to do this, then it may be determined that medical assistance is required and voluntary or involuntary psychiatric **hospitalization** may be implemented. Most individuals with thoughts of suicide do not require hospitalization and respond well to outpatient treatment. Educating family and friends and seeking their support is an important aspect of suicide intervention. Individual therapy, **family therapy**, substance abuse treatment, and/or psychiatric medication may be recommended.

Critical incident stress debriefing and management

Definition

Critical incident stress debriefing (CISD) uses a structured, small group format to discuss a distressing crisis event. It is the best known and most widely used debriefing model. Critical incident stress management (CISM) refers to a system of interventions that includes CISD as well as other interventions, such as one-on-one crisis intervention, **support groups** for family and significant others, stress management education programs, and follow up programs. It was originally designed to be used with high-risk professional groups, such as emergency services, public safety, disaster response, and military personnel. It can be used with any population, including children. A trained personnel team conducts this intervention. The team usually includes professional support personnel, such as mental health professionals and clergy. In some settings, peer support personnel, such as emergency services workers, will be part of the debriefing team. It is recommended that a debriefing occur after the first 24 hours following a crisis event, but before 72 hours have passed since the incident.

Purpose

This process aims to prevent excessive emotional, mental, physical, and behavioral reactions and **post-traumatic stress disorder** (PTSD) from developing in response to a crisis. Its goal is to help individuals recover as quickly as possible from the stress associated with a crisis.

Phases of CISD

There are seven phases to a formal CISD.

1. Introductory remarks: team sets the tone and rules for the discussion, encourages participant cooperation.
2. Fact phase: participants describe what happened during the incident.
3. Thought phase: participants state the first or main thoughts while going through the incident.
4. Reaction phase: participants discuss the elements of the situation that were worst.
5. Symptom phase: participants describe the symptoms of distress experienced during or after the incident.
6. Teaching phase: team provides information and suggestions that can be used to reduce the impact of stress.
7. Re-entry phase: team answers participants' questions and makes summary comments.

Precautions

Some concern has been expressed in the research literature about the effectiveness of CISD. It is thought that as long as the provider(s) of CISD have been properly trained, the process should be helpful to individuals in distress. If untrained personnel conduct CISD, then it may result in harm to the participants. CISD is not **psychotherapy** or a substitute for it. It is not designed to solve all problems presented during the meeting. In some cases, a referral for follow-up assessment and/or treatment is recommended to individuals after a debriefing.

Medical crisis counseling

Medical crisis counseling is a brief intervention used to address psychological (anxiety, fear and depression) and social (family conflicts) problems related to chronic illness in the health care setting. It uses coping techniques and building social support to help patients manage the stress of being newly diagnosed with a chronic illness or suffering a worsening medical condition. It aims to help patients understand their reactions as normal responses to a stressful circumstance and to help them function better. Preliminary studies of medical crisis counseling indicate that one to four sessions may be needed. Research is also promising in terms of its effectiveness at decreasing patients' level of distress and improving their functioning.

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American Association of Suicidology. 4201 Connecticut Avenue, NW, Suite 408, Washington D.C. 20008. (202) 237-2280. <http://www.suicidology.org>.

American Foundation for Suicide Prevention. 120 Wall Street, 22nd Floor, New York, New York, 10005. (212) 363-3500, or 888-333-AFSP. <http://www.afsp.org>.

International Critical Incident Stress Foundation. 10176 Baltimore National Pike, Unit 201, Ellicott City, MD 21042. (410) 750-9600. <http://www.icisf.org>.

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National Strategy for Suicide Prevention. <www.mentalhealth.org/suicideprevention>.

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Cyclothymic disorder

Definition

Cyclothymic disorder, also known as cyclothymia, is a relatively mild form of bipolar II disorder characterized by mood swings that may appear to be almost within the normal range of emotions. These mood swings range from mild depression, or dysthymia, to mania of low intensity, or hypomania.

Description

Cyclothymic disorder, a symptomatically mild form of bipolar II disorder, involves mood swings ranging from mild depression to mild mania. It is possible for cyclothymia to go undiagnosed, and for individuals with the disorder to be unaware that they have a treatable disease. Individuals with cyclothymia may experience episodes of low-level depression, known as dysthymia; periods of intense energy, creativity, and/or irritability, known as hypomania; or they may alternate between both mood states. Like other **bipolar disorders**, cyclothymia is a chronic illness characterized by mood swings that can occur as often as every day and last for several days, weeks, months, or as long as two years. Individuals with this disorder are never free of symptoms of either hypomania or mild depression for more than two months at a time.

The noted **psychiatrist** Emil Kraepelin first described the symptoms of cyclothymic disorder in the late nineteenth century. Kraepelin described four types of **personality disorders**: depressive (gloomy); manic (cheerful and uninhibited); irritable (emotionally unstable and explosive); and cyclothymic. He viewed the irritable personality as simultaneously depressive and manic, and the cyclothymic personality as alternating between depressive and manic states.

Persons with cyclothymic disorder differ in the relative proportion of depressive versus hypomanic episodes that they experience. Some individuals have more frequent depressive episodes, whereas others are more likely to feel hypomanic. Most individuals who seek help for the disorder alternate between feelings of mild depression and intense irritability. Those who feel energized and creative when they are hypomanic, and who find their emotional low periods tolerable, may never seek treatment.

Causes and symptoms

Causes

Controversy exists over whether cyclothymic disorder is truly a mood disorder in either biological or psychological terms, or whether it belongs in the class of disorders known as personality disorders. Despite this controversy, most of the evidence from biological and genetic research supports the placement of cyclothymia within the mood disorder category.

Genetic data provide strong support that cyclothymia is indeed a mood disorder. About 30% of all patients with cyclothymia have family histories of bipolar I disorder, which involves full-blown manic episodes alternating with periods of relative emotional stability. Full-blown depressive episodes are frequently, but not always, part of the picture in bipolar I disorder. Reviews of the family histories of bipolar I patients show a tendency toward illnesses that alternate across generations: bipolar I in one generation, followed by cyclothymia in the next, followed again by bipolar I in the third generation. The general prevalence of cyclothymia in families with bipolar I diagnoses is much higher than in families with other mental disorders or in the general population. It has been reported that about one-third of patients with cyclothymic disorder subsequently develop a major mood disorder.

Most psychodynamic theorists believe that the psychosocial origins of cyclothymia lie in early traumas and unmet needs dating back to the earliest stages of childhood development. Hypomania has been described as a deficiency of self-criticism and an absence of inhibitions. The patient is believed to use **denial** to avoid external problems and internal feelings of depression. Hypomania is also believed to be frequently triggered by profound interpersonal loss. The false feeling of euphoria (giddy or intense happiness) that arises in such instances serves as a protection against painful feelings of sadness, and even possibly anger against the lost loved one.

Symptoms

The symptoms of cyclothymic disorder are identical to those of bipolar I disorder except that they are usually less severe. It is possible, however, for the symptoms of cyclothymia to be as intense as those of bipolar I, but of shorter duration. About one-half of all patients with cyclothymic disorder have depression as their major symptom. These persons are most likely to seek help for their symptoms, especially during their depressed episodes. Other patients with cyclothymic disorder experience primarily hypomanic symptoms. They are less likely to seek help than those who suffer primarily from depression. Almost all patients with cyclothymic disorder have periods of mixed symptoms (both depression and hypomania together) during which time they are highly irritable.

KEY TERMS

Bipolar I disorder—A major mood disorder characterized by full-blown manic episodes, often interspersed with episodes of major depression.

Bipolar II disorder—Disorder with major depressive episodes and mild manic episodes known as hypomania.

Borderline personality disorder—A severe and usually life-long mental disorder characterized by violent mood swings and severe difficulties in sustaining interpersonal relationships.

Cyclothymia—An alternate name for cyclothymic disorder.

Cyclothymic disorder—A relatively mild mood disorder characterized by mood swings between mild depression to mild mania.

Denial—A psychological defense mechanism that reduces anxiety by excluding recognition of an addiction or similar problem from the conscious mind.

Dysthymia—Depression of low intensity.

Hypomania—A milder form of mania that is characteristic of bipolar II disorder.

Psychodynamic theorists—Therapists who believe that the origins of mental problems lie in a person's internal conflicts and complexes.

Psychosocial—A term that refers to the emotional and social aspects of psychological disorders.

Cyclothymic disorder may cause disruption in all areas of the person's life. Many individuals with this disorder are unable to succeed in their professional or personal lives as a result of their symptoms. A few who suffer primarily from hypomanic episodes are high achievers who work long hours and require little sleep. A person's ability to manage the symptoms of the disorder depends upon a number of personal, social, and cultural factors.

The lives of most people suffering from cyclothymic disorder are difficult. The cycles of the disorder tend to be much shorter than in bipolar I. In cyclothymic disorder, mood changes are irregular and abrupt, and can occur within hours. While there are occasional periods of normal mood, the unpredictability of the patient's feelings and behavior creates great **stress** for him or her and for those who must live or work with the patient. Patients often feel that their moods are out of control. During mixed periods, when they are highly irritable, they may become involved in unprovoked arguments with family, friends, and co-workers, causing stress to all around them.

It is common for cyclothymic disorder patients to abuse alcohol and/or other drugs as a means of self-medicating. It is estimated that about 5–10% of all patients with cyclothymic disorder suffer also from substance dependence.

Demographics

Patients with cyclothymic disorder are estimated to constitute from 3–10% of all psychiatric outpatients. They may be particularly well represented among those with complaints about marital and interpersonal difficulties. In the general population, the lifetime chance of developing cyclothymic disorder is about 1%. The actual percentage of the general population with cyclothymia is probably somewhat higher, however, as many patients may not be aware that they have a treatable disease.

Cyclothymic disorder frequently coexists with **borderline personality disorder**, which is a severe lifelong illness characterized by emotional instability and relationship problems. An estimated 10% of outpatients and 20% of inpatients with borderline personality disorder have a coexisting **diagnosis** of cyclothymic disorder. The female-to-male ratio in cyclothymic disorder is approximately 3:2. It is estimated that 50%–75% of all patients develop the disorder between the ages of 15 and 25.

Diagnosis

The diagnosis of cyclothymic disorder is usually made when a person with the disorder is sufficiently disturbed by the symptoms or their consequences to seek help. While there are no laboratory tests or **imaging studies** that can detect the disorder as of 2002, the doctor will usually give the patient a general physical examination to rule out general medical conditions that are often associated with depressed mood. The doctor will also take a detailed medical and psychiatric history. If the patient's history or other aspects of his or her behavior during the interview suggest the diagnosis of cyclothymic disorder, the doctor may follow up the interview with the patient by talking with friends or family members to confirm the diagnosis.

The manual used by mental health professionals to diagnose mental illnesses is called the *Diagnostic and Statistical Manual of Mental Disorders,* 4th edition, text revision, also known as the *DSM-IV-TR*. This manual specifies six criteria that must be met for a diagnosis of cyclothymic disorder. These include:

- Numerous episodes of hypomania and depression that are not severe enough to be considered major depression. These episodes must have lasted for at least two years.
- During the same two-year period (one year for children and adolescents), the individual has not been free from either hypomania or mild depression for more than two months at a time.
- No major depression, mania, or mixed (both depression and mania together) condition has been present during the first two years of the disorder.
- The individual does not have a thought disorder such as **schizophrenia** or other psychotic condition.
- The symptoms are not due to the direct effects of substance use (such as a drug of abuse or a prescribed medication) or to a medical condition.
- The symptoms cause significant impairment in the patient's social, occupational, family, or other important areas of life functioning.

Treatments

Biological therapy

Medication is an important component of treatment for cyclothymic disorder. A class of drugs known as antimanic medications is usually the first line of treatment for these patients. Drugs such as lithium, **carbamazepine** (Tegretol), and sodium valproate (Depakene), have all been reported to be effective. While antidepressant medications might be prescribed, they should be used with caution, because these patients are highly susceptible to hypomanic or full-blown manic episodes induced by antidepressants. It is estimated that 40–50% of all patients with cyclothymic disorder who are treated with antidepressants experience such episodes.

Psychosocial therapy

Psychotherapy with individuals diagnosed with cyclothymic disorder is best directed toward increasing the patient's awareness of his or her condition and helping to develop effective coping strategies for mood swings. Often, considerable work is needed to improve the patient's relationships with family members and workplace colleagues because of damage done to these relationships during hypomanic episodes. Because cyclothymic disorder is a lifelong condition, psychotherapy is also a long-term commitment. Working with families of cyclothymic patients can help them adjust more effectively to the patients' mood swings as well.

Prognosis

While some patients later diagnosed with cyclothymic disorder were considered sensitive, hyperactive, or moody as children, the onset of cyclothymic disorder usually occurs gradually during the patient's late teens or early 20s. Often school performance becomes a problem along with difficulty establishing peer relationships. Approximately one-third of all patients with cyclothymic disorder develop a major mood disorder during their lifetime, usually bipolar II disorder.

Prevention

Cyclothymic disorder appears to have a strong genetic component. It is far more common among the first-degree biological relatives of persons with bipolar I disorder than among the general population. At this time, there are no known effective preventive measures that can reduce the risk of developing cyclothymic disorder. Genetic counseling, which assists a couple in understanding their risk of producing a child with the disorder, may be of some help.

See also Affect; Amitriptyline; Borderline personality disorder; Bupropion; Depression and depressive disorders overview; Fluoxetine; Mixed episode; Personality disorders

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American Psychiatric Association. 1400 K Street NW, Washington, DC 20002. (202) 336-5500.

Mental Illness Foundation. 420 Lexington Avenue, Suite 2104, New York, NY 10170. (212) 682-4699.

Barbara S. Sternberg, Ph.D.

Cylert *see* **Pemoline**

D

Dalmane *see* **Flurazepam**

Dance therapy *see* **Bodywork therapies**

Deinstitutionalization

Definition

Deinstitutionalization is a long-term trend wherein fewer people reside as patients in mental hospitals and fewer mental health treatments are delivered in public hospitals. This trend is directly due to the process of closing public hospitals and the ensuing transfers of patients to community-based mental health services in the late twentieth century. It represents the dissipation of patients over a wider variety of health care settings and geographic areas. Deinstitutionalization also illustrates evolution in the structure, practice, experiences, and purposes of mental health care in the United States.

History

Hospital care for mental health

In the United States in the nineteenth century, hospitals were built to house and care for people with chronic illness, and mental health care was a local responsibility. As with most chronic illness, **hospitalization** did not always provide a cure. Individual states assumed primary responsibilities for mental hospitals beginning in 1890. In the first part of the twentieth century, while mental health treatments had very limited efficacy, many patients received custodial care in state hospitals. Custodial care refers to care in which the patient is watched and protected, but a cure is not sought.

After the founding of the National Institutes of Mental Health (NIMH), new psychiatric medications were developed and introduced into state mental hospitals beginning in 1955. These new medicines brought new hope, and helped address some of the symptoms of mental disorders.

President John F. Kennedy's 1963 Community Mental Health Centers Act accelerated the trend toward deinstitutionalization with the establishment of a network of **community mental health** centers. In the 1960s, with the introduction of Medicare and Medicaid, the federal government assumed an increasing share of responsibility for the costs of mental health care. That trend continued into the 1970s with the implementation of the Supplemental Security Income program in 1974. State governments helped accelerate deinstitutionalization, especially of elderly people. In the 1960s and 1970s, state and national policies championed the need for comprehensive community mental health care, though this ideal was slowly and only partially realized.

Beginning in the 1980s, **managed care** systems began to review systematically the use of inpatient hospital care for mental health. Both public concerns and private health insurance policies generated financial incentives to admit fewer people to hospitals and discharge inpatients more rapidly, limit the length of patient stays, or to transfer responsibility to less costly forms of care.

Indicators and trends

Many statistical indicators show the amount of inpatient hospital care for persons with mental illness decreased during the latter half of the twentieth century, while the total volume of mental health care increased.

A patient care episode is a specific measure of the volume of care provided by an organization or system. It begins when a person visits a health care facility for treatment and ends when the person leaves the facility. In 1955, 77% of all patient care episodes in mental health organizations took place in 24-hour hospitals. By 1994, although the numbers of patient care episodes increased by more than 500%, only 26% of mental health treatment episodes were in these hospitals. The timing of this trend

KEY TERMS

Patient care episodes—A specific measure of the volume of care provided by an organization or system. It begins with a treatment visit to a health care facility (a hospital or residential treatment center, for example) and ends when a person leaves the facility, so it may vary by patient and visit. Over time, the volume of patient care episodes indicates the degree to which a population uses certain health care capacities. Other measures that may be used to measure volume of care include number of beds or bed-days, total number of patients served, and also more specific measures like patient-contact hours.

varied across different states and regions, but it was consistent across a variety of indicators.

The number of inpatient beds available to each group of 100,000 civilians decreased from over 200 beds in 1970 to less than 50 in 1992. The average number of patients in psychiatric hospitals decreased from over 2,000 in 1958 to about 500 in 1978. While adjusted per-capita spending on mental health rose from $16.53 in 1969 to $19.33 in 1994, the portion of funds spent on state and county mental hospitals fell from $9.11 to $4.56.

Transinstutionalization

Trends toward deinstitutionalization also reflect shifting demographics and boundaries of care. For example, decreases in inpatient mental health care can be complemented by increases in outpatient mental health care. Decreases in inpatient mental health care can also be paired with increases in other forms of care, such as social welfare, criminal justice, or nursing home care. Thus deinstitutionalization is part of a process sometimes called transinstutionalization, the transfer of institutional populations from hospitals to jails, nursing homes, and shelters.

Causes and consequences

Causes

Deinstitutionalization, originally and idealistically portrayed by advocates and consumers as a liberating, humane policy alternative to restrictive care, may also be interpreted as a series of health policy reforms that are associated with the gradual demise of mental health care dependent on large, state-supported hospitals. Deinstitutionalization is often attributed to decreased need for hospital care and to the advent of new psychiatric medicines.

Consequences

Ideally, deinstitutionalization represents more humane and liberal treatment of mental illness in community-based settings. Pragmatically, it represents a change in the scope of mental health care from longer, custodial inpatient care to shorter outpatient care.

The process of deinstitutionalization, combined with the scarcity of community-based care, is also associated with the visible problems of **homelessness**. Between 30-50% of homeless people in the United States are people with mental illness, and people with mental illness are disproportionate among the homeless.

Experience and adjustment

Deinstitutionalization also describes the adjustment process whereby people with illness are removed from the effects of life within institutions. Since people may become socialized to highly structured institutional environments, they often adapt their social behavior to institutional conditions. Therefore adjusting to life outside of an institution may be difficult.

Defined experientially, deinstitutionalization allows individuals to regain freedom and empower themselves through responsible choices and actions. With the assistance of **social workers** and through psychiatric rehabilitation, former inpatients can adjust to everyday life outside of institutional rules and expectations. This aspect of deinstitutionalization promotes hope and recovery, ongoing debates over the best structure and process of mental health service delivery notwithstanding.

See also Case management

Resources

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Delirium

Definition

Delirium is a medical condition characterized by a vascillating general disorientation, which is accompanied by cognitive impairment, mood shift, self-awareness, and inability to attend (the inability to focus and maintain attention). The change occurs over a short period of time— hours to days— and the disturbance in consciousness fluctuates throughout the day.

Description

The word delirium comes from the Latin *delirare*. In its Latin form, the word means to become crazy or to rave. A phrase often used to describe delirium is "clouding of consciousness," meaning the person has a diminished awareness of their surroundings. While the delirium is active, the person tends to fade into and out of lucidity, meaning that he or she will sometimes appear to know what's going on, and at other times, may show disorientation to time, place, person, or situation. It appears that the longer the delirium goes untreated, the more progressive the disorientation becomes. It usually begins with disorientation to time, during which a patient will declare it to be morning, even though it may be late night. Later, the person may state that he or she is in a different place rather than at home or in a hospital bed. Still later, the patient may not recognize loved ones, close friends, or relatives, or may insist that a visitor is someone else altogether. Finally, the patient may not recognize the reason for his/her **hospitalization** and might accuse staff or others of some covert reason for his/her hospitalization (see example below). In fact, this waxing and waning of consciousness is often worse at the end of a day, a phenomenon known as "sundowning."

A delirious patient will have a difficult time with most mental operations. Due to the fact that the patient is unable to attend consistently to his environment, he/she can become disoriented. Nevertheless, disorientation and memory loss are not essential to the **diagnosis** of delirium; the inability to focus and maintain attention, however, is essential to rendering a correct diagnosis. Left unchecked, delirium tends to transition from inattention to increased levels of lethargy, leading to torpor, stupor, and coma. In its other form, delirious patients become agitated and almost hypervigilant, with their sleep-wake cycle dramatically altered, fluctuating between great guardedness and **hypersomnia** (excessive drowsiness) during the day and wakefulness during the night. Delirious patients can also experience **hallucinations** of the visual, auditory, or tactile type. In such cases, the patient will see things others cannot see, hear things others cannot hear, and/or feel things that others cannot, such as feeling as though his or her skin is crawling. In short, the extremes of delirium range from the appearance of simple confusion and **apathy** to the anxious, agitated, and hyperactive type, with some patients experiencing both ends of the spectrum during a single episode. It is imperative that a quick evaluation occur if delirium is suspected, because it can lead to death.

Causes and symptoms

Causes

While the symptoms of delirium are numerous and varied, the causes of delirium fall into four basic categories: metabolic, toxic, structural, and infectious. Stated another way, the bases of delirium may be medical, chemical, surgical, or neurological. Many metabolic disorders, such as hypothyroidism, hyperthyroidism, hypokalemia, anoxia, etc. can cause delirium. For example, hypothyroidism (the thyroid gland emits reduced levels of thyroid hormones) brings about a change in emotional responsiveness, which can appear similar to depressive symptoms and cause a state of delirium. Other metabolic sources of delirium involve the dysfunction of

KEY TERMS

Anoxia—Lack of oxygen.

Anticholinergic toxicity—A poisonous effect brought about by ingestion of medications or other toxins that block acetylcholine receptors. When these receptors are blocked, the person taking the medication may find that he or she gets overheated, has dry mouth, has blurry vision, and his or her body may retain urine.

Coma—Unconsciousness.

Hyperthyroidism—Condition resulting from the thyroid glands secreting excessive thyroid hormone, causing increased basal metabolic rate, and causing an increased need for food to meet the demand of the metabolic activity; generally, however, weight loss results.

Hypervigilant—Extreme attention and focus to both internal and external stimuli.

Hypokalemia—Abnormally low levels of potassium in the blood. Hypokalemia is a potential medical emergency, as it can lead to disturbances in of the heart rhythm.

Stupor—A trance-like state that causes a person to appear numb to their environment.

Subdural hematoma—Active bleeding or a blood clot inside the dura (leathery covering of the brain). This bleeding or clot causes swelling of the brain, and, untreated, the condition can cause death.

Torpor—Sluggishness or inactivity.

Tricyclic antidepressants—Antidepressant medications that have the common characteristic of a three-ring nucleus in their chemical structure. Imipramine and amitriptyline are examples of tricyclic antidepressants.

Vascular—Pertaining to the bloodstream (arteries, veins, and blood vessels).

the pituitary gland, pancreas, adrenal glands, and parathyroid glands. It should be noted that when a metabolic imbalance goes unattended, the **brain** may suffer irreparable damage.

One of the most frequent causes of delirium in the elderly is overmedication. The use of medications such as tricyclic antidepressants and antiparkinsonian medications can bring about an anticholinergic toxicity and subsequent delirium. In addition to the anticholinergic drugs, other drugs that can be the source of a delirium are:

- anticonvulsants, used to treat epilepsy
- antihypertensives, used to treat high blood pressure
- cardiac glycosides, such as Digoxin, used to treat heart failure
- cimetidine, used to reduce the production of stomach acid
- **disulfiram**, used in the treatment of alcoholism
- insulin, used to treat diabetes
- opiates, used to treat pain
- phencyclidine (PCP), used originally as an anesthetic, but later removed from the market, now only produced and used illicitly
- salicylates, basically found in aspirin
- steroids, sometimes used to prevent muscle wasting in bedridden or other immobile patients

Additionally, systemic poisoning by chemicals or compounds such as carbon monoxide, lead, mercury, or other industrial chemicals can be the source of delirium.

Just as the ingestion of certain drugs may cause delirium in some patients, the withdrawal of drugs can also cause it. Alcohol is the most widely used and most well known of these drugs whose withdrawal symptoms may include delirium. Delirium onset from the abstinence of alcohol in a chronic user can begin within three days of cessation of drinking. The term delirium tremens is used to describe this form of delirium. The resulting symptoms of this delirium are similar in nature to other delirious states, but may be preceded by clear-headed auditory hallucinations. In other words, the delirium has not begun, but the patient may experience auditory hallucinations. Delirium tremens follow and can have ominous consequences with as many as 15% dying.

Some of the structural causes of delirium include vascular blockage, subdural hematoma, and brain tumors. Any of these can damage the brain, through oxygen deprivation or direct insult, and cause delirium. Some patients become delirious following surgery. This can be due to any of several factors, such as: effects of anesthesia, infections, or a metabolic imbalance.

Infectious diseases can also cause delirium. Commonly diagnosed diseases such as urinary tract infections, pneumonia, or fever from a viral infection can induce delirium. Additionally, diseases of the liver, kidney, lungs, and cardiovascular system can cause delirium. Finally, an infection, specific to the brain, can cause delirium. Even a deficiency of thiamin (vitamin B1) can be a trigger for delirium.

Symptoms

Symptoms of delirium include a confused state of mind accompanied by poor attention, impaired recent memory, irritability, inappropriate behavior (such as the use of vulgar language, despite lack of a history of such behavior), and anxiety and fearfulness. In some cases, the person can appear to be psychotic, fostering illusions, **delusions**, hallucinations, and/or **paranoia**. In other cases, the patient may simply appear to be withdrawn and apathetic. In still other cases, the patient may become agitated and restless, unable to remain in bed, and feel a strong need to pace the floor.

A few examples of people affected by delirium follow:

- One gentleman, who had already been in the hospital for three days, when asked if he knew where he was, stated the correct city and hospital. He immediately followed this by saying, "but I started out in Dallas, Texas this morning." The hospital location was some 1,800 miles from Dallas, Texas, and as previously indicated, he had been in the same hospital for three days.
- In another case, an elderly gentleman was placed in a private room that had a wonderful large mural on one wall. The mural was that of a forest scene—no animals or people, only trees and sunlight. His chief complaint at various points during the day was that evil people were watching him from behind the trees in the forest scene.
- An elderly woman had to be subdued while attempting to flee from the hospital, because she was convinced that she had been brought there so surgeons could harvest her organs. Despite the lack of surgical scars or incisions, she insisted that she had been taken to the basement of the hospital the previous night and a surgeon had removed one of her kidneys.

Demographics

Delirium occurs most frequently in the elderly and the young, but can occur in anyone at any age. Of persons over 65 who are brought to the hospital for a general medical condition, roughly 10% show signs of delirium at admission. It is suspected that another 10%-15% may develop delirium while in the hospital. There appears to be no gender difference—delirium seems to affect males and females equally.

Diagnosis

Whether or not delirium is diagnosed in a patient depends on the type manifest. If the case is an elderly, postoperative patient who appears quiet and apathetic, the condition may go undiagnosed. However, if the patient presents with the agitated, uncooperative type of delirium, it will certainly be noticed. In any case, where there is sudden onset of a confused state accompanied by a behavioral change, delirium should be considered. This is not intended to imply that such a diagnosis will be made easily.

Frequent mental status examinations, at various times throughout the day, may be required to render a diagnosis of delirium. This is generally done using the **Mini-Mental State Examination** (MMSE). This abbreviated form of mental status examination begins by first assessing the patient's ability to attend. If the patient is inattentive or in a stuporous state, further examination of mental status cannot be done. However, assuming the patient is able to respond to questions asked, the examination can proceed. The Mini-Mental State Exam assesses the areas of orientation, registration, attention and concentration, recall, language, and spatial perception. Another recently evaluated and recommended tool for use in diagnosing delirium is the Delirium Rating Scale-Revised-98. This clinician-rated, 16-item scale allows for the assessment of 13 severity items and three diagnostic items. This test has been reported as more sensitive than the MMSE at detecting delirium.

At times, the untrained observer may mistake psychotic features of delirium for another primary mental illness such as **schizophrenia** or a **manic episode** such as that associated with **bipolar disorder**. However, it should be noted that there are major differences between these diagnoses and delirium. In people who have schizophrenia, their odd behavior, stereotyped motor activity, or abnormal speech persists in the absence of disorientation like that seen with delirium. The schizophrenic appears alert and although his/her delusions and/or hallucinations persist, he/she could be formally tested. In contrast, the delirious patient appears hapless and disoriented, between episodes of lucidity. The delirious patient may not be testable. A manic episode could be misconstrued for agitated delirium, but consistency of elevated mood would contrast sharply to the less consistent mood of the delirious patient. Once again, delirium should always be considered when there is a rapid onset and especially when there is waxing and waning of the ability to attend and the confusion state.

Since delirium can be superimposed into a pre-existing **dementia**, the most often posed question, when diagnosing delirium, is whether the person might have dementia instead. Both cause disturbances of memory, but a person with dementia does not reflect the disturbance of consciousness depicted by someone with delirium. Expert history taking is a must in differentiating dementia from delirium. Dementia is insidious in nature and thus progresses slowly, while delirium begins with a

sudden onset and acute symptoms. A person with dementia can appear clear-headed, but can harbor delusions not elicited during an interview. One does not see the typical fluctuation of consciousness in dementia that manifests itself in delirium. It has been stated that, as a general rule, delirium comes and goes, but dementia comes and stays. Delirium rarely lasts more than a month. Usually, by the end of that period, a patient with dementia has full-blown dementia or has died. As a final caution, the clinician must be prepared to rule out **factitious disorder** and **malingering** as possible causes for the delirium.

When a state of delirium is confirmed, the clinician is faced with the task of making the diagnosis in appropriate context to its cause. The delirium may be caused by a general medical condition. In such a case, the clinician must identify the source of the delirium within the diagnosis. For example, if the delirium is caused by liver dysfunction, wherein the liver is unable to clean the system of toxins, thereby allowing them to enter the system and so the brain, the diagnosis would be Delirium Due to Hepatic Encephalopathy. The delirium might also be caused by a substance such as alcohol. To render a diagnosis of Alcohol Intoxication Delirium, the cognitive symptoms should be more exaggerated than those found in intoxication syndrome. The delirium could also be caused by withdrawal from a substance. Continuing the alcohol theme, the diagnosis would be Alcohol Withdrawal Delirium (delirium tremens could be a feature of this diagnosis).

There may be instances in which delirium has multiple causes, such as when a patient has a head trauma and liver failure, or viral encephalitis and alcohol withdrawal. When delirium comes from multiple sources, a diagnosis of delirium precedes each medical condition that contributes. As an example, the multiple causes would be reflected as Delirium Due to Head Trauma and Delirium Due to Hepatic Encephalopathy. Finally, when delirium is the focus of clinical attention, but insufficient evidence exists to identify a specific causal factor, a diagnosis of Delirium Not Otherwise Specified is rendered. An example of this can occur in people who are exposed to sensory deprivation, such as might occur in Intensive Care Units or Cardiac Care Units where the patient is allowed no stimulation save that of the occasional member of the hospital staff.

In summary, delirium develops rapidly, has a fluctuating course involving waxing and waning lucidity, severely affects attention, must receive immediate medical attention, and is reversible in most cases.

Treatment

Treating delirium means treating the underlying illness that is its basis. This could include correcting any chemical disparities within the body, such as electrolyte imbalances, the treatment of an infection, reduction of a fever, or removal of a medication or toxin. A review of anticholinergic effects of medications administered to the patient should take place. It is suggested that sedatives and hypnotic-type medications not be used; however, despite the fact that they can sometimes contribute to delirium, in cases of agitated delirium, the use of these may be necessary. Medications that are often used to treat agitated delirium include **haloperidol**, **thioridazine** and **risperidone**. These can reduce the psychotic features and curb some of the volatility of the patient, but they are only treating symptoms of the delirium and not the source. Benzodiazepines (medications that slow the central nervous system to relax the patient) can also assist in controlling agitated patients, but since they can contribute to delirium, they should be used in the lowest therapeutic doses possible. The reduction and discontinuance of all psychotropic drugs should be the goal of treatment and occur as soon as possible to permit recovery and viable assessment of the patient.

Prognosis

If a quick diagnosis and treatment of delirium occurs, the condition is frequently reversible. However, if the condition goes unchecked or is treated too late, there is a high incidence of mortality or permanent brain damage associated with it. The underlying illness may respond quickly to a treatment regimen, but improvement in mental functioning may lag behind, especially in the elderly. Moreover, one study disclosed that one group of elderly survivors of delirium, at three years following hospital discharge, had a 33% higher rate of death than other patients. As a final note, delirium is a medical emergency, requiring prompt attention to avoid the potential for permanent brain damage or even death.

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Delusional disorder

Definition

Delusional disorder is characterized by the presence of recurrent, persistent non-bizarre **delusions**.

Delusions are irrational beliefs, held with a high level of conviction, that are highly resistant to change even when the delusional person is exposed to forms of proof that contradict the belief. Non-bizarre delusions are considered to be plausible; that is, there is a possibility that what the person believes to be true could actually occur a small proportion of the time. Conversely, bizarre delusions focus on matters that would be impossible in reality. For example, a non-bizarre delusion might be the belief that one's activities are constantly under observation by federal law enforcement or intelligence agencies, which actually does occur for a small number of people. By contrast, a man who believes he is pregnant with German Shepherd puppies holds a belief that could never come to pass in reality. Also, for beliefs to be considered delusional, the content or themes of the beliefs must be uncommon in the person's culture or religion. Generally, in delusional disorder, these mistaken beliefs are organized into a consistent world-view that is logical other than being based on an improbable foundation.

In addition to giving evidence of a cluster of interrelated non-bizarre delusions, persons with delusional disorder experience **hallucinations** far less frequently than do individuals with **schizophrenia** or **schizoaffective disorder**.

Description

Unlike most other psychotic disorders, the person with delusional disorder typically does not appear obviously odd, strange or peculiar during periods of active illness. Yet the person might make unusual choices in day-to-day life because of the delusional beliefs. Expanding on the previous example, people who believe they are under government observation might seem typical in most ways but could refuse to have a telephone or use credit cards in order to make it harder for "those Federal agents" to monitor purchases and conversations. Most mental health professionals would concur that until the person with delusional disorder discusses the areas of life affected by the delusions, it would be difficult to distinguish the sufferer from members of the general public who are not psychiatrically disturbed. Another distinction of delusional disorder compared with other psychotic disorders is that hallucinations are either absent or occur infrequently.

The person with delusional disorder may or may not come to the attention of mental health providers. Typically, while delusional disorder sufferers may be distressed about the delusional "reality," they may not have the insight to see that anything is wrong with the way they are thinking or functioning. Regarding the earlier example, those suffering delusion might state that the only thing wrong or upsetting in their lives is that the government is spying, and if the surveillance would cease, so would the problems. Similarly, the people suffering the disorder attribute any obstacles or problems in functioning to the delusional reality, separating it from their internal control. Furthermore, whether unable to get a good job or maintain a romantic relationship, the difficulties would be blamed on "government interference" rather than on their own failures or omissions. Unless the form of the delusions causes illegal behavior, somehow affects an ability to work, or otherwise deal with daily activities, the delusional disorder sufferer may adapt well enough to navigate life without coming to clinical attention. When people with delusional disorder decide to seek mental health care, the motivation for getting treatment is usually to decrease the negative emotions of depression, fearfulness, rage, or constant worry caused

KEY TERMS

Hallucinations—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Psychosis or psychotic symptoms—Disruptions in perceiving reality, thinking logically, and speaking or behaving in normal fashion. Hallucinations, delusions, catatonic behavior and peculiar speech are all symptoms of psychosis. In *DSM-IV-TR*, psychosis is usually one feature of an over-arching disorder, not a disorder in itself (with the exception of the diagnosis *psychosis not otherwise specified.*

by living under the cloud of delusional beliefs, not to change the unusual thoughts themselves.

Forms of delusional disorder

An important aspect of delusional disorder is the identification of the form of delusion from which a person suffers. The most common form of delusional disorder is the persecutory or paranoid subtype, in which the patients are certain that others are striving to harm them.

In the erotomanic form of delusional disorder, the primary delusional belief is that some important person is secretly in love with the sufferer. The erotomanic type is more common in women than men. Erotomanic delusions may prompt stalking the love object and even violence against the beloved or those viewed as potential romantic rivals.

The grandiose subtype of delusional disorder involves the conviction of one's importance and uniqueness, and takes a variety of forms: believing that one has a distinguished role, has some remarkable connections with important persons, or enjoys some extraordinary powers or abilities.

In the somatic subtype, there is excessive concern and irrational ideas about bodily functioning, which may include worries regarding infestation with parasites or insects, imagined physical deformity, or a conviction that one is emitting a foul stench when there is no problematic odor.

The form of disorder most associated with violent behavior, usually between romantic partners, is the jealous subtype of delusional disorder. Patients are firmly convinced of the infidelity of a spouse or partner, despite contrary evidence and based on minimal data (like a messy bedspread or more cigarettes than usual in an ashtray, for instance). Delusional jealousy sufferers may gather scraps of conjectured "evidence," and may try to constrict their partners' activities or confine them to home. Delusional disorder cases involving aggression and injury toward others have been most associated with this subtype.

Delusion and other disorders

Even though the main characteristic of delusional disorder is a noticeable system of delusional beliefs, delusions may occur in the course of a large number of other psychiatric disorders. Delusions are often observed in persons with other psychotic disorders such as schizophrenia and schizoaffective disorder. In addition to occurring in the psychotic disorders, delusions also may be evident as part of a response to physical, medical conditions (such as **brain** injury or brain tumors), or reactions to ingestion of a drug.

Delusions also occur in the dementias, which are syndromes wherein psychiatric symptoms and memory loss result from deterioration of brain tissue. Because delusions can be shown as part of many illnesses, the **diagnosis** of delusional disorder is partially conducted by process of elimination. If the delusions are not accompanied by persistent, recurring hallucinations, then schizophrenia and schizoaffective disorder are not appropriate diagnoses. If the delusions are not accompanied by memory loss, then **dementia** is ruled out. If there is no physical illness or injury or other active biological cause (such as drug ingestion or drug withdrawal), then the delusions cannot be attributed to a general medical problem or drug-related causes. If delusions are the most obvious and pervasive symptom, without hallucinations, medical causation, drug influences or memory loss, then delusional disorder is the most appropriate categorization.

Because delusions occur in many different disorders, some clinician-researchers have argued that there is little usefulness in focusing on what diagnosis the person has been given. Those who ascribe to this view believe it is more important to focus on the symptom of delusional thinking, and find ways to have an effect on delusions, whether they occur in delusional disorder or schizophrenia or schizoaffective disorder. The majority of **psychotherapy** techniques used in delusional disorder come from symptom-focused (as opposed to diagnosis-focused) researcher-practitioners.

Causes and symptoms

Causes

Because clear identification of delusional disorder has traditionally been challenging, scientists have conducted far less research relating to the disorder than studies for schizophrenia or mood disorders. Still, some theories of causation have developed, which fall into several categories.

GENETIC OR BIOLOGICAL. Close relatives of persons with delusional disorder have increased rates of delusional disorder and paranoid personality traits. They do not have higher rates of schizophrenia, schizoaffective disorder or mood disorder compared to relatives of non-delusional persons. Increased incidence of these psychiatric disorders in individuals closely genetically related to persons with delusional disorder suggest that there is a genetic component to the disorder. Furthermore, a number of studies comparing activity of different regions of the brain in delusional and non-delusional research participants yielded data about differences in the functioning of the brains between members of the two groups. These differences in brain activity suggest that persons neurologically with delusions tend to react as if threatening conditions are consistently present. Non-delusional persons only show such patterns under certain kinds of conditions where the interpretation of being threatened is more accurate. With both brain activity evidence and family heritability evidence, a strong chance exists that there is a biological aspect to delusional disorder.

DYSFUNCTIONAL COGNITIVE PROCESSING. An elaborate term for thinking is "cognitive processing." Delusions may arise from distorted ways people have of explaining life to themselves. The most prominent cognitive problems involve the manner in which delusion sufferers develop conclusions both about other people, and about causation of unusual perceptions or negative events. Studies examining how people with delusions develop theories about reality show that the subjects have ideas which which they tend to reach an inference based on less information than most people use. This "jumping to conclusions" bias can lead to delusional interpretations of ordinary events. For example, developing flu-like symptoms coinciding with the week new neighbors move in might lead to the conclusion, "the new neighbors are poisoning me." The conclusion is drawn without considering alternative explanations—catching an illness from a relative with the flu, that a virus seems to be going around at work, or that the tuna salad from lunch at the deli may have been spoiled. Additional research shows that persons prone to delusions "read" people differently than non-delusional individuals do. Whether they do so more accurately or particularly poorly is a matter of controversy. Delusional persons develop interpretations about how others view them that are distorted. They tend to view life as a continuing series of threatening events. When these two aspects of thought co-occur, a tendency to develop delusions about others wishing to do them harm is likely.

MOTIVATED OR DEFENSIVE DELUSIONS. Some predisposed persons might suffer the onset of an ongoing delusional disorder when coping with life and maintaining high self-esteem becomes a significant challenge. In order to preserve a positive view of oneself, a person views others as the cause of personal difficulties that may occur. This can then become an ingrained pattern of thought.

Symptoms

The criteria that define delusional disorder are furnished in the ***Diagnostic and Statistical Manual of Mental Disorders*** Fourth Edition Text Revision, or *DSM-IV-TR*, published by the American Psychiatric Association. The criteria for delusional disorder are as follows:

- non-bizarre delusions which have been present for at least one month
- absence of obviously odd or bizarre behavior
- absence of hallucinations, or hallucinations that only occur infrequently in comparison to other psychotic disorders
- no memory loss, medical illness or drug or alcohol-related effects are associated with the development of delusions

Demographics

The base rate of delusional disorder in adults is unclear. The prevalence is estimated at 0.025-0.03%, lower than the rates for schizophrenia (1%). Delusional disorder may account for 1–2% of admissions to inpatient psychiatric hospitals. Age at onset ranges from 18–90 years, with a mean age of 40 years. More females than males (overall) suffer from delusional disorder, especially the late onset form that is observed in the elderly.

Diagnosis

Client interviews focused on obtaining information about the sufferer's life situation and past history aid in identification of delusional disorder. With the client's permission, the clinician obtains details from earlier medical records, and engages in thorough discussion with the client's immediate family—helpful measures in determining whether delusions are present. The clinician

may use a semi-structured interview called a mental status examination to assess the patient's concentration, memory, understanding the individual's situation and logical thinking. The mental status examination is intended to reveal peculiar thought processes in the patient. The Peters Delusion Inventory (PDI) is a psychological test that focuses on identifying and understanding delusional thinking; but its use is more common in research than in clinical practice.

Even using the *DSM-IV-TR* criteria listed above, classification of delusional disorder is relatively subjective. The criteria "non-bizarre" and "resistant to change" and "not culturally accepted" are all subject to very individual interpretations. They create variability in how professionals diagnose the illness. The utility of diagnosing the syndrome rather than focusing on successful treatment of delusion in any form of illness is debated in the medical community. Some researchers further contend that delusional disorder, currently classified as a psychotic disorder, is actually a variation of depression and might respond better to antidepressants or therapy more similar to that utilized for depression. Also, the meaning and implications of "culturally accepted" can create problems. The cultural relativity of "delusions,"—most evident where the beliefs shown are typical of the person's subculture or religion yet would be viewed as strange or delusional by the dominant culture—can force complex choices to be made in diagnosis and treatment. An example could be that of a Haitian immigrant to the United States who believed in voodoo. If that person became aggressive toward neighbors issuing curses or hexes, believing that death is imminent at the hands of those neighbors, a question arises. The belief is typical of the individual's subculture, so the issue is whether it should be diagnosed or treated. If it were to be treated, whether the remedy should come through Western medicine, or be conducted through voodoo shamanistic treatment is the problem to be solved.

Treatments

Delusional disorder treatment often involves *atypical* (also called *novel* or *newer-generation*) antipsychotic medications, which can be effective in some patients. **Risperidone** (Risperdal), **quetiapine** (Seroquel), and **olanzapine** (Zyprexa) are all examples of atypical or novel antipsychotic medications. If *agitation* occurs, a number of different antipsychotics can be used to conclude the outbreak of acute agitation. Agitation, a state of frantic activity experienced concurrently with anger or exaggerated fearfulness, increases the risk that the client will endanger self or others. To decrease anxiety and slow behavior in emergency situations where agitation is a factor, an injection of **haloperidol** (Haldol) is often given usually in combination with other medications (often **lorazepam**, also known as Ativan). Agitation in delusional disorder is a typical response to severe or harsh confrontation when dealing with the existence of the delusions. It can also be a result of blocking the individual from performing inappropriate actions the client views as urgent in light of the delusional reality. A novel antipsychotic is generally given orally on a daily basis for ongoing treatment meant for long-term effect on the symptoms. Response to antipsychotics in delusional disorder seems to follow the "rule of thirds," in which about one-third of patients respond somewhat positively, one-third show little change, and one-third worsen or are unable to comply.

Cognitive therapy has shown promise as an emerging treatment for delusions. The cognitive therapist tries to capitalize on any doubt the individual has about the delusions; then attempts to develop a joint effort with the sufferer to generate alternative explanations, assisting the client in checking the evidence. This examination proceeds in favor of the various explanations. Much of the work is done by use of empathy, asking hypothetical questions in a form of therapeutic Socratic dialogue—a process that follows a basic question and answer format, figuring out what is known and unknown before reaching a logical conclusion. Combining pharmacotherapy with cognitive therapy integrates both treating the possible underlying biological problems and decreasing the symptoms with psychotherapy.

Prognosis

Evidence collected to date indicates about 10% of cases will show some improvement of delusional symptoms though irrational beliefs may remain; 33–50% may show complete remission; and, in 30–40% of cases there will be persistent non-improving symptoms. The prognosis for clients with delusional disorder is largely related to the level of conviction regarding the delusions and the openness the person has for allowing information that contradicts the delusion.

Prevention

Little work has been done thus far regarding prevention of the disorder. Effective means of prevention have not been identified.

See also Dementia; Depression (with psychotic features); Paranoia; Paranoid personality disorder

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ORGANIZATIONS

National Alliance for the Mentally Ill. Colonial Place Three, 2107 Wilson Blvd., Suite 300, Arlington, VA 22201. Telephone: (703) 524-7600. NAMI HelpLine: 1-800-950-NAMI (6264). <http://www.nami.org>.

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Delusions

Description

A delusion is a belief that is clearly false and that indicates an abnormality in the affected person's content of thought. The false belief is not accounted for by the person's cultural or religious background or his or her level of intelligence. The key feature of a delusion is the degree to which the person is convinced that the belief is true. A person with a delusion will hold firmly to the belief regardless of evidence to the contrary. Delusions can be difficult to distinguish from overvalued ideas, which are unreasonable ideas that a person holds, but the affected person has at least some level of doubt as to its truthfulness. A person with a delusion is absolutely convinced that the delusion is real.

KEY TERMS

Delusion—A false belief that is resistant to reason or contrary to actual fact.

Overvalued idea—An unreasonable, sustained belief that is held with less than delusional intensity (i.e., the person can acknowledge, to some degree, that the belief may be false). The belief is not accounted for by the individual's cultural or religious background.

Delusions are a symptom of either a medical, neurological, or mental disorder. Delusions may be present in any of the following mental disorders:

- psychotic disorders, or disorders in which the affected person has a diminished or distorted sense of reality and cannot distinguish the real from the unreal, including **schizophrenia**, **schizoaffective disorder**, **delusional disorder**, **schizophreniform disorder**, **shared psychotic disorder**, **brief psychotic disorder**, and substance-induced psychotic disorder
- **bipolar disorder**
- **major depressive disorder** with psychotic features
- **delirium**
- **dementia**

Overvalued ideas may be present in **anorexia nervosa**, **obsessive-compulsive disorder**, **body dysmorphic disorder**, or **hypochondriasis**.

Types

Delusions are categorized as either bizarre or non-bizarre and as either mood-congruent or mood-incongruent. A bizarre delusion is a delusion that is very strange and completely implausible for the person's culture; an example of a bizarre delusion would be that aliens have removed the affected person's **brain**. A non-bizarre delusion is one whose content is definitely mistaken, but is at least possible; an example may be that the affected person mistakenly believes that he or she is under constant police surveillance. A mood-congruent delusion is any delusion whose content is consistent with either a depressive or manic state; for example, a depressed person may believe that the world is ending, or a person in a manic state (a state in which the person feels compelled to take on new projects, has a lot of energy, and needs little sleep) believes that he or she has special talents or abilities, or is a famous person. A mood-incongruent delusion is any delusion whose content is not consistent with either a depressed or

manic state or is mood-neutral. An example is a depressed person who believes that thoughts are being inserted into his or her mind from some outside force, person, or group of people, and these thoughts are not recognized as the person's own thoughts (called "thought insertion").

In addition to these categories, delusions are often categorized according to theme. Although delusions can have any theme, certain themes are more common. Some of the more common delusion themes are:

- Delusion of control: This is a false belief that another person, group of people, or external force controls one's thoughts, feelings, impulses, or behavior. A person may describe, for instance, the experience that aliens actually make him or her move in certain ways and that the person affected has no control over the bodily movements. Thought broadcasting (the false belief that the affected person's thoughts are heard aloud), thought insertion, and thought withdrawal (the belief that an outside force, person, or group of people is removing or extracting a person's thoughts) are also examples of delusions of control.
- Nihilistic delusion: A delusion whose theme centers on the nonexistence of self or parts of self, others, or the world. A person with this type of delusion may have the false belief that the world is ending.
- Delusional jealousy (or delusion of infidelity): A person with this delusion falsely believes that his or her spouse or lover is having an affair. This delusion stems from pathological jealousy and the person often gathers "evidence" and confronts the spouse about the nonexistent affair.
- Delusion of guilt or sin (or delusion of self-accusation): This is a false feeling of remorse or guilt of delusional intensity. A person may, for example, believe that he or she has committed some horrible crime and should be punished severely. Another example is a person who is convinced that he or she is responsible for some disaster (such as fire, flood, or earthquake) with which there can be no possible connection.
- Delusion of mind being read: The false belief that other people can know one's thoughts. This is different from thought broadcasting in that the person does not believe that his or her thoughts are heard aloud.
- Delusion of reference: The person falsely believes that insignificant remarks, events, or objects in one's environment have personal meaning or significance. For instance, a person may believe that he or she is receiving special messages from the news anchorperson on television. Usually the meaning assigned to these events is negative, but the "messages" can also have a grandiose quality.
- Erotomania: A delusion in which one believes that another person, usually someone of higher status, is in love with him or her. It is common for individuals with this type of delusion to attempt to contact the other person (through phone calls, letters, gifts, and sometimes stalking).
- Grandiose delusion: An individual exaggerates his or her sense of self-importance and is convinced that he or she has special powers, talents, or abilities. Sometimes, the individual may actually believe that he or she is a famous person (for example, a rock star or Christ). More commonly, a person with this delusion believes he or she has accomplished some great achievement for which they have not received sufficient recognition.
- Persecutory delusions: These are the most common type of delusions and involve the theme of being followed, harassed, cheated, poisoned or drugged, conspired against, spied on, attacked, or obstructed in the pursuit of goals. Sometimes the delusion is isolated and fragmented (such as the false belief that co-workers are harassing), but sometimes are well-organized belief systems involving a complex set of delusions ("systematized delusions"). A person with a set of persecutory delusions may be believe, for example, that he or she is being followed by government organizations because the "persecuted" person has been falsely identified as a spy. These systems of beliefs can be so broad and complex that they can explain everything that happens to the person.
- Religious delusion: Any delusion with a religious or spiritual content. These may be combined with other delusions, such as grandiose delusions (the belief that the affected person was chosen by God, for example), delusions of control, or delusions of guilt. Beliefs that would be considered normal for an individual's religious or cultural background are not delusions.
- Somatic delusion: A delusion whose content pertains to bodily functioning, bodily sensations, or physical appearance. Usually the false belief is that the body is somehow diseased, abnormal, or changed. An example of a somatic delusion would be a person who believes that his or her body is infested with parasites.

Delusions of control, nihilistic delusions, and thought broadcasting, thought insertion, and thought withdrawal are usually considered bizarre delusions. Most persecutory, somatic, grandiose, and religious delusions, as well as most delusions of jealousy, delusions of mind being read, and delusions of guilt would be considered non-bizarre.

See also Hallucinations

Resources

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Dementia

Definition

Dementia is not a specific disorder or disease. It is a syndrome (group of symptoms) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with performing the tasks of daily life. Dementia can occur to anyone at any age from an injury or from oxygen deprivation, although it is most commonly associated with aging. It is the leading cause of institutionalization of older adults.

Description

The definition of dementia has become more inclusive over the past several decades. Whereas earlier descriptions of dementia emphasized memory loss, the last three editions of the professional's diagnostic handbook, ***Diagnostic and Statistical Manual of Mental Disorders*** (also known as the *DSM*) define dementia as an overall decline in intellectual function, including difficulties with language, simple calculations, planning and judgment, and motor (muscular movement) skills as well as loss of memory. Although dementia is not caused by aging itself— most researchers regard it as resulting from injuries, infections, **brain** diseases, tumors, or other disorders— it is quite common in older people. The prevalence of dementia increases rapidly with age; it doubles every five years after age 60. Dementia affects only 1% of people aged 60–64 but 30%–50% of those older than 85. About four to five million persons in the United States are affected by dementia as of 2002. Surveys indicate that dementia is the condition most feared by older adults in the United States.

Causes and symptoms

Causes

Dementia can be caused by nearly forty different diseases and conditions, ranging from dietary deficiencies and metabolic disorders to head injuries and inherited diseases. The possible causes of dementia can be categorized as follows:

- Primary dementia. These dementias are characterized by damage to or wasting away of the brain tissue itself. They include **Alzheimer's disease** (AD), frontal lobe dementia (FLD), and Pick's disease. FLD is dementia caused by a disorder (usually genetic) that affects the front portion of the brain, and Pick's disease is a rare type of primary dementia that is characterized by a progressive loss of social skills, language, and memory, leading to personality changes and sometimes loss of moral judgment.
- Multi-infarct dementia (MID). Sometimes called **vascular dementia**, this type is caused by blood clots in the small blood vessels of the brain. When the clots cut off the blood supply to the brain tissue, the brain cells are damaged and may die. (An infarct is an area of dead tissue caused by obstruction of the circulation.)
- Lewy body dementia. Lewy bodies are areas of injury found on damaged nerve cells in certain parts of the brain. They are associated with Alzheimer's and Parkinson's disease, but researchers do not yet know whether dementia with Lewy bodies is a distinct type of dementia or a variation of Alzheimer's or Parkinson's disease.
- Dementia related to alcoholism or exposure to heavy metals (arsenic, antimony, bismuth).
- Dementia related to infectious diseases. These infections may be caused by viruses (HIV, viral encephalitis); spirochetes (Lyme disease, syphilis); or prions (Creutzfeldt-Jakob disease). Spirochetes are certain kinds of bacteria, and prions are protein particles that lack nucleic acid.
- Dementia related to abnormalities in the structure of the brain. These may include a buildup of spinal fluid in the brain (hydrocephalus); tumors; or blood collecting beneath the membrane that covers the brain (subdural hematoma).

Dementia may also be associated with depression, low levels of thyroid hormone, or niacin or vitamin B_{12} deficiency. Dementia related to these conditions is often reversible.

Genetic factors in dementia

Genetic factors play a role in several types of dementia, but the importance of these factors in the

KEY TERMS

Age-associated memory impairment (AAMI)—A condition in which an older person suffers some memory loss and takes longer to learn new information. AAMI is distinguished from dementia in that it is not progressive and does not represent a serious decline from the person's previous level of functioning. Benign senescent forgetfulness is another term for AAMI.

Agnosia—Loss of the ability to recognize familiar people, places, and objects.

Amyloid—A waxy translucent substance composed mostly of protein, that forms plaques (abnormal deposits) in the brain during the progression of Alzheimer's disease.

Aphasia—Loss of previously acquired ability to understand or use written or spoken language, due to brain damage or deterioration.

Apraxia—Inability to perform purposeful movements that is not caused by paralysis or loss of feeling.

Creutzfeldt-Jakob disease—A degenerative disease of the central nervous system caused by a prion, or "slow virus."

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness. It can be distinguished from dementia by its relatively sudden onset and variation in the severity of the symptoms.

Diagnostic and Statistical Manual of Mental Disorders—A handbook for mental health professionals that includes lists of symptoms that indicate diagnoses of mental disorders.

Frontal lobe dementia—Dementia caused by a disorder, usually genetic, that affects the front portion of the brain.

Hematoma—An accumulation of blood, often clotted, in a body tissue or organ, usually caused by a break or tear in a blood vessel.

Huntington's disease—A hereditary disorder that appears in middle age and is characterized by gradual brain deterioration, progressive dementia, and loss of voluntary movement. It is sometimes called Huntington's chorea.

Hydrocephalus—The accumulation of cerebrospinal fluid (CSF) in the ventricles of the brain.

Lewy bodies—Areas of injury found on damaged nerve cells in certain parts of the brain associated with dementia.

Multi-infarct dementia—Dementia caused by damage to brain tissue resulting from a series of blood clots or clogs in the blood vessels. It is also called vascular dementia.

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness. It may be related in some way to Lewy body dementia.

Pick's disease—A rare type of primary dementia that affects the frontal lobes of the brain. It is characterized by a progressive loss of social skills, language, and memory, leading to personality changes and sometimes loss of moral judgment.

Prion—A protein particle that lacks nucleic acid.

Pseudodementia—A term for a depression with symptoms resembling those of dementia. The term "dementia of depression" is now preferred.

development of the dementia varies considerably. Alzheimer's disease (AD) is known, for example, to have an autosomal (non-sex-related) dominant pattern in most early-onset cases as well as in some late-onset cases, and to show different degrees of penetrance (frequency of expression) in late-life cases. Moreover, researchers have not yet discovered how the genes associated with dementia interact with other risk factors to produce or trigger the dementia. One non-genetic risk factor presently being investigated is toxic substances in the environment.

EARLY-ONSET ALZHEIMER'S DISEASE. In early-onset AD, which accounts for 2%–7% of cases of AD, the symptoms develop before age 60. It is usually caused by an inherited genetic mutation. Early-onset AD is also associated with Down syndrome, in that persons with trisomy 21 (three forms of human chromosome 21 instead of a pair) often develop early-onset AD.

LATE-ONSET ALZHEIMER'S DISEASE. Recent research indicates that late-onset Alzheimer's disease is a polygenic disorder; that is, its development is influenced by more than one gene. It has been known since 1993 that a

specific form of a gene (the APOE gene) on human chromosome 19 is a genetic risk factor for late-onset AD. In 1998 researchers at the University of Pittsburgh reported on another gene that controls the production of bleomycin hydrolase (BH) as a second genetic risk factor that acts independently of the APOE gene. In December 2000, three separate research studies reported that a gene on chromosome 10 that may affect the processing of a protein (called amyloid-beta protein) is also involved in the development of late-onset AD. When this protein is not properly broken down, a starchy substance builds up in the brains of people with AD to form the plaques that are characteristic of the disease.

MULTI-INFARCT DEMENTIA (MID). While the chief risk factors for MID are high blood pressure, advanced age, and male sex, there is an inherited form of MID called CADASIL, which stands for cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy. CADASIL can cause psychiatric disturbances and severe headaches as well as dementia.

FRONTAL LOBE DEMENTIAS. Researchers think that between 25% and 50% of cases of frontal lobe dementia involve genetic factors. Pick's dementia appears to have a much smaller genetic component than FLD. It is not yet known what other risk factors combine with inherited traits to influence the development of frontal lobe dementias.

FAMILIAL BRITISH DEMENTIA (FBD). FBD is a rare autosomal dominant disorder that was first reported in the 1940s in a large British family extending over nine generations. FBD resembles Alzheimer's in that the patient develops a progressive dementia related to amyloid deposits in the brain. In 1999, a mutated gene that produces the amyloid responsible for FBD was discovered on human chromosome 13. Studies of this mutation may yield further clues to the development of Alzheimer's disease as well as FBD itself.

CREUTZFELDT-JAKOB DISEASE. Although Creutzfeldt-Jakob disease is caused by a prion, researchers think that 5%–15% of cases may have a genetic component.

Symptoms

The fourth edition, text revised version of the *DSM* was published in 2000, and is known as *DSM-IV-TR*. *DSM-IV-TR* identifies certain symptoms as criteria that must be met for a patient to be diagnosed with dementia. One criterion is significant weakening of the patient's memory with regard to learning new information as well as recalling previously learned information. In addition, the patient must be found to have one or more of the following disturbances:

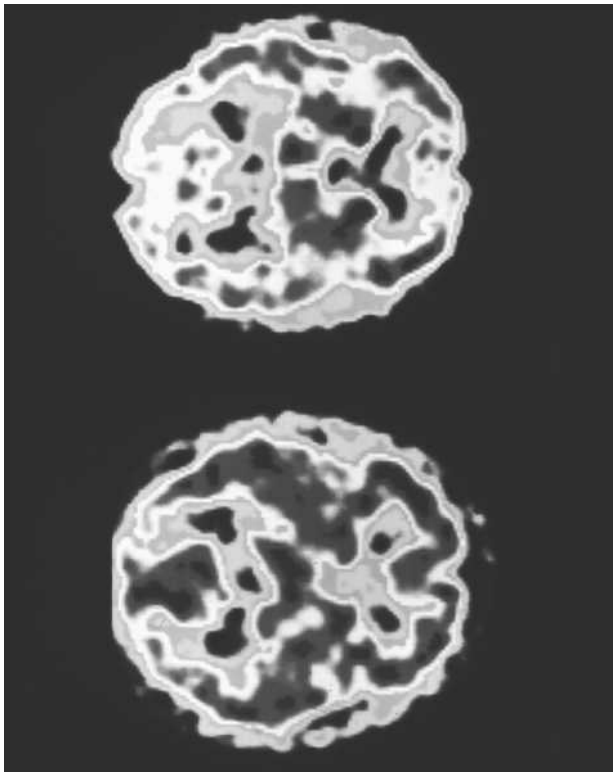
- Aphasia. Aphasia refers to loss of language function. A person with dementia may use vague words like "it" or "thing" often because he or she can't recall the exact name of an object; the affected person may echo what other people say, or repeat a word or phrase over and over. People in the later stages of dementia may stop speaking at all.
- Apraxia. Apraxia refers to loss of the ability to perform intentional movements even though the person is not paralyzed, has not lost the sense of touch, and knows what he or she is trying to do. For example, a patient with apraxia may stop brushing their teeth, or have trouble tying their shoelaces.
- Agnosia. Agnosia refers to loss of the ability to recognize objects even though the person's sight and sense of touch are normal. People with severe agnosia may fail to recognize family members or even their own face reflected in a mirror.
- Problems with abstract thinking and complex behavior. This criterion refers to the loss of the ability to make plans, carry out the steps of a task in the proper order, make appropriate decisions, evaluate situations, show good judgment, etc. For example, a patient might light a stove burner under a saucepan before putting food or water in the pan, or be unable to record checks and balance their checkbook.

DSM-IV-TR also specifies that these disturbances must be severe enough to cause problems in the person's daily life, and that they must represent a decline from a previously higher level of functioning.

In addition to the changes in cognitive functioning, the symptoms of dementia may also include personality changes and emotional instability. Patients with dementia sometimes become mildly paranoid because their loss of short-term memory leads them to think that mislaid items have been stolen. About 25% of patients with dementia develop a significant degree of **paranoia**, that is, generalized suspiciousness or specific **delusions** of persecution. Mood swings, anxiety, and irritability or anger are also frequent occurrences, particularly when patients with dementia are in situations that force them to recognize the extent of their impairment.

The following sections describe the signs and symptoms that are used to differentiate among the various types of dementia during a diagnostic evaluation.

ALZHEIMER'S DISEASE. Dementia related to AD often progresses slowly; it may be accompanied by irritability, wide mood swings, and personality changes in the early stage. Many patients, however, retain their normal degree of sociability in the early stages of Alzheimer's. In second-stage AD, the patient typically gets lost easily, is



Colored positron emission tomography scan of an AIDS patient with dementia. *(Photo Researchers, Inc. Reproduced by permission.) See color insert for color version of photo.*

completely disoriented with regard to time and space, and may become angry, uncooperative, or aggressive. Patients in second-stage AD are at high risk for falls and other accidents. In final-stage AD, the patient is completely bedridden, has lost control over bowel and bladder functions, and may be unable to swallow or eat. The risk of **seizures** increases as the patient progresses from early to end-stage Alzheimer's. Death usually results from an infection or from malnutrition.

MULTI-INFARCT DEMENTIA. In MID, the symptoms are more likely to occur after age 70. In the early stages, the patient retains his or her personality more fully than a patient with AD. Another distinctive feature of this type of dementia is that it often progresses in a stepwise fashion; that is, the patient shows rapid changes in functioning, then remains at a plateau for a while rather than showing a continuous decline. The symptoms of MID may also have a "patchy" quality; that is, some of the patient's mental functions may be severely affected while others are relatively undamaged. Other symptoms of MID include exaggerated reflexes, an abnormal gait (manner of walking), loss of bladder or bowel control, and inappropriate laughing or crying.

DEMENTIA WITH LEWY BODIES. This type of dementia may combine some features of AD, such as severe memory loss and confusion, with certain symptoms associated with Parkinson's disease, including stiff muscles, a shuffling gait, and trembling or shaking of the hands. Visual **hallucinations** may be one of the first symptoms of dementia with Lewy bodies.

FRONTAL LOBE DEMENTIAS. The frontal lobe dementias are gradual in onset. Pick's dementia is most likely to develop in persons between 40 and 60, while FLD typically begins before the age of 65. The first symptoms of the frontal lobe dementias often include socially inappropriate behavior (rude remarks, sexual acting-out, disregard of personal hygiene, etc.). Patients are also often obsessed with eating and may put non-food items in their mouths as well as making frequent sucking or smacking noises. In the later stages of frontal lobe dementia or Pick's disease, the patient may develop muscle weakness, twitching, and delusions or hallucinations.

CREUTZFELDT-JAKOB DISEASE. The dementia associated with Creutzfeldt-Jakob disease occurs most often in persons between 40 and 60. It is typically preceded by a period of several weeks in which the patient complains of unusual **fatigue**, anxiety, loss of appetite, or difficulty concentrating. This type of dementia also usually progresses much more rapidly than other dementias, frequently over a span of a few months.

Demographics

The demographic distribution of dementia varies somewhat according to its cause. Moreover, recent research indicates that dementia in many patients has overlapping causes, so that it is not always easy to assess the true rates of occurrence of the different types. For example, AD and MID are found together in about 15%–20% of cases.

Alzheimer's disease

AD is by far the most common cause of dementia in the elderly, accounting for 60%–80% of cases. It is estimated that four million adults in the United States suffer from AD. The disease strikes women more often than men, but researchers don't know yet whether the sex ratio simply reflects the fact that women in developed countries tend to live longer than men, or whether female sex is itself a risk factor for AD. One well-known long-term study of Alzheimer's in women is the Nun Study, begun in 1986 and presently conducted at the University of Kentucky.

Multi-infarct dementia

MID is responsible for between 15% and 20% of cases of dementia (not counting cases in which it coexists with AD). Unlike AD, MID is more common in men than in women. Diabetes, high blood pressure, a history of smoking, and heart disease are all risk factors for MID. Researchers in Sweden have suggested that MID is underdiagnosed, and may coexist with other dementias more frequently than is presently recognized.

Dementia with Lewy bodies

Dementia with Lewy bodies is now thought to be the second most common form of dementia after Alzheimer's disease. But because researchers don't completely understand the relationship between Lewy bodies, AD, and Parkinson's disease, the demographic distribution of this type of dementia is also unclear.

Other dementias

FLD, Pick's disease, Huntington's disease, Parkinson's disease, HIV infection, alcoholism, head trauma, etc. account for about 10% of all cases of dementia. In FLD and Pick's dementia, women appear to be affected slightly more often than men.

Diagnosis

In some cases, a patient's primary physician may be able to diagnose the dementia; in many instances, however, the patient will be referred to a neurologist or a gerontologist (specialist in medical care of the elderly). Distinguishing one disorder from other similar disorders is a process called differential **diagnosis**. The differential diagnosis of dementia is complicated because of the number of possible causes; because more than one cause may be present at the same time; and because dementia can coexist with such other conditions as depression and **delirium**. Delirium is a temporary disturbance of consciousness marked by confusion, restlessness, inability to focus one's attention, hallucinations, or delusions. In elderly people, delirium is frequently a side effect of surgery, medications, infectious illnesses, or dehydration. Delirium can be distinguished from dementia by the fact that delirium usually comes on fairly suddenly (in a few hours or days) and may vary in severity— it is often worse at night. Dementia develops much more slowly, over a period of months or years, and the patient's symptoms are relatively stable. It is possible for a person to have delirium and dementia at the same time.

Another significant diagnostic distinction in elderly patients is the distinction between dementia and age-associated memory impairment (AAMI), which is sometimes called benign senescent forgetfulness. Older people with AAMI have a mild degree of memory loss; they do not learn new information as quickly as younger people, and they may take longer to recall a certain fact or to balance their checkbook. But they do not suffer the degree of memory impairment that characterizes dementia, and they do not get progressively worse.

Clinical interview

The doctor will begin by taking a full history, including the patient's occupation and educational level as well as medical history. The occupational and educational history allows the examiner to make a more accurate assessment of the extent of the patient's memory loss and other evidence of intellectual decline. In some cases, the occupational history may indicate exposure to heavy metals or other toxins. A complete medical history allows the doctor to assess such possibilities as delirium, depression, alcohol-related dementia, dementia related to head injury, or dementia caused by infection. It is particularly important for the doctor to have a list of all the patient's medications, including over-the-counter and alternative herbal preparations, because of the possibility that the patient's symptoms are related to side effects of these substances.

Whenever possible, the examiner will consult the patient's family members or close friends as part of the history-taking process. In many cases, friends and relatives can provide more detailed information about the patient's memory problems and loss of function.

Mental status examination

A mental status examination (MSE) evaluates the patient's ability to communicate, follow instructions, recall information, perform simple tasks involving movement and coordination, as well as his or her emotional state and general sense of space and time. The MSE includes the doctor's informal evaluation of the patient's appearance, vocal tone, facial expressions, posture, and gait as well as formal questions or instructions. A common form that has been used since 1975 is the so-called Folstein Mini-Mental Status Examination, or MMSE. Questions that are relevant to diagnosing dementia include asking the patient to count backward from 100 by 7s, to make change, to name the current President of the United States, to repeat a short phrase after the examiner (such as, "no ifs, ands, or buts"); to draw a clock face or geometric figure, and to follow a set of instructions involving movement (such as, "Show me how to throw a ball" or "Fold this piece of paper and place it under the lamp on the bookshelf.") The examiner may test the patient's abstract reasoning ability by asking him or her

to explain a familiar proverb ("People who live in glass houses shouldn't throw stones," for example) or test the patient's judgment by asking about a problem with a common-sense solution, such as what one does when a prescription runs out.

Neurological examination

A neurological examination includes an evaluation of the patient's cranial nerves and reflexes. The cranial nerves govern the ability to speak as well as sight, hearing, taste, and smell. The patient will be asked to stick out the tongue, follow the examiner's finger with the eyes, raise the eyebrows, etc. The patient is also asked to perform certain actions (such as touching the nose with the eyes closed) that test coordination and spatial orientation. The doctor will usually touch or tap certain areas of the body, such as the knee or the sole of the foot, to test the patient's reflexes. Failure to respond to the touch or tap may indicate damage to certain parts of the brain.

Laboratory tests

Blood and urine samples may be collected in order to rule out such conditions as thyroid deficiency, niacin or vitamin B_{12} deficiency, heavy metal poisoning, liver disease, HIV infection, syphilis, anemia, medication reactions, or kidney failure. A lumbar puncture (spinal tap) may be done to rule out neurosyphilis.

Diagnostic imaging

The patient may be given a **computed tomography** (CT) scan or **magnetic resonance imaging** (MRI) to detect evidence of strokes, disintegration of the brain tissue in certain areas, blood clots or tumors, a buildup of spinal fluid, or bleeding into the brain tissue. Positron-emission tomography (PET) or single-emission computed tomography (SPECT) imaging is not used routinely to diagnose dementia, but may be used to rule out Alzheimer's disease or frontal lobe degeneration if a patient's CT scan or MRI is unrevealing.

Treatments

Reversible and responsive dementias

Some types of dementia are reversible, and a few types respond to specific treatments related to their causes. Dementia related to dietary deficiencies or metabolic disorders is treated with the appropriate vitamins or thyroid medication. Dementia related to HIV infection often responds well to zidovudine (Retrovir), a drug given to prevent the AIDS virus from replicating. Multi-infarct dementia is usually treated by controlling the patient's blood pressure and/or diabetes; while treatments for these disorders cannot undo damage already caused to brain tissue, they can slow the progress of the dementia. Patients with alcohol-related dementia often improve over the long term if they are able to stop drinking. Dementias related to head injuries, hydrocephalus, and tumors are treated by surgery.

It is important to evaluate and treat elderly patients for depression, because the symptoms of depression in older people often mimic dementia. This condition is sometimes called pseudodementia. In addition, patients who suffer from both depression and dementia often show some improvement in intellectual functioning when the depression is treated. The medications most often used for depression related to dementia are the selective serotonin reuptake inhibitors (SSRIs) **paroxetine** and **sertraline**. The mental status examination should be repeated after six–12 weeks of antidepressant medication.

Irreversible dementias

As of 2001, there are no medications or surgical techniques that can cure Alzheimer's disease, the frontal lobe dementias, MID, or dementia with Lewy bodies. There are also no "magic bullets" that can slow or stop the progression of these dementias. There is, however, one medication, Aricept, that is being used to halt the progression of Alzheimer's disease. In addition, another medication called **galantamine** (Reminyl) is also being used to treat the symptoms of Alzheimer's disease. Patients may be given medications to ease the depression, anxiety, sleep disturbances, and other behavioral symptoms that accompany dementia, but most physicians prescribe relatively mild dosages in order to minimize the troublesome side effects of these drugs. Dementia with Lewy bodies appears to respond better to treatment with the newer antipsychotic medications than to treatment with such older drugs as **haloperidol** (Haldol).

Patients in the early stages of dementia can often remain at home with some help from family members or other caregivers, especially if the house or apartment can be fitted with safety features (handrails, good lighting, locks for cabinets containing potentially dangerous products, nonslip treads on stairs, etc.). Patients in the later stages of dementia, however, usually require skilled care in a nursing home or hospital.

Prognosis

The prognosis for reversible dementia related to nutritional or thyroid problems is usually good once the cause has been identified and treated. The prognoses for dementias related to alcoholism or HIV infection depend on the patient's age and the severity of the underlying disorder.

The prognosis for the irreversible dementias is gradual deterioration of the patient's functioning ending in death. The length of time varies somewhat. Patients with Alzheimer's disease may live from two–20 years with the disease, with an average of seven years. Patients with frontal lobe dementia or Pick's disease live on average between five and 10 years after diagnosis. The course of Creutzfeldt-Jakob disease is much more rapid, with patients living between five and 12 months after diagnosis.

Prevention

The reversible dementias related to thyroid and nutritional disorders can be prevented in many cases by regular physical checkups and proper attention to diet. Dementias related to toxic substances in the workplace may be prevented by careful monitoring of the work environment and by substituting less hazardous materials or substances in manufacturing processes. Dementias caused by infectious diseases are theoretically preventable by avoiding exposure to the prion, spirochete, or other disease agent. Multi-infarct dementia may be preventable in some patients by attention to diet and monitoring of blood pressure. Dementias caused by abnormalities in the structure of the brain are not preventable as of 2002.

With regard to genetic factors, tests are now available for the APOE gene implicated in late-onset Alzheimer's, but these tests are used primarily in research instead of clinical practice. One reason is that the test results are not conclusive; about 20% of people who eventually develop AD do not carry this gene. Another important reason is the ethical implications of testing for a disease that presently has no cure. These considerations may change, however, if researchers discover better treatments for primary dementia, more effective preventive methods, or more reliable genetic markers.

See also Respite care

Resources

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Alzheimer's Association. 919 North Michigan Avenue, Suite 1000, Chicago, IL 60611. (800) 272-3900.

Alzheimer's Disease International. 45/46 Lower Marsh, London SE1 7RG, United Kingdom. (+44 20) 7620 3011. E-mail: adi@alz.co.uk. <www.alz.co.uk>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

National Institute of Neurological Disorders and Stroke (NINDS). Building 31, Room 8A06, 9000 Rockville Pike, Bethesda, MD 20892. (301) 496-5751. <www.ninds.nih.gov>.

National Institute on Aging Information Center. P.O. Box 8057, Gaithersburg, MD 20898. (800) 222-2225 or (301) 496-1752.

National Organization for Rare Disorders (NORD). P. O. Box 8923, New Fairfield, CT 06812. (800) 447-6673 or (203) 746-6518.

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Dementia of the Alzheimer's type *see* **Alzheimer's disease**

Denial

Definition

Denial is the refusal to acknowledge the existence or severity of unpleasant external realities or internal thoughts and feelings.

Theory of denial

In psychology, denial is a concept originating with the psychodynamic theories of Sigmund Freud.

KEY TERMS

Antisocial behavior—Behavior characterized by high levels of anger, aggression, manipulation, or violence.

Cognitive-behavioral therapies—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

Defense mechanisms—Indirect strategies used to reduce anxiety rather than directly facing the issues causing the anxiety.

Dependent personality disorder—Personality disorder characterized by a constant, unhealthy need to be liked and appreciated by others at all costs.

Ego—In Freudian psychology, the conscious, rational part of the mind that experiences and reacts to the outside world.

Humanistic and existential therapies—Therapies that focus on achieving one's full potential, guided by subjective experience.

Id—A construct in Freudian psychodynamic theory that represents the irrational, self-centered aspects of human thought.

Motivational enhancement therapy—Therapy that focuses on increasing motivation for change by empathically comparing and contrasting the consequences and benefits of changing or not changing.

Narcissistic personality disorder—Personality characterized by continually exaggerating one's own positive qualities and refusing to recognize personal defects or flaws.

Psychoanalytic therapy—Therapy based on the psychodynamic theory of Sigmund Freud.

Psychodynamic—Referring to the motivational forces, unconscious as well as conscious, that form human attitudes and behavior.

Superego—According to Freud, the part of the mind that represents traditional parental and societal values. The superego is the source of guilt feelings.

According to Freud, three mental dynamics, or motivating forces, influence human behavior: the id, ego, and superego. The id consists of basic survival instincts and what Freud believed to be the two dominant human drives: sex and aggression. If the id were the only influence on behavior, humans would exclusively seek to increase pleasure, decrease pain, and achieve immediate gratification of desires. The ego consists of logical and rational thinking. It enables humans to analyze the realistic risks and benefits of a situation, to tolerate some pain for future profit, and to consider alternatives to the impulse-driven behavior of the id. The superego consists of moralistic standards and forms the basis of the conscience. Although the superego is essential to a sense of right and wrong, it can also include extreme, unrealistic ideas about what one should and should not do.

These three forces all have different goals (id, pleasure; ego, reality; superego, morality) and continually strive for dominance, resulting in internal conflict. This conflict produces anxiety. The ego, which functions as a mediator between the two extremes of the id and the superego, attempts to reduce this anxiety by using defense mechanisms. Defense mechanisms are indirect ways of dealing or coping with anxiety, such as explaining problems away or blaming others for problems. Denial is one of many defense mechanisms. It entails ignoring or refusing to believe an unpleasant reality. Defense mechanisms protect one's psychological well-being in traumatic situations, or in any situation that produces anxiety or conflict. However, they do not resolve the anxiety-producing situation and, if overused, can lead to psychological disorders. Although Freud's model of the id, ego, and superego is not emphasized by most psychologists today, defense mechanisms are still regarded as potentially maladaptive behavioral patterns that may lead to psychological disorders.

Examples of denial

Death is a common occasion for denial. When someone learns of the sudden, unexpected death of a loved one, at first he or she may not be able to accept the reality of this loss. The initial denial protects that person from the emotional shock and intense **grief** that often accompanies news of death. Chronic or terminal illnesses also encourage denial. People with such illnesses may think, "It's not so bad; I'll get over it," and refuse to make any lifestyle changes.

Denial can also apply to internal thoughts and feelings. For instance, some children are taught that anger is wrong in any situation. As adults, if these individuals experience feelings of anger, they are likely to deny their feelings to others. Cultural standards and expectations can encourage denial of subjective experience. Men who belong to cultures with extreme notions of masculinity may view fear as a sign of weakness and deny internal feelings of fear. The Chinese culture is thought to discourage the acknowledgment of mental illness, resulting

in individuals denying their psychological symptoms and often developing physical symptoms instead.

Certain **personality disorders** tend to be characterized by denial more than others. For example, those with **narcissistic personality disorder** deny information that suggests they are not perfect. Antisocial behavior is characterized by denial of the harm done to others (such as with sexual offenders or substance abusers).

Denial can also be exhibited on a large scale—among groups, cultures, or even nations. Lucy Bregman gives an example of national denial of imminent mortality in the 1950s: school children participated in drills in which they hid under desks in preparation for atomic attacks. Another example of large-scale denial is the recent assertion by some that the World War II Holocaust never occurred.

Treatment of denial

Denial is treated differently in different types of therapy. In psychoanalytic therapy, denial is regarded as an obstacle to progress that must eventually be confronted and interpreted. Timing is important, however. Psychoanalytic therapists wait until clients appear emotionally ready or have some degree of insight into their problems before confronting them. In the humanistic and existential therapies, denial is considered the framework by which clients understand their world. Not directly confronting denial, therapists assist clients in exploring their world view and considering alternative ways of being. In cognitive-behavioral therapies, denial is not regarded as an important phenomenon. Rather, denial would suggest that an individual has not learned the appropriate behaviors to cope with a stressful situation. Therapists assist individuals in examining their current thoughts and behaviors and devising strategic ways to make changes.

Traditional treatment programs for substance abuse and other addictions view denial as a central theme. Such programs teach that in order to overcome **addiction**, one must admit to being an alcoholic or addict. Those who are unable to accept such labels are informed they are in denial. Even when the labels are accepted, individuals are still considered to be in denial if they do not acknowledge the severity of their addictions. From this perspective, progress cannot be made until individuals recognize the extent of their denial and work toward acceptance. However, there is much controversy in the field of addictions regarding the role of denial and how it should be addressed. Traditional programs **stress** direct confrontation. Other professionals do not insist on the acceptance of labels. They believe that denial should be worked through more subtly, empathically focusing on the personal reasons surrounding denial and seeking to strengthen the desire to change. This subtle form of addressing denial is known as motivational enhancement therapy, and can be used with other types of disorders as well.

See also Grief; Psychoanalysis; Psychodynamic psychotherapy; Substance abuse and related disorders

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The American Psychoanalytic Association. 309 East 49th Street, New York, New York 10017. (212) 752-0450. <http://www.aapsa.org>.

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Depakene *see* **Valproic acid**

Depakote *see* **Divalproex sodium**

Dependent personality disorder

Definition

Dependent personality disorder is characterized by an excessive need to be taken care of or depend upon others. Persons with this disorder are typically submissive and display clinging behavior toward those from whom they fear being separated.

Dependent personality disorder is one of several **personality disorders** listed in the newest edition of the stan-

KEY TERMS

Millon Clinical Multiaxial Inventory (MCMI-II)—A self-report instrument designed to help the clinician assess DSM-IV-related personality disorders and clinical syndromes. It provides insight into 14 personality disorders and 10 clinical syndromes.

Minnesota Multiphasic Personality Inventory (MMPI-2)—A comprehensive assessment tool widely used to diagnose personality disorders.

Rorschach Psychodiagnostic Test—This series of 10 "ink blot" images allows the patient to project their interpretations which can be used to diagnose particular disorders.

Thematic Apperception Test (TAT)—A projective test using stories and descriptions of pictures to reveal some of the dominant drives, emotions, sentiments, conflicts, and complexes of a personality.

dard reference guide: ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM-IV-TR*.

Description

Persons with dependent personality disorder are docile, passive, and nonassertive. They exert a great deal of energy to please others, are self-sacrificing, and constantly attempt to elicit the approval of others. They are reluctant to express disagreement with others, and are often willing to go to abnormal lengths to win the approval of those on whom they rely. They are easily influenced and can be taken advantage of easily. This **compliance** and reliance upon others leads to a subtle message that someone should assume responsibility for significant areas of the patient's life. This is often displayed as helplessness, even for completion of seemingly simple tasks.

Patients with dependent personality disorder have a low level of confidence in their own intelligence and abilities. They often have difficulty making decisions and undertaking projects on their own. They are prone to be pessimistic, self-doubting, and belittle their own accomplishments. They shy away from responsibility in occupational settings.

Affected individuals are uneasy being alone and are preoccupied with the fear of being abandoned or rejected by others. Their moods are characterized by frequent bouts of anxiety or fearfulness; generally, their demeanor is sad. Their style of thinking is naïve, uncritical, and lacks discretion.

Causes and symptoms

Causes

It is commonly thought that the development of dependence in these individuals is a result of overinvolvement and intrusive behavior by their primary caretakers. Caretakers may foster dependence in the child to meet their own dependency needs, and may reward extreme loyalty but reject attempts the child makes towards independence. Families of those with dependent personality disorder are often do not express their emotions and are controlling; they demonstrate poorly defined relational roles within the family unit.

Individuals with dependent personality disorder often have been socially humiliated by others in their developmental years. They may carry significant doubts about their abilities to perform tasks, take on new responsibilities, and generally function independently of others. This reinforces their suspicions that they are incapable of living autonomously. In response to these feelings, they portray a helplessness that elicits caregiving behavior from some people in their lives.

Symptoms

DSM-IV-TR specifies eight diagnostic criteria for dependent personality disorder. Individuals with this disorder:

- Have difficulty making common decisions. These individuals typically need an excessive amount of advice and reassurance before they can make even simple decisions, such as the clothing to wear on a given day.
- Need others to assume responsibility for them. Because they view themselves as incapable of being autonomous, they withdraw from adult responsibilities by acting passive and helpless. They allow others to take the initiative for many areas of their life. Adults with this disorder typically depend on a parent or spouse to make major decisions for them, such as where to work, to live, or with whom to be friends.
- Have difficulty expressing disagreement with others. Disagreeing with others is often viewed as too risky. It might sever the support or approval of those they upon whom they depend. They are often overly agreeable, as they fear alienating other people.
- Have difficulty initiating or doing things on their own. They lack self-confidence and believe they need help to begin or sustain tasks. They often present themselves as

inept and unable to understand or accomplish the task at hand.

- Go to excessive lengths to obtain support or nurturing from others. They may even volunteer to do unpleasant tasks if they believe that doing so will evoke a positive response from others. They may subject themselves to great personal sacrifice or tolerate physical, verbal, or sexual **abuse** in their quest to get what they believe they need from others.
- Feel helpless when alone. Because they feel incapable of caring for themselves, they experience significant anxiety when alone. To avoid being alone, they may be with people in whom they have little interest.
- Quickly seek a new relationship when a previous one ends. When a marriage, dating, or other close relationship ends, there is typically an urgency to find a new relationship that will provide the support of the former relationship.
- Are preoccupied with fears of being left to take care of themselves. Their greatest fear is to be left alone and to be responsible for themselves. Even as adults, their dependence upon others may appear childlike.

Demographics

Dependent personality disorder should rarely, if ever, be diagnosed in children or adolescents because of their dependence on others because of their age and developmental limitations.

Diagnosis

Age and cultural factors should be considered in diagnosing dependent personality disorder. Certain cultural norms suggest a submissive, polite, or dependent posture in relating to the opposite sex, or authority figures. Dependent personality disorder should only be diagnosed when it meets the above criteria and is clearly outside one's cultural norms.

The **diagnosis** of dependent personality disorder is based on a clinical interview to assess symptomatic behavior. Other assessment tools helpful in confirming the diagnosis of dependent personality disorder include:

- **Minnesota Multiphasic Personality Inventory** (MMPI-2)
- Millon Clinical Multi-axial Inventory (MCMI-II)
- Rorschach Psychodiagnostic Test
- **Thematic Appreception Test** (TAT)

For a person to be diagnosed with dependent personality disorder, at least five of the eight symptoms described above must be the present, and these symptoms must begin by early adulthood and be evident in a variety of contexts.

The diagnosis of dependent personality disorder must be distinguished from **borderline personality disorder**, as there are common characteristics. Borderline personality disorder is characterized by fear of abandonment, as well, but with feelings of emptiness and rage. In contrast, the dependent personality responds to this fear of abandonment with submissiveness, and searches for a replacement relationship to maintain dependency.

Likewise, persons with histrionic personality disorder have a strong need for reassurance and approval, and may appear childlike in their clinging behavior. Histrionics are characterized by a gregarious demeanor and make active demands for attention, whereas dependents respond with docile and self-deprecating behavior.

The **avoidant personality disorder** can also be confused with dependent personality disorder. Both are characterized by feelings of inadequacy, an oversensitivity to criticism, and a frequent need for assurance. However, patients with avoidant personality disorder typically have such an intense fear of rejection that they will instinctively withdraw until they are certain of acceptance. People with dependent personality disorder, in contrast, actually seek out contact with others because they need the approval of others.

Treatments

The general goal of treatment of dependent personality disorder is to increase the individual's sense of autonomy and ability to function independently.

Psychodynamically oriented therapies

A long-term approach to psychodynamic treatment can be successful, but may lead to heightened dependencies and difficult separation in the therapeutic relationship over time. The preferred approach is a time-limited treatment plan consisting of a predetermined number of sessions. This has been proved to facilitate the exploration process of dependency issues more effectively than long-term therapy in most patients.

Cognitive-behavioral therapy

Cognitive-behavioral approaches attempt to increase the affected person's ability to act independently of others, improve their self-esteem, and enhance the quality of their interpersonal relationships. Often, patients will play an active role in setting goals. Methods often used in **cognitive-behavioral therapy** (CBT) include assertiveness and **social skills training** to help reduce reliance on others, including the therapist.

Interpersonal therapy

Treatment using an interpersonal approach can be useful because the individual is usually receptive to treatment and seeks help with interpersonal relationships. The therapist would help the patient explore their long-standing patterns of interacting with others, and understand how these have contributed to dependency issues. The goal is to show the patient the high price they pay for this dependency, and to help them develop healthier alternatives. **Assertiveness training** and learning to identify feelings is often used to improve interpersonal behavior.

Group therapy

When a person is highly motivated to see growth, a more interactive therapeutic group can be successful in helping him/her to explore passive-dependent behavior. If the individual is socially reluctant or impaired in his/her assertiveness, decision-making, or negotiation, a supportive decision-making group would be more appropriate. Time-limited assertiveness-training groups with clearly defined goals have been proven to be effective.

Family and marital therapy

Individuals with dependent personality disorder are usually brought to therapy by their parents. They are often young adults who are struggling with neurotic or psychotic symptoms. The goal of **family therapy** is often to untangle the enmeshed family relationships, which usually elicits considerable resistance by most family members unless all are in therapy.

Marital therapy can be productive in helping couples reduce the anxiety of both partners who seek and meet dependency needs that arise in the relationship.

Medications

Individuals with dependent personality disorder can experience anxiety and depressive disorders as well. In these cases, it may occasionally prove useful to use antidepressants or anti-anxiety agents. Unless the anxiety or depression is considered worthy of a primary diagnosis, medications are generally not recommended for treatment of the dependency issues or the anxiety or depressive responses. Persons with dependent personality disorder may become overly dependent on any medication used.

Prognosis

The general prognosis for individuals with dependent personality disorder is good. Most people with this disorder have had a supportive relationship with at least one parent. This enables them to engage in treatment to varying degrees and to explore the source of their dependent behavior. If persons who enter treatment can learn to become more autonomous, improved functioning can be expected.

Prevention

Since dependent personality disorder originates in the patient's family, the only known preventive measure is a nurturing, emotionally stimulating, and expressive caregiving environment.

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American Psychiatric Association. 1400 K Street NW, Washington D.C. 20005 <http://www.psych.org>.

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Depersonalization

Definition

Depersonalization is a mental state in which a person feels detached or disconnected from his or her personal identity or self. This may include the sense that one is "outside" oneself, or is observing one's own actions, thoughts or body.

Description

A person experiencing depersonalization may feel so detached that he or she feels more like a robot than a human being. However, the person always is aware that this is just a feeling; there is no delusion that one is a lifeless robot or that one has no personal identity. The sense of detachment that characterizes the state may result in mood shifts, difficulty thinking, and loss of some sensations— a state that can be described as numbness or sensory anesthesia. Twice as many women as men are treated for depersonalization, which can last from a few seconds to years. Episodes may increase after traumatic events such as exposure to combat, accidents or other forms of violence or **stress**. Treatment is difficult and the state is often chronic, although it may occur during discrete periods or increase and decrease in intensity over time. Individuals with depersonalization often feel that events and the environment are unreal or strange, a state called derealization.

See also Acute stress disorder; Dissociation and dissociative disorders; Post-traumatic stress disorder; Schizophrenia

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Depersonalization disorder

Definition

Depersonalization is a state in which the individual ceases to perceive the reality of the self or the environment. The patient feels that his or her body is unreal, is changing, or is dissolving; or that he or she is outside of the body.

Depersonalization disorder is classified by the ***Diagnostic and Statistical Manual of Mental Disorders***, 4th Edition, text Revision, also known as the *DSM-IV-TR* as one of the dissociative disorders. These are mental disorders in which the normally well-integrated functions of memory, identity, perception, and consciousness are separated (dissociated). The dissociative disorders are usually associated with trauma in the recent or distant past, or with an intense internal conflict that forces the mind to separate incompatible or unacceptable knowledge, information, or feelings. In depersonalization disorder, the patient's self-perception is disrupted. Patients feel as if they are external observers of their own lives, or that they are detached from their own bodies. Depersonalization disorder is sometimes called "depersonalization neurosis."

Depersonalization as a symptom may occur in **panic disorder**, **borderline personality disorder**, **post-traumatic stress disorder** (PTSD), **acute stress disorder**, or another dissociative disorder. The patient is not given the **diagnosis** of depersonalization disorder if the episodes of depersonalization occur only during panic attacks or following a traumatic stressor.

The symptom of depersonalization can also occur in normal individuals under such circumstances as sleep deprivation, the use of certain anesthetics, experimental conditions in a laboratory (experiments involving weightlessness, for example), and emotionally stressful situations (such as taking an important academic examination or being in a traffic accident). One such example involves some of the rescue personnel from the September 11, 2001 terrorist attacks on the World Trade Center and the Pentagon. These individuals experienced episodes of depersonalization after a day and a half without sleep. A more commonplace example is the use of nitrous oxide, or "laughing gas" as an anesthetic during oral surgery. Many dental patients report a sense of unreality or feeling of being outside their bodies during nitrous oxide administration.

To further complicate the matter, depersonalization may be experienced in different ways by different individuals. Common descriptions include a feeling of being outside one's body; "floating on the ceiling looking down at myself" feeling as if one's body is dissolving or changing; feeling as if one is a machine or robot; "unreal" feeling that one is in a dream or that one"is on automatic pilot." Most patients report a sense of emotional detachment or uninvolvement, or a sense of emotional numbing. Depersonalization differs from "derealization," which is a dissociative symptom in which people perceive the external world as unreal, dreamlike, or changing. The various ways that people experience depersonalization are related to their bodies or their sense of self.

Depersonalization is a common experience in the general adult population. However, when a patient's symptoms of depersonalization are severe enough to cause significant emotional distress, or interfere with normal functioning, the criteria of the *DSM-IV-TR* for "depersonalization disorder" are met.

Description

A person suffering from depersonalization disorder experiences subjective symptoms of unreality that make him or her uneasy and anxious. "Subjective" is a word

KEY TERMS

Abuse—Physical, emotional, or sexual harm.

Depersonalization—A dissociative symptom in which the patient feels that his or her body is unreal, is changing, or is dissolving; or that he or she is outside the body.

Depersonalization neurosis—Another name for depersonalization disorder.

Derealization—A dissociative symptom in which the external environment is perceived as unreal or dreamlike.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Dissociative disorders—A group of disorders marked by the separation (dissociation) of perception, memory, personal identity, and consciousness. Depersonalization disorder is one of five dissociative disorders defined by *DSM-IV-TR*.

Hypothalamic-pituitary-adrenal (HPA) system—A part of the brain involved in the human stress response. The HPA system releases cortisol, the primary human stress hormone, and neurotransmitters that activate other brain structures associated with the "fight-or-flight" reaction. The HPA system appears to function in abnormal ways in patients diagnosed with depersonalization disorder. It is sometimes called the HPA axis.

Reality testing—A phrase that refers to a person's ability to distinguish between subjective feelings and objective reality. A person who knows that their body is real even though they may be experiencing it as unreal, for example, is said to have intact reality testing. Intact reality testing is a *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision (also known as the *DSM-IV-TR*) criterion for depersonalization disorder.

Selective serotonin reuptake inhibitor—Commonly prescribed drugs for treating depression. SSRIs affect the chemicals that nerves in the brain use to send messages to one another. These chemical messengers (neurotransmitters) are released by one nerve cell and taken up by others. Neurotransmitters not taken up by other nerve cells are taken up by the same cells that released them. This process is termed "reuptake." SSRIs work by inhibiting the reuptake of serotonin, an action which allows more serotonin to be taken up by other nerve cells.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Stress—A physical and psychological response that results from being exposed to a demand or pressure.

Stressor—A stimulus or event that provokes a stress response in an organism. Stressors can be categorized as acute or chronic, and as external or internal to the organism.

Subjective—Referring to a person's unique internal thoughts and feelings, as distinct from the objects of those thoughts and feelings in the eternal world.

Tricyclic antidepressants (TCAs)—Antidepressant medications that have the common characteristic of a three-ring nucleus in their chemical structure. Imipramine and amitriptyline are examples of tricyclic antidepressants.

that refers to the thoughts and perceptions inside an individual's mind, as distinct from the objects of those thoughts and perceptions outside the mind. Because depersonalization is a subjective experience, many people who have chronic or recurrent episodes of depersonalization are afraid others will not understand if they try to describe what they are feeling, or will think they are "crazy." As a result, depersonalization disorder may be underdiagnosed because the symptom of depersonalization is underreported.

Causes and symptoms

Causes

Depersonalization disorder, like the dissociative disorders in general, has been regarded as the result of severe **abuse** in childhood. This can be of a physical, emotional, and/or sexual nature.

Findings in 2002 indicate that emotional abuse in particular is a strong predictor of depersonalization disorder in adult life, as well as of depersonalization as a

symptom in other mental disorders. Analysis of one study of 49 patients diagnosed with depersonalization disorder indicated much higher scores than the control subjects for the total amount of emotional abuse endured and for the maximum severity of this type of abuse. The researchers concluded that emotional abuse has been relatively neglected by psychiatrists compared to other forms of childhood trauma.

It is thought that abuse in childhood or trauma in adult life may account for the distinctive cognitive (knowledge-related) profile of patients with depersonalization disorder. These patients have significant difficulties focusing their attention, with spatial reasoning, and with short-term visual and verbal memory. However, they have intact reality testing. (Reality testing refers to a person's ability to distinguish between their internal experiences and the objective reality of persons and objects in the outside world.) Otherwise stated, a patient with depersonalization disorder may experience his/her body as unreal, but knows that "feelings aren't facts." The *DSM-IV-TR* specifies intact reality testing as a diagnostic criterion for depersonalization disorder.

The causes of depersonalization disorder are not completely understood. Recent advances in **brain** imaging and other forms of neurological testing, however, have confirmed that depersonalization disorder is a distinct diagnostic entity and should not be considered a subtype of PTSD.

No specific genes have been associated with susceptibility to depersonalization disorder as of early 2002. It is possible that a genetic factor will be identified in the future.

NEUROBIOLOGICAL. In the past few years, several features of depersonalization disorder have been traced to differences in brain functioning. A group of British researchers found that the emotional detachment that characterizes depersonalization is associated with a lower level of nerve cell responses in regions of the brain that are responsible for emotional feeling; an increased level of nerve cell responses was found in regions of the brain related to emotional regulation.

A group of American researchers concluded that patients with depersonalization disorder had different patterns of response to tests of the hypothalamic-pituitary-adrenal axis (HPA, the part of the brain involved in the "fight-or-flight" reaction to **stress**) than did patients with PTSD. Other tests by the same research team showed that patients with depersonalization disorder can be clearly distinguished from patients with major depression by tests of the functioning of the HPA axis.

Other neurobiological studies involving **positron emission tomography** (PET) measurements of glucose (sugar) metabolism in different areas of the brain found that patients with depersonalization disorder appear to have abnormal functioning of the sensory cortex. The sensory cortex is the part of the brain that governs the senses of sight, hearing, and perceptions of the location of one's body in space. These studies indicate that depersonalization is a symptom that involves differences in sensory perception and subjective experiences.

HISTORICAL. Depersonalization disorder may be a reflection of changes in people's sense of self or personal identity within Western cultures since the eighteenth century. Historians of psychiatry have noted that whereas some mental disorders, such as depression, have been reported since the beginnings of Western medicine, no instances of the dissociative disorders were recorded before the 1780s. It seems that changes in social institutions and the structure of the family since the mid-eighteenth century may have produced a psychological structure in Westerners that makes individuals increasingly vulnerable to self disorders—as they are now called. Experiences of the unreality of one's body or one's self, such as those that characterize depersonalization disorder, presuppose a certain notion of how the self is presumed to feel. The emphasis on individualism and detachment from one's family is a mark of adult maturity in contemporary Western societies that appears to be a contributing factor to the frequency of dissociative symptoms and disorders.

Symptoms

The symptoms of depersonalization disorder have been described earlier. Although *DSM-IV-TR* does not specify a list of primary symptoms of depersonalization, British clinicians generally consider the triad of emotional numbing, changes in visual perception, and altered experience of one's body to be important core symptoms of depersonalization disorder.

DSM-IV-TR notes that patients with depersonalization disorder frequently score high on measurements of hypnotizability.

Demographics

The lifetime prevalence of depersonalization disorder in the general population is unknown, possibly because many people are made anxious by episodes of depersonalization and afraid to discuss them with a primary care physician. One survey done by the National Institutes of Mental Health (NIMH) indicates that about half of the adults in the U.S. have had one or two brief episodes of depersonalization in their lifetimes, usually

resulting from severe stress. About a third of people exposed to life-threatening dangers develop brief periods of depersonalization, as do 40% of psychiatric inpatients.

Depersonalization disorder is diagnosed about twice as often in women as in men. It is not known, however, whether this sex ratio indicates that women are at greater risk for the disorder or if they are more likely to seek help for its symptoms, or both. Little information is available about the incidence of the disorder in different racial or ethnic groups.

Diagnosis

The diagnosis of depersonalization disorder is usually a diagnosis of exclusion. The doctor will take a detailed medical history, give the patient a physical examination, and order blood and urine tests in order to rule out depersonalization resulting from epilepsy, substance abuse, medication side effects, or recent periods of sleep deprivation.

There are several standard diagnostic questionnaires that may be given to evaluate the presence of a dissociative disorder. The Dissociative Experiences Scale, or DES, is a frequently administered self-report screener for dissociation. The Structured Clinical Interview for DSM-IV Dissociative Disorders, or SCID-D, can be used to make the diagnosis of depersonalization disorder distinct from the other dissociative disorders defined by *DSM-IV*. The SCID-D is a semi-structured interview, which means that the examiner's questions are open-ended and allow the patient to describe experiences of depersonalization in some detail—distinct from simple "yes" or "no" answers.

In addition to these instruments, a six-item Depersonalization Severity Scale, or DSS, has been developed to discriminate between depersonalization disorder and other dissociative or post-traumatic disorders, and to measure the effects of treatment in patients.

Treatments

Depersonalization disorder sometimes resolves on its own without treatment. Specialized treatment is recommended only if the symptoms are persistent, recurrent, or upsetting to the patient. Insight-oriented **psychodynamic psychotherapy**, **cognitive-behavioral therapy**, and hypnosis have been demonstrated to be effective with some patients. There is, however, no single form of **psychotherapy** that is effective in treating all patients diagnosed with depersonalization disorder.

Medications that have been helpful to patients with depersonalization disorder include the benzodiazepine tranquilizers, such as **lorazepam** (Ativan), **clorazepate** (Tranxene), and **alprazolam** (Xanax), and the tricyclic antidepressants, such as **amitriptyline** (Elavil), **doxepin** (Sinequan), and **desipramine** (Norpramin). As of 1999, newer, promising medications called selective serotonin reuptake inhibitors (SSRIs) became available. Some SSRIs include **fluoxetine** (Prozac), **sertraline** (Zoloft), and **paroxetine** (Paxil). SSRIs act on brain chemicals that nerve cells use to send messages to each another. These chemical messengers (**neurotransmitters**) are released by one nerve cell and taken up by others. Those that are not taken up by other cells are taken up by the ones that released them. This is called "reuptake." SSRIs work by preventing the reuptake of serotonin—an action which allows more serotonin to be taken up by nerve cells.

Unfortunately, there have been very few well-designed studies comparing different medications for depersonalization disorder. Because depersonalization disorder is frequently associated with trauma, effective treatment must include other stress-related symptoms, as well.

Relaxation techniques have been reported to be a beneficial adjunctive treatment for persons diagnosed with depersonalization disorder, particularly for those who are worried about their sanity.

Prognosis

The prognosis for recovery from depersonalization disorder is good. Most patients recover completely, particularly those who developed the disorder in connection with traumas that can be explored and resolved in treatment. A few patients develop a chronic form of the disorder; this is characterized by periodic episodes of depersonalization in connection with stressful events in their lives.

Prevention

Some clinicians think that depersonalization disorder has an undetected onset in childhood, even though most patients first appear for treatment as adolescents or young adults. Preventive strategies could include the development of screening techniques for identifying children at risk, as well as further research into the effects of emotional abuse on children.

It is also hopeful that further neurobiological research will lead to the development of medications or other treatment modalities for preventing, as well as treating, depersonalization.

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ORGANIZATIONS

International Society for the Study of Dissociation (ISSD). 60 Revere Drive, Suite 500, Northbrook, IL 60062. (847) 480-0899. Fax: (847) 480-9282. <www.issd.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

National Organization for Rare Disorders, Inc. P. O. Box 8923, New Fairfield, CT 06812-8923. (203) 746-6518. <www.rarediseases.org>.

Society for Traumatic Stress Studies. 60 Revere Dr., Ste. 500, Northbrook, IL 60062. (708) 480-9080.

Rebecca J. Frey, Ph.D.

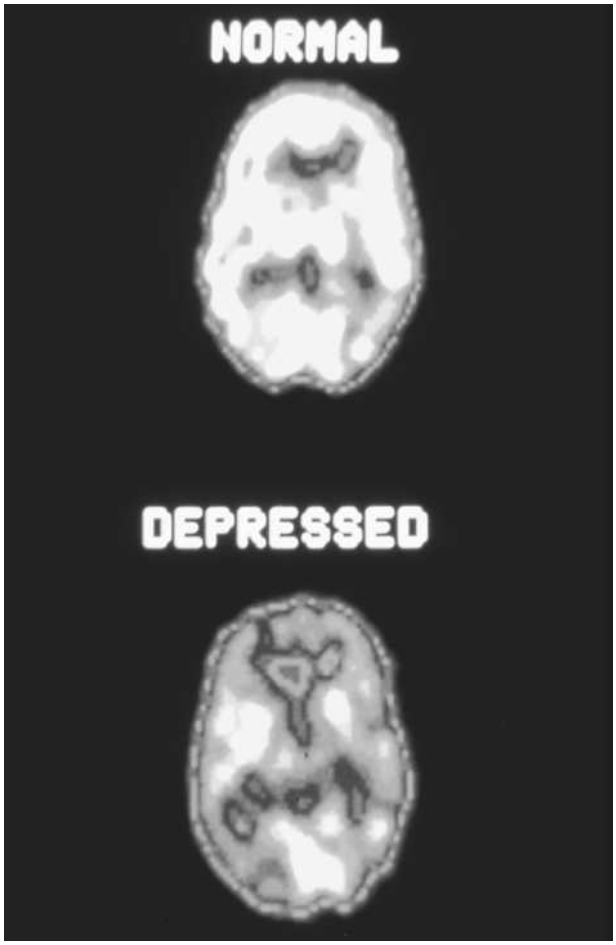
Depression and depressive disorders

Definition

Depression or depressive disorders (unipolar depression) are mental illnesses characterized by a profound and persistent feeling of sadness or despair and/or a loss of interest in things that were once pleasurable. Disturbance in sleep, appetite, and mental processes are a common accompaniment.

Description

Everyone experiences feelings of unhappiness and sadness occasionally. But when these depressed feelings start to dominate everyday life and cause physical and mental deterioration, they become what are known as depressive disorders. Each year in the United States, depressive disorders affect an estimated 17 million people at an approximate annual direct and indirect cost of $53 billion. One in four women is likely to experience an episode of severe depression in her lifetime, with a



Two false-colored positron emission tomography (PET) scans of human brains. At the top is the brain of a healthy person, and below that is the brain of a depressed person. *(NIH/Science Source, National Audubon Society Collection/ Photo Researchers, Inc. Reproduced by permission.) See color insert for color version.*

10–20% lifetime prevalence, compared to 5–10% for men. The average age a first depressive episode occurs is in the mid-20s, although the disorder strikes all age groups indiscriminately, from children to the elderly.

There are two main categories of depressive disorders: **major depressive disorder** and **dysthymic disorder**. Major depressive disorder is a moderate to severe episode of depression lasting two or more weeks. Individuals experiencing this major depressive episode may have trouble sleeping, lose interest in activities they once took pleasure in, experience a change in weight, have difficulty concentrating, feel worthless and hopeless, or have a preoccupation with death or **suicide**. In children, the major depression may appear as irritability.

While major depressive episodes may be acute (intense but short-lived), dysthymic disorder is an ongoing, chronic depression that lasts two or more years (one or more years in children) and has an average duration of 16 years. The mild to moderate depression of dysthymic disorder may rise and fall in intensity, and those afflicted with the disorder may experience some periods of normal, non-depressed mood of up to two months in length. Its onset is gradual, and dysthymic patients may not be able to pinpoint exactly when they started feeling depressed. Individuals with dysthymic disorder may experience a change in sleeping and eating patterns, low self-esteem, **fatigue**, trouble concentrating, and feelings of hopelessness.

Depression can also occur in **bipolar disorder**, a mood disorder that causes radical emotional changes and mood swings, from manic highs to depressive lows. The majority of bipolar individuals experience alternating episodes of mania and depression.

Some theories about the causes of depression

The causes behind depression are complex and not yet fully understood. While an imbalance of certain neurotransmitters—the chemicals in the **brain** that transmit messages between nerve cells— are thought to be key to depression, external factors such as upbringing (more so in dysthymia than major depression) may be as important. For example, it is speculated that, if an individual is abused and neglected throughout childhood and adolescence, a pattern of low self-esteem and negative thinking may emerge. From that, a lifelong pattern of depression may follow.

Depression is also associated with an imbalance of cortisol, the main hormone secreted by the adrenal glands. Other physiological factors sometimes associated with depression include viral infections, low thyroid hormone levels, and biological rhythms, including women's menstrual cycles—depression is a prominent symptom of premenstrual syndrome (PMS).

Heredity seems to play a role in the development of depressive disorders. Individuals with major depression in their immediate family are up to three times more likely to have the disorder themselves. It would seem that biological and genetic factors may make certain individuals pre-disposed or prone to depressive disorders, but environmental circumstances may often trigger the disorder.

External stressors and significant life changes, such as chronic medical problems, death of a loved one, divorce or estrangement, miscarriage, or loss of a job, can also result in a form of depression known as **adjustment disorder**. Although periods of adjustment disorder usually resolve themselves, occasionally they may evolve into a major depressive disorder.

ANTIDEPRESSANT DRUGS	
Brand Name (Generic Name)	***Possible Common Side Effects Include:***
Desyrel (trazodone hydrochloride)	Allergic skin reactions, blurred vision, decreased appetite, fluid retention, headache
Effexor (venlafaxine hydrochloride)	Diarrhea, dizziness, gas, headache, insommia, rash, vomiting
Elavil (amitriptyline hydrochloride)	Constipation, dizziness, high blood pressure, fever, nausea, rash, weight gain or loss
Nardil (phenelzine sulfate)	Dry mouth, fatigue, headache, muscle spasms, tremors
Norpramin (desipramine hydrochloride)	Blurred vision, cramps, hallucinations, hair loss, vomiting
Pamelor (nortriptyline hydrochloride)	Diarrhea, fatigue, headache, decreased coordination
Paxil (paroxetine hydrochloride)	Cold symptoms, drowsiness, nervousness, stomach pain
Prozac (fluoxetine hydrochloride)	Bronchitis, drowsiness, fatigue, nausea, tremors
Sinequan (doxepin hydrochloride)	Bruising, constipation, fluid retention, itching, increased heartbeat
Surmontil (trimipramine maleate)	Disorientation, flushing, headache, nausea, vomiting
Tofranil (imipramine hydrochloride)	Bleeding sores, fever, hives, decreased coordination
Travil	Asthma, diarrhea, dizziness, fatigue, seizures
Wellbutrin (bupropion hydrochloride)	Agitation, dry mouth, headache, nausea, rash
Zoloft (sertraline)	Diarrhea, fainting, gas, headache, nervousness

Some of the antidepressant medications that may be prescribed and some of their potential side effects. *(Stanley Publishing.)*

Paula Anne Ford-Martin, M.A.

Desensitization *see* **Systematic desensitization**

Desipramine

Definition

Desipramine is an antidepressant drug used to elevate mood and promote recovery of a normal range of emotions in patients with depressive disorders. In addition, desipramine has uses in a number of other psychiatric and medical conditions. In the United States, the drug is also known by its brand name, Norpramin.

Purpose

Desipramine is known principally as an antidepressant drug used to promote recovery of depressed patients. It also has therapeutic uses in **panic disorder**, pain management, **attention-deficit/hyperactivity disorder** (ADHD), sleep attacks (**narcolepsy** and cataplexy), **binge eating** (bulimia), and in cocaine craving in the treatment of **addiction**.

Description

Desipramine is one of the tricyclic antidepressants, so-called because of the three-ring chemical structure common to these drugs. Until the late 1980s, desipramine and other tricyclic antidepressants, such as **imipramine**, formed the mainstay of the pharmacological treatment of depressive disorders.

KEY TERMS

Autonomic—The part of the nervous system that governs the heart, involuntary muscles, and glands.

Cataplexy—A symptom of narcolepsy marked by a sudden episode of muscle weakness triggered by strong emotions. The muscle weakness may cause the person's knees to buckle, or the head to drop. In severe cases, the patient may become paralyzed for a few seconds to minutes.

Epilepsy—A neurological disorder characterized by the onset of seizures. Seizures are caused by a disturbance in the electrical activity in the brain and can cause loss of consciousness, muscle spasms, rhythmic movements, abnormal sensory experiences, or altered mental states.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

The therapeutic action of antidepressants is not completely understood. It is known that these drugs boost the levels of certain messenger chemicals, called **neurotransmitters**, which are involved in transmitting signals between nerve cells in the **brain**. This action may help to restore normal emotional feelings by counteracting abnormalities of nerve signal transmission that occur in depressive disorders.

Desipramine is one of a large number of tricyclic antidepressant compounds. Each was developed for somewhat differing pharmacological effects and side-effect profiles. The effects of desipramine are very similar to those of other tricyclics, although some individual patients may find one drug of this group more effective or more tolerable than another. It is available as Norpramin in 10-, 25-, 50-, 75-, 100-, and 150-mg tablets, although generic manufacturers may supply a somewhat different set of dosages.

Recommended dosage

For adults, desipramine is usually administered in dosages of 100–200 mg per day. Doses ranging from 75 mg to 300 mg per day are sometimes prescribed. The initial daily dose is usually low to avoid side effects, and it is usually increased, as necessary, until a therapeutic effect is achieved. Desipramine may be administered in divided doses or a single daily dose.

Geriatric patients, children, and adolescents are more sensitive to the side effects and toxicities of tricyclic antidepressants than other people. For geriatric patients, the dose may range from 25 to 100 mg per day. For children six to 12 years old, the recommended dose ranges from 10 to 30 mg per day in divided doses. For adolescents, daily dosages range from 25 to 50 mg but may be increased up to 100 mg, if needed.

Precautions

Desipramine and other tricyclic antidepressants may cause drowsiness. Activities requiring alertness, such as driving, may be impaired. Dizziness or lightheadedness may occur on arising due to sudden decreases in blood pressure. Fainting may occur. Some patients may experience difficulty urinating, especially men with prostate enlargement. Glaucoma may be aggravated. Sensitivity to ultraviolet light may be increased, and sunburns may occur more easily. Sweating may be reduced, causing sensitivity to heat and hot weather. Among patients with epilepsy, **seizures** may become more frequent.

Tricyclic antidepressants, including desipramine, should be used with caution in patients with heart disease because of the possibility of adverse effects on heart rhythm. Adverse effects on the heart occur frequently when tricyclics are taken in overdose. Only small quantities of these drugs should be given to patients who may be suicidal.

Tricyclic antidepressants may cause dry mouth due to decreased saliva, possibly contributing to the development of tooth decay, gum disease, and mouth infections. Patients should avoid sweets, sugary beverages, and chewing gum containing sugar.

It has not been determined whether desipramine is safe to take during pregnancy, and the patient's need for this medicine should be balanced against the possibility of harm to the fetus. Tricyclic antidepressants may be secreted in breast milk and may cause sedation and depressed breathing in a nursing infant.

Side effects

Desipramine may cause many side effects. Initially, the side effects of tricyclic drugs may be more pronounced, but sensitivity may decrease with continued treatment. The following more common side effects are grouped by the body system affected:

- Cardiovascular: decreases of blood pressure on rising from a sitting or lying position, which may cause dizziness or fainting; increases of blood pressure, rapid heart rate, pounding heart, altered heart rhythm.
- Nervous system: sedation, confusion, nervousness, restlessness, sleep difficulties, numbness, tingling sensations, tremors, increased seizure tendency.
- Autonomic: blurred vision, dry mouth, decreased sweating, difficulty urinating, constipation.
- Skin: rashes, sensitivity to sunlight.
- Body as a whole: weight gain.

Less commonly, tricyclic drugs may cause adverse effects on almost any organ or system of the body, particularly the blood, hormones, kidney, and liver. Patients should consult their physicians if symptoms develop or bodily changes occur.

Interactions

Tricyclic antidepressants, such as desipramine, may interact with many other drugs. Patients should inform their physicians about all other drugs they are taking. Tricyclic drugs may intensify the effects of drugs causing sedation, including alcohol, **barbiturates**, narcotic pain medications, minor tranquilizers, and antihistamines. Tricyclics may cause excessive reductions of blood pressure in patients taking blood pressure medicine, especially on arising or standing up. Conversely, these drugs may interfere with the pressure-reducing effects of certain other blood pressure medicines. Tricyclics may interact with thyroid medications to produce heart rhythm abnormalities. Also, they may increase seizure tendency in patients with epilepsy, requiring adjustment of anti-epileptic medication. Concurrent use of tricyclic antidepressants with other antidepressants or other psychiatric medicines may result in intensification of certain side effects.

Certain drugs may interfere with the elimination of tricyclic antidepressants from the body causing higher blood levels and increased side effects. This effect may occur with cimetidine (Tagamet), other antidepressants, **methylphenidate** (Ritalin, Concerta), and some antipsychotic medications.

See also Addiction; Cocaine and related disorders; Depression and depressive disorders; Panic attack; Psychosis

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Richard Kapit, M.D.

Desoxyn *see* **Amphetamines**

Desyrel *see* **Trazodone**

Detoxification

Definition

Detoxification is a process in which the body is allowed to free itself of a drug. During this period, the symptoms of withdrawal are also treated. Detoxification is the primary step in any drug treatment program, and is used as the initial phase in treating alcohol, heroin, inhalant, sedative, and hypnotic addictions.

Purpose

The goal of detoxification is to clear the toxins out of the body so that the body can adjust and heal itself after being dependent on a substance. In order for the recovering person to stay abstinent on a long-term basis, detoxification needs to lead into long-term community residential program treatment or outpatient drug treatment lasting three to six months.

Precautions

When individuals are physically dependent on a substance, they experience withdrawal symptoms when they abstain from the drug. Withdrawal symptoms vary with each drug of abuse, but can be severe, and even dangerous. Patients who want to overcome their dependence need help managing the withdrawal symptoms. The patient's medical team strives to get the patient off a substance on which he or she is physically dependent, while treating the withdrawal symptoms.

KEY TERMS

Agonist—A chemical that reproduces the mechanism of action of a neurotransmitter.

Antagonist—A substance whose actions counteract the effects of or work in the opposite way from another chemical or drug.

Buprenorphine—A medication that blocks some of the withdrawal effects during heroin detoxification.

Detoxification—A process in which the body is allowed to free itself of a drug while the symptoms of withdrawal are treated. It is the primary step in any treatment program for drug or alcohol abuse.

Disulfiram—A medication that helps reinforce abstinence in people who are recovering from alcohol abuse. If a person taking disulfiram drinks even a small amount of alcohol, he or she experiences facial flushing, headache, nausea and vomiting.

Lofexidine—A medication approved for use in England to aid the opioid detoxification process.

Methadone—A drug often prescribed legally as a replacement for heroin. It induces a slight high but blocks heroin from producing a more powerful euphoric effect. It may be used in heroin detoxification to ease the process, or it may be used daily after detoxification as maintenance therapy. Methadone maintenance therapy is controversial.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

Pregnant women cannot be detoxified from opiates (also called narcotics, including morphine, heroin, and similar drugs) because strict detoxification can increase the risk of spontaneous abortion or premature birth. These women are treated with **methadone** as an alternative. (Methadone acts as a replacement for the heroin in the woman's body, but the methadone does not provide the "high" that the heroin provides. In addition, methadone is safer for the fetus than heroin.)

In order to be an effective first step of treatment, detoxification must be an individualized process because patients have varying needs.

Description

The body, when allowed to be free from drugs, detoxifies itself through its normal metabolic processes. The withdrawal symptoms are treated during this process so that the patient will be comfortable while the body detoxifies itself.

The process of substance addiction

Before discussing detoxification, it may be useful to understand how the body becomes addicted and why withdrawal symptoms are experienced. In physical **addiction** or dependence, as a person uses a substance or chemical over a long period of time, his or her body chemistry changes. Once a substance enters the body through drinking, smoking, injecting or inhaling, it travels through the bloodstream to the **brain**. The brain has a complex reward system built in— when people engage in activities that are important for survival (such as eating), special nerve cells in the brain release chemicals (**neurotransmitters**, including dopamine) that induce feelings of pleasure. Because of this reward system in the brain, humans are programmed to want to repeat actions that elicit those pleasant sensations. In other words, feelings of pleasure reinforce certain activities or behaviors. Addictive substances interfere with this reward system. Some drugs mimic the effects of a natural chemical, some block the communication between nerve cells, and some substances trigger a larger-than-normal release of neurotransmitters like dopamine. The result of this interference is that dependent drug users physically need the drug to feel pleasure. As they become more dependent, their bodies becomes less responsive to the substance, and need more of it to get the desired response—a phenomenon called tolerance. Also as a result of the interference with the brain's system, when the dependent user does not have the drug in his or her system, feelings of depression or unpleasant withdrawal symptoms may be experienced. These consequences also reinforce the substance use— people dependent on substances resort to using more drugs to avoid the depression or the withdrawal symptoms.

Withdrawal symptoms

The symptoms and severity of these symptoms vary from one substance to another.

ALCOHOL. After a person who has used alcohol heavily for a long time stops drinking, he or she may experience increased heart rate, shaking, difficulty sleeping, nausea, restlessness, anxiety, and even **seizures**. The affected person may also experience **hallucinations** (seeing, hearing, or feeling something that isn't really present). In some cases, delirium tremens (DTs) may occur as part of the withdrawal. Delirium tremens is a violent **delirium** (fading in and out of consciousness) with tremors, increased motor activity, visual hallucina-

tions, disorientation, confusion, and fever that happens 48-96 hours after the alcohol-dependent person has had his or her last drink. These symptoms can last anywhere from three to 10 days. This state is a medical emergency because it could be fatal.

HEROIN AND OTHER OPIATES. Heroin is part of a family of drugs called opiates or opioids, which are made up of drugs that come from the seeds of the Asian poppy (heroin, opium and morphine, for example) and also manufactured drugs that act like the natural drugs (meperidine or Demerol). Symptoms of opiate withdrawal include restlessness, **insomnia**, anxiety, irritability, loss of appetite, diarrhea, abdominal cramps, nausea, sweating, chills, and runny eyes and nose.

SEDATIVES AND HYPNOTICS. Sedatives and hypnotics are drug families that are often considered in one group called the sedative-hypnotics. These drugs depress or slow down the body's functions, and can be used to calm anxiety or to induce sleep. When taken in high doses or when abused, these drugs can cause unconsciousness or death. These drugs include **barbiturates** and benzodiazepines. Some barbiturates are amobarbital (Amytal), pentobarbital (Nembutal), and secobarbital (Seconal). Some benzodiazepines include **diazepam** (Valium), **chlordiazepoxide** (Librium), and **lorazepam** (Ativan). When a person dependent on these drugs stops taking them suddenly, he or she might experience restlessness, muscle cramps, anxiety, insomnia, irritability, paranoid behavior, and even seizures or death.

Alcohol detoxification

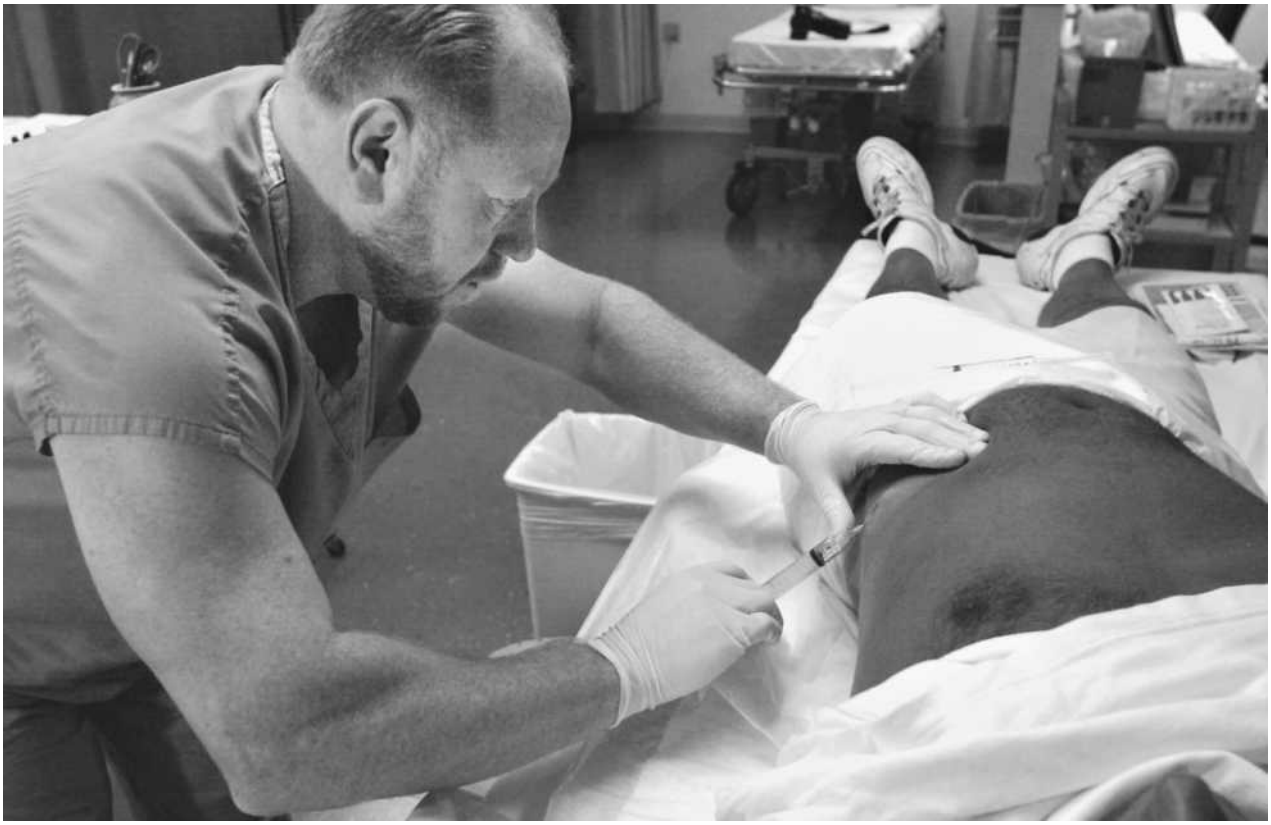
Patients being detoxified from alcohol can safely be treated with rest, nutrition, vitamins, and thiamin (a B vitamin whose absorption is affected by alcohol abuse). Detoxification can be completed in an inpatient setting, or patients may participate in intensive outpatient (day hospital) treatment. People with mild or moderate withdrawal symptoms undergo detoxification over a five-day period and receive a benzodiazepine or phenobarbital to help ease the withdrawal symptoms. Delirium tremens can be treated with very high-dose benzodiazepines (such as chlordiazepoxide or diazepam) or with antipsychotic medications such as Haldol (**haloperidol**). The patient usually receives medication at doses high enough to give 60 mg or more of the medication over a 24- to 36-hour period, and the doses of these medications are gradually decreased by 20% each day. Patients who have liver disease, **dementia**, or patients who are over the age of 65 or with significant medical problems may receive lorazepam for the withdrawal symptoms.

Heroin detoxification

Patients with heroin dependence may receive help with their detoxification in one of two forms. Opioid agonists are drugs that act like heroin in the patient's body but do not provide the same "high," and are given in gradually decreasing doses. Because these medications "act" like heroin, the person does not experience withdrawal symptoms. Some examples of this kind of medication are methadone and levo-alpha-acetylmethadol (LAAM); buprenorphine is a partial opioid agonist, which means that it acts like heroin or methadone, but it limits the effects of opioids so that higher doses produce no greater effects. The second form of help for patients undergoing heroin detoxification is the use of a drug, such as **clonidine** (Catapres), that blocks some of the withdrawal symptoms. There is also a new method of heroin detoxification called ultra-rapid opioid detoxification under anesthesia/sedation, and there is an experimental method using a medication called lofexidine.

METHADONE SUBSTITUTION. Methadone substitution can occur in outpatient or inpatient settings, and is a method of detoxification that involves helping patients off substances such as heroin by substituting these substances with methadone to ease the withdrawal symptoms, and gradually decreasing the dose until no methadone is needed for the symptoms. Patients may begin with a dose of methadone that is between 20 mg and 40 mg per day. The initial dose may be adjusted so that the most beneficial dose can be discovered, based on the patient's withdrawal symptoms. The dose is then gradually decreased over the next several days. The decrease in methadone dosage is called tapering. If the detoxification is being completed in an inpatient setting, the methadone dose can be tapered more quickly, because medical staff can closely monitor patients for withdrawal, and detoxification can be achieved in about five to 10 days. However, in the case of outpatient detoxification, the taper has to be done much more slowly to assure that the patient does not have an adverse reaction or relapse (use the drug of abuse again) to treat their withdrawal symptoms. The dose may be decreased about 10% per week initially until a dose of 20 mg is reached. Then the dose can be decreased by 3% per week for the rest of the time that the patient needs to be detoxified. Patients are usually comfortable with the slow decrease of the medication until the dose gets below 20 mg/day. At that point, patients tend to become fearful of being off opioids and having symptoms of withdrawal.

Clonidine is used much more frequently than methadone in detoxification. Methadone is used frequently as long-term maintenance treatment for heroin addiction.



A doctor implants a naltrexone pellet into a patient's stomach. The patient had undergone rapid opiate detoxification months before this procedure. The naltrexone blocks the euphoria-producing effects of heroin, discouraging recovering addicts to relapse and abuse the drug from which they are recovering. *(AP Photo/ Charles Rex Arbogast. Photo reproduced with permission.)*

BUPRENORPHINE. Buprenorphine is another medication that is used during opioid detoxification. Because it also acts like heroin in the body, the patient does not experience the withdrawal symptoms as the heroin is being eliminated from the body. It is given as an intramuscular injection or intravenously. It begins to work within 15 minutes and its effects last six hours. A patient receiving buprenorphine receives this medication for at least three days, and then the medication is either gradually withdrawn or discontinued abruptly.

CLONIDINE. Clonidine is a medication that decreases many of the symptoms of opioid withdrawal. Patients may require nonsteroidal anti-inflammatory drugs (NSAIDS, such as ibuprofen) for the treatment of muscle aches. Clonidine's major side effects include sedation and hypotension (low blood pressure). Patients undergoing detoxification using clonidine will have their blood pressure and pulse checked regularly. The starting dose of clonidine is 0.1–0.3 mg every four to six hours— the maximum amount that can be given in one day is 1 mg. During days two through four of the detoxification, the dose of clonidine is adjusted to control the withdrawal symptoms. Again, however, the dose cannot exceed the maximum dose. On the fifth day of detoxification, the dose starts to get slowly tapered.

The clonidine patch is a transdermal patch, meaning that the drug is delivered through the skin and causes the patient to be exposed to a constant amount of the drug over a seven-day period. It also allows the person to experience a more comfortable heroin detoxification. It comes in three doses: 0.1-mg, 0.2-mg, and 0.3-mg. Patients who will use the clonidine patch need to have both the patch on and take oral clonidine during the first two days of the detoxification, because it takes the patch two days to reach a steady state and be effective. The patient takes 0.2 mg of oral clonidine three times a day, and the weight of the patient determines the dose of the patch. On day two, the amount of clonidine that the patient takes by mouth is reduced by half and then it is completely stopped after day three. After seven days, the patch is removed and replaced with a patch that is half the amount of the original dose. The patch is continued for as long as the patient continues to have symptoms of withdrawal. Blood pressure is monitored for the patient

using the patch, as well. The detoxification process in general takes about seven days using clonidine.

CLONIDINE-NALTREXONE ULTRA-RAPID DETOXIFICATION. Clonidine-naltrexone ultra-rapid detoxification is a faster means of detoxification than using clonidine alone. The higher the dose of **naltrexone** that the person receives, the faster he or she can be detoxified. Very close monitoring for withdrawal symptoms is necessary, however, particularly during the first eight hours of the detoxification process. (Naltrexone accelerates the withdrawal.) On the first day of the detoxification process, the patient is premedicated with **oxazepam** and clonidine. A couple hours later, the patient receives naltrexone. Throughout the rest of the first day, the patient receives oxazepam and clonidine every four to six hours. On the second and third days, the patient receives a larger dose of naltrexone, and continues to receive oxazepam and clonidine throughout the day. After day three, the naltrexone is no longer given, and the patient continues to take the clonidine and oxazepam for two to three more days. Additional medications to help with muscle cramping and nausea may be necessary. In an inpatient setting, the naltrexone can be increased so that the patient can complete detoxification in two to three days.

LOFEXIDINE. Lofexidine is approved for use in England for opioid detoxification. It appears to cause less sedation and fewer cases of low blood pressure than clonidine. In the United States, the National Institute of Drug Abuse (NIDA) is conducting studies on this drug.

ULTRA-RAPID OPIOID DETOXIFICATION UNDER ANESTHESIA/SEDATION. In this new procedure, anesthesia is induced and the patient receives a tracheal tube— a tube in the throat— and a tube in the nose (a nasogastric tube). The patient is given intravenous naloxone or naltrexone through the nasogastric tube. The procedure takes only eight hours and the patient can leave the hospital in one or two days. The patient's withdrawal symptoms are treated with a variety of medications including clonidine, antidiarrheal medications, and benzodiazepines.

Mixed substance abuse

Mixed substance abuse (also called polysubstance abuse) occurs when individuals abuse more than one substance. Many doctors prefer to use phenobarbital to detoxify patients with polysubstance abuse problems. Patients receiving phenobarbital may receive a test dose, and then based on his or her tolerance and symptoms, the dose will be adjusted. Patients cannot receive more than 600 mg of phenobarbital a day. After two to three days, once the patient is doing well, the dose can be reduced by 30-60 mg. Whether detoxification for polysubstance abusers will be completed on an inpatient or outpatient status depends on the drugs the patient abuses.

Benzodiazepines

These medications are often used to help patients during detoxification, but these substances themselves can be abused and addictive. Patients who have taken a prescribed benzodiazepine for two weeks, even in a therapeutic dose, need to be safely detoxified with a slow taper. The amount of drug the person takes is dropped by 10-25% every week if the patient has minimal withdrawal symptoms. If the patient has taken very high doses for long periods of time, he or she is at increased risk for addiction. If the person has been taking a benzodiazepine medication for years, it can take months before he or she can get off the drug. Anticonvulsant medications like **carbamazepine** (Tegretol) and **divalproex sodium** (Depakote) can be used to make the detoxification process faster and more comfortable for the patient.

Preparation

The first step in any detoxification, regardless of the substance, is a physical exam and history taken by a physician. This information-gathering and examination will help the treatment team assess the patient's overall health. In general, the healthier the patient is, the better the chances are that the patient will experience a detoxification without serious or life-threatening complications. Patients also need to give urine and blood samples to test for drugs and alcohol.

Aftercare

After the patient has completed detoxification, he or she needs further treatment either at an outpatient, inpatient, residential, or day hospital program in order to remain drug-free for the long term. Patients are treated by trained health care professionals, and some patients are also counseled by people who are recovering from addiction themselves. Many patients also benefit from 12-step programs or **self-help groups**, such as Alcoholics Anonymous (AA) or Narcotics Anonymous (NA).

Most opioid users are treated with ambulatory or outpatient detoxification or residential treatment followed by outpatient counseling. Some people who have abused opioids and have undergone detoxification and counseling are able to remain drug-free. Many, however, relapse, even after receiving **psychotherapy**. People recovering from opioid addiction can receive methadone or LAAM as maintenance therapy to prevent relapse. Similar to the aid these medications can give patients during detoxification, when taken daily as a therapy, they

continue to "act" as heroin, keeping the withdrawal symptoms from appearing. Methadone maintenance therapy can be provided through either residential or therapeutic communities and outpatient drug-free programs. Methadone maintenance treatment therapy is controversial, however, because it does not cure the person's addiction— it replaces it with another substance. Proponents of methadone maintenance therapy argue that people receiving methadone are able to function much better in society than people addicted to heroin. Because their drug-seeking behavior is reduced, these patients are able to become productive at work and their interpersonal relations improve.

People recovering from alcoholism can also benefit from counseling and support after detoxification, and a maintenance therapy is available to them, as well. **Disulfiram** (Antabuse) is a medication that interferes with the body's breakdown and processing of alcohol. When alcohol is consumed while a patient is taking disulfiram, the medication makes the effects of the alcohol much worse than the patient would normally experience—facial flushing, headache, nausea and vomiting occurs, even if alcohol is consumed in a small amount. In order for disulfiram to be effective, the patient must want this kind of **reinforcement** to maintain abstinence and must be committed to it. Patients also must note that any form of alcohol can trigger the undesired effects, including cooking wine or mouthwash with alcohol.

Risks

When benzodiazepines are the drug to which a person is addicted, they have to be discontinued and cannot be given on an outpatient basis because of their potential for abuse. For all patients undergoing detoxification, benzodiazepine use must be monitored carefully because of the potential for new addiction. Elderly patients undergoing detoxification and receiving benzodiazepines must be monitored closely because they are more sensitive to the sedating effects of these drugs, and are also more prone to falls while receiving these drugs. If benzodiazepines are not discontinued gradually, patients can have withdrawal symptoms such as irritability, poor sleep, agitation and seizures. Ultra-rapid opioid detoxification under anesthesia/sedation remains a new and serious procedure. Patients have died receiving this procedure, and this procedure is still being researched.

It should also be noted that many of the substances used in detoxification can themselves cause addictions. An example of this risk has already been given with benzodiazepines—these medications ease withdrawal symptoms during detoxification, but patients can get addicted to these medications, as well.

Normal results

Normal results for a well-managed detoxification would include freedom from the drug of addiction and ability to enter long-term treatment.

Success rates vary among people recovering from substance abuse. As might be expected, patients who successfully complete a full treatment program after detoxification (that includes counseling, psychotherapy, **family therapy**, and/or **group therapy** or some combination of those therapy types) achieve higher rates of success at remaining drug-free. Patients who were addicted for shorter periods of time and patients who spend longer periods in treatment are generally more successful at remaining abstinent from drugs over the long term.

Studies indicate that people who abuse alcohol and who want to stop have a higher chance of success if they undergo inpatient detoxification versus outpatient detoxification.

Abnormal results

One abnormal result that may occur is that patients who received nasogastric or tracheal tubes for opioid detoxification under anesthesia may experience adverse effects or complications. These patients are at risk for: trauma to their lips, vocal cords, larynx, teeth; nose bleeds; high blood pressure; elevated heart rate; irregular heartbeat; and vomiting, which can lead to aspiration pneumonia.

An additional abnormal result would be a new addiction as a consequence of the detoxification.

After the detoxification is completed, patients may relapse. Support is critical for patients to continue long-term therapy and successfully overcome addiction.

See also Addiction; Disease concept of chemical dependency; Individual entries on various substances and related disorders

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Institute for Comprehensive Detoxification and Rehabilitation. (877) 704-ICDR (4237). <http://www.views.vcu.edu/cpdd/>.

National Institute on Alcohol Abuse and Alcoholism (NIAAA). 6000 Executive Boulevard, Willco Building, Bethesda, MD, 20892-7003.<http://www.niaaa.nih.gov>.

National Institute on Drug Abuse (NIDA). 6001 Executive Boulevard, Room 5213, Bethesda, MD, 20892-9561. (301) 443-1124. <http://www.nida.nih.gov>.

Susan Hobbs, M.D.

Developmental coordination disorder

Definition

Developmental coordination disorder is diagnosed when children do not develop normal motor coordination (coordination of movements involving the voluntary muscles).

Description

Developmental coordination disorder has been known by many other names, some of which are still used today. It has been called clumsy child syndrome, clumsiness, developmental disorder of motor function, and congenital maladroitness. Developmental coordination disorder is usually first recognized when a child fails to reach such normal developmental milestones as walking or beginning to dress him- or herself.

KEY TERMS

Maladroitness—Another word for awkwardness or clumsiness.

Motor skills—Skills pertaining to or involving muscular movement.

Children with developmental coordination disorder often have difficulty performing tasks that involve both large and small muscles, including forming letters when they write, throwing or catching balls, and buttoning buttons. Children who have developmental coordination disorder have often developed normally in all other ways. The disorder can, however, lead to social or academic problems for children. Because of their underdeveloped coordination, they may choose not to participate in activities on the playground. This avoidance can lead to conflicts with or rejection by their peers. Also, children who have problems forming letters when they write by hand, or drawing pictures, may become discouraged and give up academic or artistic pursuits even though they have normal intelligence.

Causes and symptoms

The symptoms of developmental coordination disorder vary greatly from child to child. The general characteristic is that the child has abnormal development of one or more types of motor skills when the child's age and intelligence quotient (IQ) are taken into account. In some children these coordination deficiencies manifest as an inability to tie shoes or catch a ball, while in other children they appear as an inability to draw objects or properly form printed letters.

Some investigators believe that there are different subtypes of developmental coordination disorder. While there is disagreement over how to define these different subtypes, they can provide a useful framework for the categorization of symptoms. There are six general groups of symptoms. These include:

- general unsteadiness and slight shaking
- an at-rest muscle tone that is below normal
- muscle tone that is consistently above normal
- inability to move smoothly because of problems putting together the subunits of the whole movement
- inability to produce written symbols

Children with developmental coordination disorder have difficulty performing tasks that require motor skills or eye-hand coordination, such as catching a ball. *(Anthony Nex. CORBIS. Photo reproduced by permission.)*

- visual perception problems related to development of the eye muscles

Children can have one or more of these types of motor difficulties.

Developmental coordination disorder usually becomes apparent when children fail to meet normal developmental milestones. Some children with developmental coordination disorder do not learn large motor skills such as walking, running, and climbing until a much later point in time than their peers. Others have problems with such small muscle skills as learning to fasten buttons, close or open zippers, or tie shoes. Some children have problems learning how to handle silverware properly. In others, the disorder does not appear until they are expected to learn how to write in school. Some children just look clumsy and often walk into objects or drop things.

There are no known causes of developmental coordination disorder. There are, however, various theories about its possible causes. Some theories attribute the disorder to biological causes. Some of the possible biological causes include such prenatal complications as fetal malnutrition. Low birth weight or prematurity are thought to be possible causes, but there is no hard evidence supporting these claims.

Demographics

It is estimated that as many as 6% of children between the ages of five and 11 have developmental coordination disorder. Males and females are thought to be equally likely to have this disorder, although males may be more likely to be diagnosed. Developmental coordination disorder and speech-language disorders seem to be closely linked, although it is not clear why this is the case. Children with one disorder are more likely to have the other also.

Diagnosis

The **diagnosis** of developmental coordination disorder is most commonly made when a child's parents or teachers notice that he or she is lagging behind peers in learning motor skills, is having learning problems in school, or is suffering frequent injuries from falls and other accidents resulting from clumsiness. In most cases, the child's pediatrician will perform a physical examination in order to rule out problems with eyesight or hearing that interfere with muscular coordination, and to rule out disorders of the nervous system. In addition to a medical examination, a learning specialist or child **psychiatrist** may be consulted to rule out other types of learning disabilities.

The types of motor impairment that lead to a diagnosis of developmental coordination disorder are somewhat vague, as the disorder has different symptoms in different children. There are many ways in which this kind of motor coordination problem can manifest itself, all of which may serve as criteria for a diagnosis of developmental coordination disorder. The core of the diagnosis rests on the child's being abnormally clumsy. To make this determination, the child's motor coordination must be compared to that of other children of a similar age and intelligence level.

The difference between a child who has developmental coordination disorder and one who is simply clumsy and awkward can be hard to determine. For a child to be diagnosed with developmental coordination disorder there must be significant negative consequences for the child's clumsiness. The negative effects may be seen in the child's performance in school, activities at play, or other activities that are necessary on a day-to-day basis. Also, for developmental coordination disorder to be diagnosed, the child's problems with motor coordination cannot result from such general medical conditions as muscular dystrophy, and cannot result directly from

mental retardation. Some criteria require that the child have an IQ of at least 70 to be diagnosed with developmental coordination disorder.

Treatments

No treatments are known to work for all cases of developmental coordination disorder. Experts recommend that a specialized course of treatment, possibly involving work with an occupational therapist, be drawn up to address the needs of each child. Many children can be effectively helped in special education settings to work more intensively on such academic problems as letter formation. For other children, physical education classes designed to improve general motor coordination, with emphasis on skills the child can use in playing with peers, can be very successful. Any kind of physical training that allows the child to safely practice motor skills and motor control may be helpful.

It is important for children who have developmental coordination disorder to receive individualized therapy, because for many children the secondary problems that result from extreme clumsiness can be very distressing. Children who have developmental coordination disorder often have problems playing with their peers because of an inability to perform the physical movements involved in many games and sports. Unpopularity with peers or exclusion from their activities can lead to low self-esteem and poor self-image. Children may go to great lengths to avoid physical education classes and similar situations in which their motor coordination deficiencies might be noticeable. Treatments that focus on skills that are useful on the playground or in the gymnasium can help to alleviate or prevent these problems.

Children with developmental coordination disorder also frequently have problems writing letters and doing sums, or performing other motor activities required in the classroom— including coloring pictures, tracing designs, or making figures from modeling clay. These children may become frustrated by their inability to master tasks that their classmates find easy, and therefore may stop trying or become disruptive. Individualized programs designed to help children master writing or skills related to arts and crafts may help them regain confidence and interest in classroom activities.

Prognosis

For many people, developmental coordination disorder lasts into adulthood. Through specialized attention and teaching techniques it is possible over time for many children to develop the motor skills that they lack. Some children, however, never fully develop the skills they need. Although many children improve their motor skills significantly, in most cases their motor skills will never match those of their peers at any given age.

Prevention

There is no known way to prevent developmental coordination disorder, although a healthy diet throughout pregnancy and regular prenatal care may help, as they help to prevent many childhood problems.

See also Disorder of written expression

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American Academy of Pediatrics. 141 Northwest Point Boulevard, Elk Grove Village, IL 60007-1098. (847) 434-4000. <www.aap.org>.

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Developmental disorders *see* **Pervasive developmental disorders**

Developmental reading disorder *see* **Reading disorder**

Deviance *see* **Paraphilias**

Dexedrine *see* **Amphetamines**

Dextroamphetamine *see* **Amphetamines**

Diagnosis

Definition

Diagnosis can be defined as the identification and labeling of a disease based on its signs and symptoms. Mental health clinicians (psychiatrists, psychologists, and psychiatric nurse practitioners) diagnose mental disorders using the criteria listed in the ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM,* published by the American Psychiatric Association.

Description

According to the *DSM,* fourth edition, text revised (the *DSM-IV-TR*), the term *mental disorder* is unfortunate because it implies that a mental disorder is separate from a physical illness, when actually, according to the American Psychiatric Association (APA), researchers and scientists now know that that distinction is not a clear one to make. The APA argues that "there is much 'physical' in 'mental disorders' and much 'mental' in 'physical disorders,'" and continues to use the term "mental disorders" because a better term has not yet been found. The APA defines a mental disorder as "a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual and that is associated with present distress or disability or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom." Many people fear that when a mental disorder is classified, *people* are actually classified, and the *DSM-IV-TR* strives to contradict this notion. The American Psychiatric Association believes their manual to be strictly a manual classifying mental disorders themselves, and does not advocate the use of the diagnoses to discriminate.

The manual lists various criteria for each mental disorder included in the book. When an individual seeks the help of a mental health clinician, the clinician interviews the client (along with family members when appropriate), gathers a medical history, and may administer psychological evaluations (various checklists or tests that the patient may complete) in order to establish a diagnosis. Once the clinician has gathered the necessary information, a diagnosis based on the symptoms may be assigned from the *DSM.*

One of the main purposes of diagnosis is to guide treatment planning. If doctors know that a particular disorder has shown to be treated effectively with a drug or with a specific therapy, then the best practice can be applied to a new case of that disorder. The diagnosis also helps to establish a prognosis for the patient and his or her family, and it helps to enable communication among the professionals (including insurers) involved in a patient's care. Additionally, a formal diagnosis as recognized by the *DSM* may be necessary in order for insurers to pay for medical services. The act of labeling a mental disorder may have unintended effects for the person with the disorder, however. Although the *DSM* states that its diagnoses do not label people, in reality, many people who have received diagnoses of mental disorders may feel affected by the label their disorder has been given. People diagnosed with mental disorders may feel stigmatized, and that others' perceptions of them—as well as their self-perceptions— have changed as a result of their diagnosis.

See also Assessment and diagnosis; Stigma

Resources

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Diagnostic and Statistical Manual of Mental Disorders

Nature and purposes

The *Diagnostic and Statistical Manual of Mental Disorders* is a reference work consulted by psychiatrists, psychologists, physicians in clinical practice, **social workers**, medical and nursing students, pastoral counselors, and other professionals in health care and social service fields. The book's title is often shortened to *DSM*, or an abbreviation that also indicates edition, such as *DSM-IV-TR,* which indicates fourth edition, text revision of the manual, published in 2000. The *DSM-IV-TR* provides a classification of mental disorders, criteria sets to guide the process of differential **diagnosis**, and numerical codes for each disorder to facilitate medical record-keeping. The stated purpose of the *DSM* is threefold: to provide "a helpful guide to clinical practice"; "to facilitate research and improve communication among clinicians and researchers"; and to serve as "an educational tool for teaching psychopathology."

The multi-axial system

The third edition of *DSM*, or *DSM-III*, which was published in 1980, introduced a system of five axes or dimensions for assessing all aspects of a patient's mental and emotional health. The multi-axial system is designed to provide a more comprehensive picture of complex or concurrent mental disorders. According to the *DSM-IV-TR,* the system is also intended to "promote the application of the biopsychosocial model in clinical, educational and research settings." The reference to the *biopsychosocial model* is significant, because it indicates that the *DSM-IV-TR* does not reflect the view of any specific "school" or tradition within psychiatry regarding the cause or origin (also known as "etiology") of mental disorders. In other words, the *DSM-IV-TR* is atheoretical in its approach to diagnosis and classification— the axes and categories do not represent any overarching theory about the sources or fundamental nature of mental disorders.

The biopsychosocial approach was originally proposed by a **psychiatrist** named George Engel in 1977 as a way around the disputes between psychoanalytically and biologically oriented psychiatrists that were splitting the field in the 1970s. The introduction to *DSM-IV-TR* is quite explicit about the manual's intention to be "applicable in a wide variety of contexts" and "used by clinicians and researchers of many different orientations (e.g., biological, psychodynamic, cognitive, behavioral, interpersonal, family/systems)."

The atheoretical stance of *DSM-IV-TR* is also significant in that it underlies the manual's approach to the legal implications of mental illness. *DSM* notes the existence of an "imperfect fit between questions of ultimate concern to the law and the information contained in a clinical diagnosis." What is meant here is that the *DSM-IV-TR* diagnostic categories do not meet forensic standards for defining a "mental defect," "mental disability," or similar terms. Because *DSM-IV-TR* states that "inclusion of a disorder in the classification ... does not require that there be knowledge about its etiology," it advises legal professionals against basing decisions about a person's criminal responsibility, competence, or degree of behavioral control on *DSM* diagnostic categories.

The five diagnostic axes specified by *DSM-IV-TR* are:

- Axis I: Clinical disorders, including anxiety disorders, mood disorders, **schizophrenia** and other psychotic disorders.
- Axis II: **Personality disorders** and **mental retardation**. This axis includes notations about problematic aspects of the patient's personality that fall short of the criteria for a personality disorder.
- Axis III: General medical conditions. These include diseases or disorders that may be related physiologically to the mental disorder; that are sufficiently severe to affect the patient's mood or functioning; or that influence the choice of medications for treating the mental disorder.
- Axis IV: Psychosocial and environmental problems. These include conditions or situations that influence the diagnosis, treatment, or prognosis of the patient's mental disorder. *DSM-IV-TR* lists the following categories of problems: family problems; social environment problems; educational problems; occupational problems; housing problems; economic problems; problems with access to health care; problems with the legal system; and other problems (war, disasters, etc.).

KEY TERMS

Atheoretical—Unrelated to any specific theoretical approach or conceptual framework. The classification system of *DSM-IV-TR* is atheoretical.

Differential diagnosis—The process of distinguishing one disorder from other, similar disorders.

Empirical—Verified by actual experience or by scientific experimentation.

Etiology—The cause or origin of a disease or disorder. The word is also used to refer to the study of the causes of disease.

Forensic—Pertaining to courtroom procedure or evidence used in courts of law.

Holistic—An approach to health care that emphasizes the totality of an individual's well-being, spiritual and psychological as well as physical; and that situates a disease or disorder within that totality.

Implicit—Implied or suggested without being clearly stated. Some critics of *DSM-IV-TR* maintain that its contributors based the criteria sets for certain disorders on an implicit notion of a mentally healthy human being.

Medical model—The basic conceptual framework in the West since the nineteenth century for understanding, researching, and classifying mental disorders.

Nosology—The branch of medicine that deals with the systematic classification of diseases and disorders.

- Axis V: Global assessment of functioning. Rating the patient's general level of functioning is intended to help the doctor draw up a treatment plan and evaluate treatment progress. The primary scale for Axis V is the Global Assessment of Functioning (GAF) Scale, which measures level of functioning on a scale of 1–100. *DSM-IV-TR* includes three specialized global scales in its appendices: the Social and Occupational Functioning Assessment Scale (SOFAS); the Defensive Functioning Scale; and the Global Assessment of Relational Functioning (GARF) Scale. The GARF is a measurement of the maturity and stability of the relationships within a family or between a couple.

Diagnostic categories

The Axis I clinical disorders are divided among 15 categories: disorders usually first diagnosed in infancy, childhood, or adolescence; **delirium**, **dementia**, amnestic, and other cognitive disorders; medical disorders due to a general medical condition; substance-related disorders; schizophrenia and other psychotic disorders; mood disorders; anxiety disorders; somatoform disorders; factitious disorders; dissociative disorders; sexual and gender identity disorders; eating disorders; **sleep disorders**; impulse control disorders not elsewhere classified; and adjustment disorders.

The diagnostic categories of *DSM-IV-TR* are essentially symptom-based, or, as the manual puts it, based "on criteria sets with defining features." Another term that is sometimes used to describe this method of classification is *phenomenological*. A phenomenological approach to classification is one that emphasizes externally observable phenomena rather than their underlying nature or origin.

Another important characteristic of *DSM-IV-TR's* classification system is its dependence on the *medical model* of mental disorders. Such terms as "psychopathology," "mental illness," "differential diagnosis," and "prognosis" are all borrowed from medical practice. One should note, however, that the medical model is not the only possible conceptual framework for understanding mental disorders. Historians of Western science have observed that the medical model for psychiatric problems was preceded by what they term the supernatural model (mental disorders understood as acts of God or the result of demon possession), which dominated the field until the late seventeenth century. The supernatural model was followed by the moral model, which was based on the values of the Enlightenment and regarded mental disorders as bad behaviors deliberately chosen by perverse or ignorant individuals.

The medical model as it came to dominate psychiatry can be traced back to the work of Emil Kraepelin, an eminent German psychiatrist whose *Handbuch der Psychiatrie* was the first basic textbook in the field and introduced the first nosology, or systematic classification, of mental disorders. By the early 1890s Kraepelin's handbook was used in medical schools across Europe. He updated and revised it periodically to accommodate new findings, including a disease that he named after one of his clinical assistants, Alois Alzheimer. The classification in the 1907 edition of Kraepelin's handbook includes 15 categories, most of which are still used nearly a century later. Kraepelin is also important in the history of diagnostic classification because he represented a biologically based view of mental disorders in opposition to the psychoanalytical approach of Sigmund Freud. Kraepelin thought that mental disorders could ultimately be traced to organic diseases of the **brain** rather than disordered emotions or psychological processes. This controversy between the two perspectives dominated psychiatric research and practice until well after the Second World War.

Background of *DSM*

The American *Diagnostic and Statistical Manual of Mental Disorders* goes back to the 1840s, when the United States Bureau of the Census attempted for the first time to count the numbers of patients confined in mental hospitals. Isaac Ray, superintendent of the Butler Hospital in Rhode Island, presented a paper at the 1849 meeting of the Association of Medical Superintendents of American Institutions for the Insane (the forerunner of the present American Psychiatric Association) in which he called for a uniform system of naming, classifying and recording cases of mental illness. The same plea was made in 1913 by Dr. James May of New York to the same organization, which by then had renamed itself the American Medico-Psychological Association. In 1933, the New York Academy of Medicine and the Medico-Psychological Association compiled the first edition of the *Statistical Manual for Mental Diseases*, which was also adopted by the American Neurological Association. The *Statistical Manual* went through several editions between 1933 and 1952, when the first edition of the *Diagnostic and Statistical Manual of Mental Disorders* appeared. The task of compiling mental hospital statistics was turned over to the newly formed National Institute of Mental Health in 1949.

DSM-I and DSM-II

DSM-I, which appeared in 1952, maintained the coding system of earlier American manuals. Many of the disorders in this edition were termed "reactions," a term borrowed from a German psychiatrist named Adolf Meyer. Meyer viewed mental disorders as reactions of an

individual's personality to a combination of psychological, social, and biological factors. *DSM-I* also incorporated the nomenclature for disorders developed by the United States Army and modified by the Veterans Administration to treat the postwar mental health problems of service personnel and veterans. The VA classification system grouped mental problems into three large categories: psychophysiological, personality, and acute disorders.

DSM-II, which was published in 1968, represented the first attempt to coordinate the American *Diagnostic and Statistical Manual of Mental Disorders* with the World Health Organization's (WHO) *International Classification of Diseases*, or *ICD*. *DSM-II* appeared before the ninth edition of the *ICD*, or *ICD-9,* which was published in 1975. *DSM-II* continued *DSM-I's* psychoanalytical approach to the etiology of the nonorganic mental disorders and personality disorders.

DSM-III, DSM-III-R and DSM-IV

DSM-III, which was published in 1980 after six years of preparatory work, represented a major break with the first two editions of *DSM. DSM-III* introduced the present descriptive symptom-based or phenomenological approach to mental disorders, added lists of explicit diagnostic criteria, removed references to the etiology of disorders, did away with the term "neurosis," and established the present multi-axial system of symptom evaluation. This sweeping change originated in an effort begun in the early 1970s by a group of psychiatrists at the medical school of Washington University in St. Louis to improve the state of research in American psychiatry. The St. Louis group began by drawing up a list of "research diagnostic criteria" for schizophrenia, a disorder that can manifest itself in a variety of ways. The group was concerned primarily with the identification of markers for schizophrenia that would allow the disease to be studied at other research sites without errors introduced by using different types of patients in different centers. What happened with *DSM-III, DSM-III-R,* and *DSM-IV,* however, was that a tool for scholarly investigation of a few mental disorders was transformed into a diagnostic method applied to all mental disorders without further distinction. The leaders of this transformation were biological psychiatrists who wanted to empty the diagnostic manual of terms and theories associated with hypothetical or explanatory concepts. The transition from an explanatory approach to mental disorders to a descriptive or phenomenological one in the period between *DSM-II* and *DSM-III* is sometimes called the "neo-Kraepelinian revolution" in the secondary literature. Another term that has been applied to the orientation represented in *DSM-III* and its successors is *empirical*, which denotes reliance on experience or experiment alone, without recourse to theories or hypotheses. The word occurs repeatedly in the description of "The DSM-IV Revision Process" in the Introduction to *DSM-IV-TR.*

DSM-IV built upon the research generated by the empirical orientation of *DSM-III.* By the early 1990s, most psychiatric diagnoses had an accumulated body of published studies or data sets. Publications up through 1992 were reviewed for *DSM-IV,* which was published in 1994. Conflicting reports or lack of evidence were handled by data reanalyses and field trials. The National Institute of Mental Health sponsored 12 *DSM-IV* field trials together with the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA). The field trials compared the diagnostic criteria sets of *DSM-III, DSM-III-R, ICD-10* (which had been published in 1992), and the proposed criteria sets for *DSM-IV.* The field trials recruited subjects from a variety of ethnic and cultural backgrounds, in keeping with a new concern for cross-cultural applicability of diagnostic standards. In addition to its inclusion of culture-specific syndromes and disorders, *DSM-IV* represented much closer cooperation and coordination with the experts from WHO who had worked on *ICD-10.* A modification of *ICD-10* for clinical practitioners, the *ICD-10-CM,* is scheduled to be introduced in the United States in 2004.

Textual revisions in DSM-IV-TR

DSM-IV-TR does not represent either a fundamental change in the basic classification structure of *DSM-IV* or the addition of new diagnostic entities. The textual revisions that were made to the 1994 edition of *DSM-IV* fall under the following categories:

- correction of factual errors in the text of *DSM-IV*
- review of currency of information in *DSM-IV*
- changes reflecting research published after 1992, which was the last year included in the literature review prior to the publication of *DSM-IV*
- improvements to enhance the educational value of *DSM-IV*
- updating of *ICD* diagnostic codes, some of which were changed in 1996

Critiques of *DSM-IV* and *DSM-IV-TR*

A number of criticisms of *DSM-IV* have arisen since its publication in 1994. They include the following observations and complaints:

- The medical model underlying the empirical orientation of *DSM-IV* reduces human beings to one-dimen-

sional sources of data; it does not encourage practitioners to treat the whole person.

- The medical model perpetuates the social **stigma** attached to mental disorders.
- The symptom-based criteria sets of *DSM-IV* have led to an endless multiplication of mental conditions and disorders. The unwieldy size of *DSM-IV* is a common complaint of doctors in clinical practice— a volume that was only 119 pages long in its second (1968) edition has swelled to 886 pages in less than thirty years.
- The symptom-based approach has also made it easier to politicize the process of defining new disorders for inclusion in *DSM* or dropping older ones. The inclusion of **post-traumatic stress disorder** (PTSD) and the deletion of homosexuality as a disorder are often cited as examples of this concern for political correctness.
- The criteria sets of *DSM-IV* incorporate implicit (implied but not expressly stated) notions of human psychological well-being that do not allow for ordinary diversity among people. Some of the diagnostic categories of *DSM-IV* come close to defining various temperamental and personality differences as mental disorders.
- The *DSM-IV* criteria do not distinguish adequately between poor adaptation to ordinary problems of living and true psychopathology. One byproduct of this inadequacy is the suspiciously high rates of prevalence reported for some mental disorders. One observer remarked that "... it is doubtful that 28% or 29% of the population would be judged [by **managed care** plans] to need mental health treatment in a year."
- The 16 major diagnostic classes defined by *DSM-IV* hinder efforts to recognize disorders that run across classes. For example, PTSD has more in common with respect to etiology and treatment with the dissociative disorders than it does with the anxiety disorders with which it is presently grouped. Another example is **body dysmorphic disorder**, which resembles the obsessive-compulsive disorders more than it does the somatoform disorders.
- The current classification is deficient in acknowledging disorders of uncontrolled anger, hostility, and aggression. Even though inappropriate expressions of anger and aggression lie at the roots of major social problems, only one *DSM-IV* disorder (**intermittent explosive disorder**) is explicitly concerned with them. In contrast, entire classes of disorders are devoted to depression and anxiety.
- The emphasis of *DSM-IV* on biological psychiatry has contributed to the widespread popular notion that most problems of human life can be solved by taking pills.

Alternative nosologies

A number of different nosologies or schemes of classification have been proposed to replace the current descriptive model of mental disorders. Three of them will be briefly described.

The dimensional model

Dimensional alternatives to *DSM-IV* would replace the categorical classification now in use with a recognition that mental disorders lie on a continuum with mildly disturbed and normal behavior, rather than being qualitatively distinct. For example, the personality disorders of Axis II are increasingly regarded as extreme variants of common personality characteristics. In the dimensional model, a patient would be identified in terms of his or her position on a specific dimension of cognitive or affective capacity rather than placed in a categorical "box."

The holistic model

The holistic approach to mental disorders places equal emphasis on social and spiritual as well as pharmacological treatments. A biochemist who was diagnosed with schizophrenia and eventually recovered compared the reductionism of the biological model of his disorder with the empowering qualities of holistic approaches. He stressed the healing potential in treating patients as whole persons rather than as isolated collections of nervous tissue with chemical imbalances: "The major task in recovering from mental illness is to regain social roles and identities. This entails focusing on the individual and building a sense of responsibility and self-determination."

The essential or perspectival model

The third and most complex alternative model is associated with the medical school of Johns Hopkins University, where it is taught as part of the medical curriculum. This model identifies four broad "essences" or perspectives that can be used to identify the distinctive characteristics of mental disorders, which are often obscured by the present categorical classifications.

The four perspectives are:

- Disease. This perspective works with categories and accounts for physical diseases or damage to the brain that produces psychiatric symptoms. It accounts for such disorders as **Alzheimer's disease** or schizophrenia.
- Dimensions. This perspective addresses disorders that arise from the combination of a cognitive or emotional weakness in the patient's constitution and a life experience that challenges their vulnerability.

- Behaviors. This perspective is concerned with disorders associated with something that the patient is doing (alcoholism, drug **addiction**, eating disorders, etc.) that has become a dysfunctional way of life.
- Life story. This perspective focuses on disorders related to what the patient has encountered in life, such as events that have injured his or her hopes and aspirations.

In the Johns Hopkins model, each perspective has its own approach to treatment: the disease perspective seeks to cure or prevent disorders rooted in biological disease processes; the dimensional perspective attempts to strengthen constitutional weaknesses; the behavioral perspective seeks to interrupt the problematic behaviors and assist patients in overcoming their appeal; and the life story perspective offers help in "rescripting" a person's life narrative, usually through cognitive behavioral treatment.

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Diazepam

Definition

Diazepam is a mild tranquilizer in the class of drugs known as benzodiazepines. It is most commonly sold in the United States under the brand name Valium. The generic form of this drug is also available.

Purpose

Diazepam is used on a short-term basis to treat patients with mild to moderate anxiety. It is also used to treat some types of **seizures** (epilepsy), muscle spasms, nervous tension, and symptoms relating to alcohol withdrawal.

Description

Diazepam is one of many chemically-related tranquilizers in the class of drugs called benzodiazepines. Benzodiazepines are sedative-hypnotic drugs that help to relieve nervousness, tension, and other anxiety symptoms by slowing the central nervous system. To do this, they block the effects of a specific chemical involved in the transmission of nerve impulses in the **brain**, decreasing the excitement level of the nerve cells. All benzodiazepines, including diazepam, cause sedation, drowsiness, and reduced mental and physical alertness.

Recommended dosage

The typical dose of diazepam used to treat anxiety or seizures in healthy adults ranges from a total of 6 milligrams (mg) to 40 mg per day given in three or four doses. Elderly people (over age 60) are usually given lower doses in the range of 4–10 mg per day to treat anx-

KEY TERMS

Albumin—A simple protein that is widely distributed in human blood.

Anxiety—A feeling of apprehension and fear characterized by physical symptoms (heart palpitations, sweating, and feelings of stress, for example).

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Tranquilizer—A medication that induces a feeling of calm and relaxation.

iety or nervous tension. For acute treatment of seizures, a higher dose of diazepam is given intravenously (directly into the vein) only in a controlled medical setting such as a hospital or emergency room. For alcohol withdrawal, the typical dose is a total of 30–40 mg per day given in three or four doses.

The typical dose for a child over age six months with anxiety or seizures is a total of 3–10 mg per divided into several doses. In general, children receive lower doses of diazepam even when they have a body weight equivalent to a small adult. Diazepam is usually taken as a pill, but an injectable form is sometimes used when a serious seizure is in progress or when muscle spasms are severe. There is also a liquid oral form of the drug available.

Precautions

The elderly, children, and those with significant health problems need to be carefully evaluated before receiving diazepam. Children under the age of six months should not take diazepam. In addition, people with a history of liver disease, kidney disease, or those with low levels of a protein in the blood called albumin need to be carefully assessed before starting this drug.

People taking diazepam should not drive, operate dangerous machinery, or engage in hazardous activities that require mental alertness, because diazepam can cause drowsiness. Alcohol and any drugs that treat mental illness should not be used when taking this medication. People who have previously had an allergic reaction to any dosage level of diazepam or any other benzodiazepine drug should not take diazepam. People with acute narrow-angle glaucoma should not take diazepam.

The prescribing physician should be consulted regularly if diazepam is taken consistently for more than two weeks. Diazepam and other drugs in this class can be habit-forming. Diazepam can become a drug of abuse and should be used with caution in patients with history of substance abuse. People taking diazepam should not stop taking the drug abruptly. This can lead to withdrawal effects such as shaking, stomach cramps, nervousness, and irritability.

Side effects

Anxiety, irregular heartbeat, forgetfulness, mental depression, and confusion are side effects that could require prompt medical attention. However, these side effects are not common when taking diazepam. Even less common, but serious events, are behavior changes, low blood pressure, muscle weakness, and the yellowing of the eyes or skin (jaundice). More common, but less serious side effects, include drowsiness, clumsiness, slurred speech, and dizziness. Rare among these less serious side effects are stomach cramps, headache, muscle spasm, nausea, vomiting, and dry mouth.

Once a person stops taking diazepam, the following side effects could occur from withdrawal: sleeping difficulties, nervousness, and irritability. Less common side effects from withdrawal include confusion, abdominal cramps, mental depression, sensitivity to light, nausea, shaking, and increased sweating. Rarely seen side effects include seizures, **hallucinations**, and feelings of distrust in the patient.

Interactions

Diazepam interacts with a long list of other medications. Anyone starting this drug should review the other medications they are taking with their physician and pharmacist for possible interactions. Patients should always inform all their health care providers, including dentists, that they are taking diazepam. Diazepam can add to the depressive effects of other central nervous system depressant drugs (for example, alcohol, other tranquilizers, or sleeping pills) when taken together. In severe cases, this can result in death.

Several drugs reduce the ability of diazepam to be broken down and cleared from the body. This results in higher levels of the drug in the blood and increases the probability that side effects will occur. These drugs include several antibiotics, such as erythromycin, anti-

stomach acid drugs, such as cimetidine (Tagamet), and antifungal drugs, such as fluconazole. Alcohol should not be used when taking diazepam and other benzodiazepine drugs. Other drugs that are used to treat mental disorders should not be combined with diazepam unless the patient is under the careful supervision and monitoring of a doctor.

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Diets

Definition

Special diets are designed to help individuals make changes in their usual eating habits or food selection. Some special diets involve changes in the overall diet, such as diets for people needing to gain or lose weight or eat more healthfully. Other special diets are designed to help a person limit or avoid certain foods or dietary components that could interfere with the activity of a medication. Still other special diets are designed to counter nutritional effects of certain medications.

Purpose

Special diets are used in the treatment of persons with certain mental disorders to:

- identify and correct disordered eating patterns
- prevent or correct nutritional deficiencies or excesses
- prevent interactions between foods or nutrients and medications

Special types of diets or changes in eating habits have been suggested for persons with certain mental disorders. In some disorders, such as eating disorders or substance abuse, dietary changes are an integral part of therapy. In other disorders, such as **attention-deficit/hyperactivity disorder**, various proposed diets have questionable therapeutic value.

Many medications for mental disorders can affect a person's appetite or nutrition-related functions such as saliva production, ability to swallow, bowel function, and activity level. Changes in diet or food choices may be required to help prevent negative effects of medications.

Finally, interactions can occur between some medications used to treat persons with mental disorders and certain foods or nutritional components of the diet. For example, grapefruit and apple juice can interact with some specific psychotropic drugs (medications taken for psychiatric conditions) and should be avoided by individuals taking those medicines. Tyramine, a natural substance found in aged or fermented foods, can interfere with the functioning of monoamine oxidase inhibitors and must be restricted in individuals using these types of medications. A person's pre-existing medical condition and nutritional needs should be taken into account when designing any special diet.

Special diets for specific disorders

Eating disorders

The two main types of eating disorders are **anorexia nervosa** and **bulimia nervosa**. Individuals with anorexia nervosa starve themselves, while individuals with bulimia nervosa usually have a normal or slightly above normal body weight but engage in **binge eating** followed by purging with laxatives, vomiting, or exercise.

Special diets for individuals with eating disorders focus on restoration of a normal body weight and control of bingeing and purging. These diets are usually carried out under the supervision of a multidisciplinary team, including a physician, **psychologist**, and dietitian.

The overall dietary goal for individuals with anorexia nervosa is to restore a healthy body weight. An initial goal might be to stop weight loss and improve food choices. Energy intake is then increased gradually until normal weight is restored. Because individuals with anorexia nervosa have an intense fear of gaining weight and becoming fat, quantities of foods eaten are increased very slowly so that the patient will continue treatments and therapy.

The overall dietary goal for individuals with bulimia nervosa is to gain control over eating behavior and to achieve a healthy body weight. An initial goal is to stabilize weight and eating patterns to help the individual gain control over the binge-purge cycle. Meals and snacks are eaten at regular intervals to lessen the possibility that

KEY TERMS

Anorexia—Loss of appetite or unwillingness to eat. Can be caused by medications, depression, or many other factors.

Anorexia nervosa—An eating disorder characterized by an intense fear of weight gain accompanied by a distorted perception of one's own underweight body.

Binge—An excessive amount of food consumed in a short period of time. Usually, while a person binge eats, he or she feels disconnected from reality, and feels unable to stop. The bingeing may temporarily relieve depression or anxiety, but after the binge, the person usually feels guilty and depressed.

Bulimia nervosa—An eating disorder characterized by binges in which large amounts of food are consumed, followed by forced vomiting.

Psychotropic drug—Medication that has an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

Purge—When a person rids him- or herself of extra food consumed. Purging may be accomplished by induced vomiting, laxative abuse, or excessive exercise.

Relapse—A person experiences a relapse when he or she re-engages in a behavior that is harmful and that he or she was trying to change or eliminate. Relapse is a common occurrence after treatment for many disorders, including addictions and eating disorders.

Thiamin—A B-vitamin that is essential to normal metabolism and nerve function, and whose absorption is affected by alcoholism.

Tyramine—Intermediate product between the chemicals tyrosine and epinephrine in the body and a substance normally found in many foods. Found especially in protein-rich foods that have been aged or fermented, pickled, or bacterially contaminated, such as cheese, beer, yeast, wine, and chicken liver.

hunger and fasting will trigger a binge. Once eating behaviors have been stabilized, energy intake can be gradually adjusted to allow the individual to reach a normal body weight healthfully.

For individuals with either anorexia nervosa and bulimia, continued follow-up and support are required even after normal weight and eating behaviors are restored, particularly since the rate of relapse is quite high. (Relapse occurs when a patient returns to the old behaviors that he or she was trying to change or eliminate.) In addition to dietary changes, **psychotherapy** is an essential part of the treatment of eating disorders and helps the individual deal with fears and misconceptions about body weight and eating behavior.

Attention-deficit/hyperactivity disorder

Attention-deficit/hyperactivity disorder (ADHD) accounts for a substantial portion of referrals to child mental health services. Children with ADHD are inappropriately active, easily frustrated or distracted, impulsive, and have difficulty sustaining concentration. Usual treatment of ADHD involves medication, behavioral management, and education.

Many dietary factors have been proposed as causes of ADHD, including sugar, food additives, and food allergies. In the 1970s the Feingold diet became popular for treatment of ADHD. The Feingold diet excludes artificial colorings and flavorings, natural sources of chemicals called salicylates (found in fruits), and preservatives called BHT and BHA. Although scientific evidence does not support the effectiveness of the Feingold diet, a modified Feingold diet including fruits has been shown to be nutritionally balanced and should not be harmful as long as the child continues to receive conventional ADHD treatment also.

A high intake of sugar and sugary foods has also been implicated as a cause of ADHD. Although carefully controlled studies have shown no association between sugar and ADHD, diets high in sugar should be discouraged because they are often low in other nutrients and can contribute to dental problems.

Food allergies have also been implicated as a cause of ADHD, and some groups have suggested using elimination diets to treat ADHD. Elimination diets omit foods that most commonly cause allergies in children, such as eggs, milk, peanuts, or shellfish. Although research does not support the value of elimination diets for all children with ADHD, children with specific food allergies can become irritable and restless. A child with a suspected food allergy should be evaluated by an allergist.

Stimulant medications used to treat ADHD, such as **methylphenidate** (Ritalin), can cause appetite loss (anorexia) and retard growth, although recent research suggests that a child's ultimate height appears not to be affected by stimulant medications. As a precaution, children on such medicines should receive close monitoring

of growth patterns, and parents should carefully observe their child's appetite and interest in meals and snacks. Providing regular meals and snacks, even when the child is not hungry, can help to assure adequate growth.

Mood disorders

Mood disorders include both depression (unipolar disorder) and episodes of mania followed by depression (**bipolar disorder**). Both types of disorders can affect appetite and eating behavior.

Although some depressed individuals eat more than usual and gain weight, depression more often causes loss of appetite and weight loss. As depressed individuals lose interest in eating and social relationships, they often skip meals and ignore feelings of hunger. Unintentional weight losses of up to 15% of body mass can occur.

Treatment with antidepressant medications often reverses weight loss and restores appetite and interest in eating. If the individual has lost a significant amount of weight, he or she may need to follow a high-calorie diet to restore weight to normal levels and replaced nutritional deficiencies. High-calorie diets usually include three balanced meals from all the food groups and several smaller snacks throughout the day. A protein/calorie supplement may also be necessary for some individuals.

Depression is sometimes treated with medications called monoamine oxidase inhibitors. Individuals on these medications will need to follow a tyramine-restricted diet (see below under monoamine oxidase inhibitors).

Individuals with mania are often treated with lithium. Sodium and caffeine intake can affect lithium levels in the blood, and intake of these should not suddenly be increased or decreased. Weight gain can occur in response to some antidepressant medications and lithium.

Schizophrenia

Individuals with **schizophrenia** can have **hallucinations**, delusional thinking, and bizarre behavior. These distorted behaviors and thought processes can also be extended to **delusions** and hallucinations about food and diet, making people with schizophrenia at risk for poor nutrition.

Individuals with schizophrenia may believe that certain foods are poisonous or have special properties. They may think they hear voices telling them not to eat. Some may eat huge quantities of food thinking that it gives them special powers. Individuals with untreated schizophrenia may lose a significant amount of weight. Delusional beliefs and thinking about food and eating usually improve once the individual is started on medication to treat schizophrenia.

Substance abuse

Substance abuse can include abuse of alcohol, cigarettes, marijuana, cocaine, or other drugs. Individuals abusing any of these substances are at risk for nutritional problems. Many of these substances can reduce appetite, decrease absorption of nutrients into the body, and cause the individual to make poor food choices.

Special diets used for withdrawal from substance abuse are designed to correct any nutritional deficiencies that have developed, aid in the withdrawal of the substance, and prevent the individual from making unhealthful food substitutions as the addictive substance is withdrawn. For example, some individuals may compulsively overeat when they stop smoking, leading to weight gain. Others may substitute caffeine-containing beverages such as soda or coffee for an addictive drug. Such harmful substitutions should be discouraged, emphasizing well-balanced eating combined with adequate rest, **stress** management, and regular exercise. Small, frequent meals and snacks that are rich in vitamins and minerals from healthful foods should be provided. Fluid intake should be generous, but caffeine-containing beverages should be limited.

Individuals withdrawing from alcohol may need extra thiamin supplementation, either intravenously or through a multivitamin supplement because alcohol metabolism in the body requires extra thiamin. Individuals taking drugs to help them avoid alcohol will need to avoid foods with even small amounts of alcohol (see below).

Common withdrawal symptoms and dietary suggestions for coping with these symptoms include:

- Appetite loss: eat small, frequent meals and snacks; limit caffeine; use nutritional supplements if necessary.
- Appetite increase: eat regular meals; eat a variety of foods; limit sweets and caffeine.
- Diarrhea: eat moderate amounts of fresh fruits, vegetables, concentrated sugars, juices, and milk; increase intake of cereal fiber.
- Constipation: drink plenty of fluids; increase fiber in the diet; increase physical activity.
- Fatigue: eat regular meals; limit sweets and caffeine; drink plenty of fluid.

Dietary considerations and medications

Medications that affect body weight

Many medications used to treat mental disorders promote weight gain, including:

- anticonvulsants (divalproex)
- certain types of antidepressants (**amitriptyline**)

- antipsychotic medications (**clozapine**, **olanzapine**, **quetiapine**, and risperidone)

Dietary treatments for individuals taking these medications should focus on a balanced, low-fat diet coupled with an increase in physical activity to counter the side effects of these medications. Nutrient-rich foods such as fruits, vegetables, and whole grain products should be emphasized in the diet, whereas sweets, fats, and other foods high in energy but low in nutrients should be limited. Regular physical activity can help limit weight gain caused by these medications.

Some medications can cause loss of appetite, restlessness, and weight loss. Individuals on such medications should eat three balanced meals and several smaller snacks of protein and calorie-rich foods throughout the day. Eating on a regular schedule rather than depending on appetite can help prevent weight loss associated with loss of appetite.

Medications that affect gastrointestinal function

Many psychiatric medications can affect gastrointestinal functioning. Some drugs can cause dry mouth, difficulty swallowing, constipation, altered taste, heartburn, diarrhea, or nausea. Consuming frequent smaller meals, drinking adequate fluids, modifying texture of foods if necessary, and increasing fiber content of foods can help counter gastrointestinal effects of medications.

Monoamine oxidase inhibitors

Individuals being treated with monoamine oxidase inhibitors (MAOIs) such as **tranylcypromine**, **phenelzine**, and isocarboxazid, must carefully follow a tyramine-restricted diet. Tyramine, a nitrogen-containing substance normally present in certain foods, is usually broken down in the body by oxidases. However, in individuals taking MAOIs, tyramine is not adequately broken down and builds up in the blood, causing the blood vessels to constrict and increasing blood pressure.

Tyramine is normally found in many foods, especially protein-rich foods that have been aged or fermented, pickled, or bacterially contaminated. Cheese is especially high in tyramine. A tyramine intake of less than 5 milligrams daily is recommended. A diet that includes even just 6 milligrams of tyramine can increase blood pressure; a diet that provides 25 milligrams of tyramine can cause life-threatening increases in blood pressure.

TYRAMINE-RESTRICTED DIET. Tyramine is found in aged, fermented and spoiled food products. The tyramine content of a specific food can vary greatly depending on storage conditions, ripeness, or contamination. Reaction to tyramine-containing foods in individuals taking MAOIs can also vary greatly depending on what other foods are eaten with the tyramine-containing food, the length of time between MAOI dose and eating the food, and individual characteristics such as weight, age, etc.

Foods to avoid on a tyramine-controlled diet include:

- all aged and mature cheeses or cheese spreads, including foods made with these cheeses, such as salad dressings, casseroles, or certain breads
- any outdated or nonpasteurized dairy products
- dry fermented sausages such as summer sausage, pepperoni, salami, or pastrami
- smoked or pickled fish
- non-fresh meat or poultry
- leftover foods containing meat or poultry
- tofu and soy products
- overripe, spoiled, or fermented fruits or vegetables
- sauerkraut
- fava or broad beans
- soups containing meat extracts or cheese
- gravies containing meat extracts or nonfresh meats
- tap beer
- nonalcoholic beer
- yeast extracts
- soy sauce
- liquid powdered protein supplements

Perishable refrigerated items such as milk, meat, or fruit should be eaten within 48 hours of purchase. Any spoiled food and food stored in questionable conditions should not be eaten.

Lithium

Lithium is often used to treat individuals with mania. Lithium can cause nausea, vomiting, anorexia, diarrhea, and weight gain. Almost one-half of individuals taking lithium gain weight.

Individuals taking lithium should maintain a fairly constant intake of sodium (found in table salt and other food additives) and caffeine in their diet. If an individual restricts sodium intake, less lithium is excreted in the urine and blood lithium levels rise. If an individual increases caffeine intake, more lithium is excreted in the urine and blood levels of lithium fall.

Anticonvulsant medications

Sodium caseinate and calcium caseinate can interfere with the action and effectiveness of some anticon-

vulsants. Individuals taking these anticonvulsants should read labels carefully to avoid foods containing these additives.

Psychotropic medications

Some psychotropic medications, such as amitriptyline, can decrease absorption of the vitamin riboflavin from food. Good food sources of riboflavin include milk and milk products, liver, red meat, poultry, fish, and whole grain, and enriched breads and cereals. Riboflavin supplements may also be needed.

Other psychotropic drugs, such as **fluvoxamine**, **sertraline**, fesasodone, **alprazolam**, **triazolam**, midazolam, **carbamazepine**, and **clonazepam**, interact with grapefruit juice, so individuals taking these drugs must take care to avoid grapefruit juice. In some cases, apple juice must be avoided, as well. Patients should discuss potential drug interactions with their doctor or pharmacist.

Caffeine-restricted diet

Caffeine is a stimulant and can interfere with the actions of certain medications. As stated, people taking lithium and people recovering from addictions may be asked by their treatment team to monitor (and, in the case of addictions, restrict) their caffeine intake. Foods and beverages high in caffeine include:

- chocolate
- cocoa mix and powder
- chocolate ice cream, milk, and pudding
- coffee
- cola beverages
- tea

Alcohol-restricted diet

Alcohol interacts with some medications used to treat mental disorders. In the case of alcoholism recovery, the negative interaction resulting from the combination of one medication (**disulfiram** or Antabuse) and alcohol consumption is actually part of treatment for some people. (The medication causes an extremely unpleasant reaction to any alcohol consumed, reinforcing or rewarding the avoidance of alcohol.)

When individuals are taking medication that requires that they avoid alcohol, foods containing alcohol must be avoided as well as beverage alcohol. The following foods contain small amounts of alcohol:

- flavor extracts, such as vanilla, almond, or rum flavorings
- cooking wines
- candies or cakes prepared or filled with liqueur
- apple cider
- cider and wine vinegar
- commercial eggnog
- bernaise or bordelaise sauces
- desserts such as crepes suzette or cherries jubilee
- teriyaki sauce
- fondues

See also Nutrition counseling; Nutrition and mental health

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Diphenhydramine

Definition

Diphenhydramine is an antihistamine used in psychiatric medicine to treat phenothiazine drug-induced abnormal muscle movement. It is also used in general medicine to treat allergies, allergic reactions, motion

KEY TERMS

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Extrapyramidal movement disorders—Involuntary movements that occur as a side effect of some psychiatric medications.

Histamine—Substance released during allergic reactions.

Hypokinesia—A condition of abnormally diminished motor activity.

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness.

Parkinsonism—A condition caused by the destruction of the brain cells that produce dopamine (a neurotransmitter), and characterized by tremors of the fingers and hands, a shuffling gait, and muscular rigidity.

Phenothiazine—A class of drugs widely used in the treatment of psychosis.

sickness, **insomnia**, cough, and nausea. When diphenhydramine is used for allergy-related symptoms, it is sold in the United States as an over-the-counter medication Benadryl. For use in the treatment of the tremors caused by phenothiazines, diphenhydramine is prescribed in the generic form.

Purpose

Some drugs called phenothiazines are used to treat psychotic disorder such as **schizophrenia**. As a side effect, these drugs may cause tremors and abnormal involuntary movements of the muscles, referred to as extrapyramidal neurologic **movement disorders**. Diphenhydramine is used to control these symptoms. Other uses of the drug include the treatment of nausea, vomiting, and itching. Diphenhydramine is used to help limit allergic reactions to transfused blood products. It can induce sleep. It is sometimes used to treat the stiffness and tremor of Parkinson's disease. In liquid form, it may relieve minor throat irritation.

Description

Diphenhydramine is an antihistamine that is readily distributed throughout the body. It is easily absorbed when taken by mouth. Maximal action occurs approximately one hour after swallowing the drug. The effects continue for four to six hours. Diphenhydramine acts on cells in the **brain**. It seems to compete with the chemical histamine for specific receptor sites on cells in the brain and central nervous system. This means that it achieves its theraputic effect by taking the place of the neurotransmitter histamine on these cells. Diphenhydramine is a useful medication for individuals with mild Parkinsonism when it is used in combination with centrally acting anticholinergic drugs.

Recommended dosage

The dosage of diphenhydramine must be adjusted according to the needs of individuals and their responses. Adults are generally given 25–50 mg orally, three to four times daily. Diphenhydramine may be administered through a vein or injected deep within a muscle. The usual dosage is 10–50 mg per injection, although some people may require 100 mg. The total daily dosage should not exceed 400 mg. People who forget to take a dose of this drug should skip the dose and take the next one at the regularly scheduled time. They should not double up subsequent doses if one is missed.

People should not take diphenhydramine if they are taking other preparations that contain antihistamines unless specifically directed to do so by a physician.

Precautions

People with peptic ulcer disease, bowel obstructions, an enlarged prostate, angle closure glaucoma, or difficulty urinating due to a blockage in the bladder should not use diphenhydramine without close physician supervision and monitoring. People with asthma, heart disease, high blood pressure, or an overactive thyroid should use this drug with caution. Before taking diphenhydramine, people with these conditions should discuss the risks and benefits of this drug with their doctor. Individuals should not take diphenhydramine for several days before an allergy test, as it will interfere with accurate results.

Elderly people are more sensitive to the sedating effects of diphenhydramine. The drug may also cause dizziness and lower blood pressure. Older people should slowly

change position from sitting or lying to standing while taking this medication to prevent dizziness and fainting.

Side effects

Drowsiness commonly occurs after taking diphenhydramine. This effect may be more pronounced if alcohol or any other central nervous system depressant, such as a tranquilizer or a particular medication for pain, is also taken. People taking the drug should not drive, or operate machinery, or perform hazardous tasks requiring mental alertness until the effects of the medication have worn off. In some people, diphenhydramine also may cause dizziness, difficulties with coordination, confusion, restlessness, nervousness, difficulty sleeping, blurry or double vision, ringing in the ears, headache, or convulsions.

Stomach distress is a relatively common side effect of diphenhydramine. Some people may develop poor appetites, nausea, vomiting, diarrhea, or constipation. Individuals also may experience low blood pressure, palpitations, rapid or irregular heartbeats, frequent urination, or difficulty urinating. Urine may be retained in the bladder. Other side effects of diphenhydramine are associated with persons in age groups that are unlikely to use the drug.

Diphenhydramine may also cause hives, a rash, sensitivity to the sun, and a dry mouth and nose. Thickened lung secretions are common among older persons.

Interactions

Alcohol, pain medications, sleeping pills, tranquilizers, and antidepressants may make the drowsiness associated with diphenhydramine more severe. Diphenhydramine should not be used by persons taking hay fever medicines, sedatives, narcotics, anesthetics, **barbiturates** or muscle relaxants.

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American College of Physicians. 190 N Independence Mall West, Philadelphia, PA 19106-1572. Telephone: (800) 523-1546, x2600 or (215) 351-2600. Web site: <http://www.acponline.org>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org>.

American Parkinson Disease Association, Inc. 1250 Hylan Boulevard, Suite 4B, Staten Island, NY 10305-1946. Telephone: (800) 223-2732 or (718) 981-8001. FAX: (718) 981-4399. Web site: <http://www.apdaparkinson.com>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. FAX (202) 682-6850.

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American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060.

Parkinson's Disease Foundation. 710 West 168th Street, New York, NY 10032-9982. Telephone: (800) 457-6676 or (212) 923-4700. FAX: (212) 923-4778. Web site: <http://www.pdf.org>.

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Disease concept of chemical dependency

Definition

Disease concept of chemical dependency is the concept that a disorder (such as chemical dependency) is like

a disease and has a characteristic set of signs, symptoms, and natural history (clinical course, or outcome).

Description

The disease concept has long been accepted by the medical community. The concept proposes that a disease is characterized by a specific set of signs and symptoms and that the disease, if left untreated, will progress to some endpoint or outcome (clinical course). However, controversy arises when the medical community is faced with new abnormal conditions, owing mostly to the new technologies in genetic engineering. This controversy becomes especially apparent when examining psychological disorders.

In the past, psychological disorders were thought in general to be due to both psychological and social abnormalities. Although these psychosocial problems are still of utmost importance, researchers have since discovered that many psychological disorders, such as alcoholism, also have genetic causes. Recent studies have identified a genetic area (locus) where a gene is located that can transmit alcoholism from affected father to son. Mental health professionals also know from clinical experience that alcoholics demonstrate a characteristic set of specific signs and symptoms. Additionally, it is well established that the ultimate clinical course for untreated alcoholism is death. Therefore alcoholism, once thought to be a disorder of those with a weak will, or "party people" can now be characterized as a disease.

Can it be inferred that other chemical dependencies may also have biological causes? There is compelling evidence that this theory may be correct. It is interesting to note that all psychoactive mood-altering drugs (alcohol, cocaine, marijuana, heroin, etc.) act in specific sites in the **brain** and on a specific neurotransmitter (a chemical that delivers impulses from one nerve cell to another) called dopamine. These mood-altering substances cause dopamine depletion, inducing an abnormality in nerve cells that "hijacks" the cells into chemical dependence. In other words, the substance introduced in the body affects the dopamine in a way that makes the affected individual unable to experience everyday pleasures—the individual instead needs that substance to experience pleasure. Thus the individual's driving force is any drug that can provide some kind of transient happiness (euphoria). In fact, the gene for alcoholism is located in the dopamine molecule. This can further suggest that chemical dependencies may have a medical (biological) cause.

The disease concept of chemical dependency is gaining worldwide acceptance, but does have some critics who argue instead that **addiction** must be understood as a general pattern of behavior, not as a medical problem. Advocates of the disease concept of chemical dependency model maintain that the identification of biological causes or correlations is crucially important for treatment. They argue that if clinicians can understand the intricate details concerning the mechanisms associated with drug effects, then measures to interrupt the effects can be devised. These interventions can be both medical (developing new drugs to chemical block effects of illicit drugs) and psychological.

According to the disease concept model, psychological **intervention** includes a vital educational component that teaches people with chemical dependency the concept of understanding addiction as disease. As a result of this understanding, affected people then view their dependency as a disease, similar to other diseases with a biological cause (heart disease, cancer, high blood pressure), and with a specific set of signs and symptoms and an outcome in the future (clinical course). Proponents of this approach believe that this understanding can help affected people to follow treatment recommendations, and can reduce shame and guilt commonly associated with chemical dependence. Alcoholics Anonymous is a prominent example of an organization that embodies the disease concept of chemical dependency.

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Disorder of written expression

Definition

Disorder of written expression, formerly called developmental expressive writing disorder, is a learning disability in which a person's ability to communicate in writing is substantially below the level normally expected based on the individual's age, intelligence, life experiences, educational background, or physical impairments. This disability affects both the physical reproduction of letters and words and the organization of thoughts and ideas in written compositions.

Description

Disorder of written expression is one of the more poorly understood learning disabilities. Learning disabilities that manifest themselves only in written work were first described in the late 1960s. These early studies described three main types of written disorders:

- inability to form letters and numbers correctly, also called dysgraphia
- inability to write words spontaneously or from dictation
- inability to organize words into meaningful thoughts

There are several difficulties in studying disorder of written expression and in implementing a remedial program. Disorder of written expression usually appears in conjunction with other reading or language disabilities, making it hard to separate manifestations of the disability related only to written expression. Delays in attention, visual-motor integration, visual processing, and expressive language may also contribute to writing disorders. Also, there are no standard tests specifically designed to evaluate disorder of written expression.

Causes and symptoms

Causes

The causes of disorder of written expression are unknown. Different manifestations of the disorder may have different causes. For example, people who cannot form letters correctly on the page (dysgraphia) may have delays in hand-eye coordination and difficulties concentrating. People who are unable to write words from memory or dictation appear to have deficits in their visual memory. They cannot remember what the words look like. People who produce legible script but cannot organize their thoughts on paper may be suffering from cognitive processing problems. Because disorder of written expression is a little-studied disorder, specific causes have not yet been determined.

Symptoms

Symptoms that suggest disorder of written expression include:

- poor or illegible handwriting
- poorly formed letters or numbers
- excessive spelling errors
- excessive punctuation errors
- excessive grammar errors
- sentences that lack logical cohesion
- paragraphs and stories that are missing elements and that do not make sense or lack logical transitions
- deficient writing skills that significantly impact academic achievement or daily life.

These symptoms must be evaluated in light of the person's age, intelligence, educational experience, and cultural or life experience. Written expression must be substantially below the level of samples produced by others of the same age, intelligence, and background. Normally, several of the symptoms are present simultaneously.

Demographics

Several studies have estimated that between 3% and 5% of students have disorder of written expression. However, it is difficult to separate this disorder from other **learning disorders**. Deficits in written work may be attributed to a reading, language, or attention disorders, limited educational background, or lack of fluency in the language of instruction. Disorder of written expression unassociated with any other learning disability is rare.

Diagnosis

There are no specific tests to diagnose disorder of written expression. This disorder is not normally diagnosed before age eight because of the variability with which children acquire writing skills. It is most commonly diagnosed in the fourth or fifth grade. Requests for testing usually originate with a teacher or parent who notes multiple symptoms of the disorder in a child's writing.

Several standardized tests accurately reflect spelling abilities, but do not assess other writing skills with the same reliability. Tests that might be helpful in diagnosing disorder of written expression include the Diagnostic Evaluation of Writing Skills (DEWS), the Test of Early Written Language (TEWL) and the Test of Adolescent Language. However, assessment using standardized tests is not enough to make a **diagnosis** of disorder of written expression. In addition, a qualified evaluator should compare multiple samples of the student's written work with the written work normally expected from students of comparable backgrounds. The person being evaluated may also be asked to perform tasks such as writing from dictation or copying written material as part of diagnostic testing. The American Psychiatric Association places disorder of written expression in the miscellaneous category of learning disorders not otherwise specified. It is likely to remain a poorly understood and diagnosed disability until more research findings are available.

Treatments

Little is known about how to treat disorder of written expression. Intense writing remediation may help, but no specific method or approach to remediation has

proved particularly successful. Since disorder of written expression usually occurs in conjunction with other learning disabilities, treatment is often directed at those better-understood learning problems.

Prognosis

Little is known about the long-term outcome for people with disorder of written expression. However, it appears that those who have this disorder may develop low self-esteem and social problems related to their lack of academic achievement. Later in life they may be more likely to drop out of school and find employment opportunities that require writing skills closed to them.

Prevention

There are no known ways to prevent disorder of written expression.

See also Learning disorders; Mathematics disorder; Reading disorder

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Learning Disabilities Association of America. 4156 Library Road Pittsburgh, PA 15234-1349. Telephone: (412) 341-1515. Web site: <www.ldanatl.org>.

National Center for Learning Disabilities, 381 Park Avenue South, Suite 1401, New York, NY 10016. (888) 575-7373 (toll free) or 212-545-7510. <www.ncld.org>.

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Dissociation and dissociative disorders

Definition

The dissociative disorders are a group of mental disorders that affect consciousness and are defined as causing significant interference with the patient's general functioning, including social relationships and employment.

Description

Dissociation is a mechanism that allows the mind to separate or compartmentalize certain memories or thoughts from normal consciousness. These split-off mental contents are not erased. They may resurface spontaneously or be triggered by objects or events in the person's environment.

Dissociation is a process that occurs along a spectrum of severity. If someone experiences dissociation, it does not necessarily mean that that person has a dissociative disorder or other mental illness. A mild degree of dissociation occurs with some physical stressors; people who have gone without sleep for a long period of time, have had "laughing gas" for dental surgery, or have been in a minor accident often have brief dissociative experiences. Another commonplace example of dissociation is a person becoming involved in a book or movie so completely that the surroundings or the passage of time are not noticed. Another example might be driving on the highway and taking several exits without noticing or remembering. Dissociation is related to hypnosis in that hypnotic trance also involves a temporarily altered state of consciousness. Most patients with dissociative disorders are highly hypnotizable.

People in other cultures sometimes have dissociative experiences in the course of religious (in certain trance states) or other group activities. These occurrences should not be judged in terms of what is considered "normal" in the United States.

Moderate or severe forms of dissociation are caused by such traumatic experiences as childhood **abuse**, combat, criminal attacks, brainwashing in hostage situations, or involvement in a natural or transportation disaster. Patients with **acute stress disorder**, **post-traumatic stress disorder** (PTSD), **conversion disorder,** or **somatization disorder** may develop dissociative symptoms. Recent studies of trauma indicate that the human **brain** stores traumatic memories in a different way than normal memories. Traumatic memories are not processed or integrated into a person's ongoing life in the same fashion as normal memories. Instead they are dissociated, or "split off," and may erupt into consciousness from time to time without warning. The affected person cannot control or "edit" these memories. Over a period of time, these two sets of memories, the normal and the traumatic, may coexist as parallel sets without being combined or blended. In extreme cases, different sets of dissociated memories may cause people to develop separate personalities for these memories— a disorder known as **dissociative identity disorder** (formerly called multiple personality disorder).

Types of dissociative disorders

Dissociative amnesia

Dissociative amnesia is a disorder in which the distinctive feature is the patient's inability to remember important personal information to a degree that cannot be explained by normal forgetfulness. In many cases, it is a reaction to a traumatic accident or witnessing a violent crime. Patients with dissociative **amnesia** may develop **depersonalization** or trance states as part of the disorder, but they do not experience a change in identity.

Dissociative fugue

Dissociative fugue is a disorder in which a person temporarily loses his or her sense of personal identity and travels to another location where he or she may assume a new identity. Again, this condition usually follows a major stressor or trauma. Apart from inability to recall their past or personal information, patients with dissociative fugue do not behave strangely or appear disturbed to others. Cases of dissociative fugue are more common in wartime or in communities disrupted by a natural disaster.

Depersonalization disorder

Depersonalization disorder is a disturbance in which the patient's primary symptom is a sense of detachment from the self. Depersonalization as a symptom (not as a disorder) is quite common in college-age populations. It is often associated with sleep deprivation or "recreational" drug use. It may be accompanied by "derealization" (where objects in an environment appear altered). Patients sometimes describe depersonalization as feeling like a robot or watching themselves from the outside. Depersonalization disorder may also involve feelings of numbness or loss of emotional "aliveness."

Dissociative identity disorder (DID)

Dissociative identity disorder (DID) is considered the most severe dissociative disorder and involves all of the major dissociative symptoms. People with this disorder have more than one personality state, and the personality state controlling the person's behavior changes from time to time. Often, a stressor will cause the change in personality state. The various personality states have separate names, temperaments, gestures, and vocabularies. This disorder is often associated with severe physical or sexual abuse, especially abuse suffered during childhood.

KEY TERMS

Amnesia—A general medical term for loss of memory that is not due to ordinary forgetfulness. Amnesia can be caused by head injuries, brain disease, or epilepsy, as well as by dissociation.

Depersonalization—A dissociative symptom in which the patient feels that his or her body is unreal, is changing, or is dissolving.

Derealization—A dissociative symptom in which the external environment is perceived as unreal.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Fugue—A dissociative experience during which a person travels away from home, has amnesia for their past, and may be confused about their identity but otherwise appears normal.

Hypnosis—The means by which a state of extreme relaxation and suggestibility is induced: used to treat amnesia and identity disturbances that occur in dissociative disorders.

Multiple personality disorder (MPD)—An older term for dissociative identity disorder (DID).

Trauma—A disastrous or life-threatening event that can cause severe emotional distress, including dissociative symptoms and disorders.

Dissociative disorder not otherwise specified (DDNOS)

DDNOS is a diagnostic category ascribed to patients with dissociative symptoms that do not meet the full criteria for a specific dissociative disorder.

Rebecca J. Frey, Ph.D.

Dissociative amnesia

Definition

Dissociative **amnesia** is classified by the ***Diagnostic and Statistical Manual of Mental Disorders***, 4th Edition, Text Revision, also known as the

KEY TERMS

Age-associated memory impairment (AAMI)—A condition in which an older person suffers some memory loss and takes longer to learn new information. AAMI is distinguished from dementia because it is not progressive and does not represent a serious decline from the person's previous level of functioning. Benign senescent forgetfulness is another term for AAMI.

Anterograde amnesia—Amnesia for events that occurred after a physical injury or emotional trauma but before the present moment. The type of amnesia that typically occurs in dissociative amnesia is anterograde.

Defense—An unconscious mental process that protects the conscious mind from unacceptable or painful thoughts, impulses, or desires. Examples of defenses include denial, rationalization, projection, and repression.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness. It can be distinguished from dissociative amnesia by its relatively sudden onset and variation in the severity of the symptoms.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Depersonalization—A dissociative symptom in which the patient feels that his or her body is unreal, is changing, or is dissolving.

Derealization—A dissociative symptom in which the external environment is perceived as unreal or dreamlike.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Dissociative amnesia—A dissociative disorder characterized by loss of memory for a period or periods of time in the patient's life. May occur as a result of a traumatic event.

Factitious disorder—A type of mental disturbance in which patients intentionally act physically or mentally ill without obvious benefits. It is distinguished from malingering by the absence of an obvious motive, and from conversion disorder by intentional production of symptoms.

Malingering—Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

Retrograde amnesia—Amnesia for events that occurred before a traumatic injury. Retrograde amnesia is not usually found in patients with dissociative amnesia.

Supportive—An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or educational approaches to treatment.

DSM-IV-TR as one of the dissociative disorders, which are mental disorders in which the normally well-integrated functions of memory, identity, perception, or consciousness are separated (dissociated). The dissociative disorders are usually associated with trauma in the recent or distant past, or with an intense internal conflict that forces the mind to separate incompatible or unacceptable knowledge, information, or feelings. In dissociative amnesia, the continuity of the patient's memory is disrupted. Patients with dissociative amnesia have recurrent episodes in which they forget important personal information or events, usually connected with trauma or severe **stress**. The information that is lost to the patient's memory is usually too extensive to be attributed to ordinary absentmindedness or forgetfulness related to aging. Dissociative amnesia was formerly called "psychogenic amnesia."

Amnesia is a symptom of other medical and mental disorders; however, the patterns of amnesia are different, depending on the cause of the disorder. Amnesia associated with head trauma is typically both retrograde (the patient has no memory of events shortly before the head injury) and anterograde (the patient has no memory of events after the injury). The amnesia that is associated with seizure disorders is sudden onset. Amnesia in patients suffering from **delirium** or **dementia** occurs in the context of extensive disturbances of the patient's cognition (knowing), speech, perceptions, emotions, and

behaviors. Amnesia associated with substance abuse, which is sometimes called "blackouts" typically affects only short-term memory and is irreversible. In dissociative amnesia, in contrast to these other conditions, the patient's memory loss is almost always anterograde, which means that it is limited to the period following the traumatic event(s). In addition, patients with dissociative amnesia do not have problems learning new information.

Dissociative amnesia as a symptom occurs in patients diagnosed with **dissociative fugue** and **dissociative identity disorder**. If the patient's episodes of dissociative amnesia occur only in the context of these disorders, a separate **diagnosis** of dissociative amnesia is not made.

Description

Patients with dissociative amnesia usually report a gap or series of gaps in their recollection of their life history. The gaps are usually related to episodes or abuse or equally severe trauma, although some persons with dissociative amnesia also lose recall of their own **suicide** attempts, episodes of self-mutilation, or violent behavior.

Five different patterns of memory loss have been reported in patients with dissociative amnesia:

- Localized. The patient cannot recall events that took place within a limited period of time (usually several hours or 1–2 days) following a traumatic event. For example, some survivors of the World Trade Center attacks do not remember how they got out of the damaged buildings or what streets they took to get away from the area.
- Selective. The patient can remember some, but not all of the events that took place during a limited period of time. For example, a veteran of D-Day (June 6, 1944) may recall some details, such as eating a meal on the run or taking prisoners, but not others (seeing a close friend hit or losing a commanding officer).
- Generalized. The person cannot recall anything in his/her entire life. Persons with generalized amnesia are usually found by the police or taken by others to a hospital emergency room.
- Continuous. The amnesia covers the entire period without interruption from a traumatic event in the past to the present.
- Systematized. The amnesia covers only certain categories of information, such as all memories related to a certain location or to a particular person.

Most patients diagnosed with dissociative amnesia have either localized or selective amnesia. Generalized amnesia is extremely rare. Patients with generalized, continuous, or systematized amnesia are usually eventually diagnosed as having a more complex dissociative disorder, such as dissociative identity disorder (DID).

Causes and symptoms

Causes

The primary cause of dissociative amnesia is stress associated with traumatic experiences that the patient has either survived or witnessed. These may include such major life stressors as serious financial problems, the death of a parent or spouse, extreme internal conflict, and guilt related to serious crimes or turmoil caused by difficulties with another person.

Susceptibility to hypnosis appears to be a predisposing factor in dissociative amnesia. As of 2002, however, no specific genes have been associated with vulnerability to dissociative amnesia.

Some personality types and character traits seem to be risk factors for dissociative disorders. A group of researchers in the United States has found that persons diagnosed with dissociative disorders have much higher scores for immature psychological defenses than normal subjects.

Symptoms

The central symptom of dissociative amnesia is loss of memory for a period or periods of time in the patient's life. The memory loss may take a variety of different patterns, as described earlier.

Other symptoms that have been reported in patients diagnosed with dissociative amnesia include the ollowing:

- Confusion.
- Emotional distress related to the amnesia. However, not all patients with dissociative amnesia are distressed. The degree of emotional upset is usually in direct proportion to the importance of what has been forgotten, or the consequences of forgetting.
- Mild depression.

Some patients diagnosed with dissociative amnesia have problems or behaviors that include disturbed interpersonal relationships, sexual dysfunction, employment problems, aggressive behaviors, self-mutilation, or suicide attempts.

Demographics

Dissociative amnesia can appear in patients of any age past infancy. Its true prevalence is unknown. In recent years, there has been an intense controversy

among therapists regarding the increase in case reports of dissociative amnesia and the accuracy of the memories recovered. Some maintain that the greater awareness of dissociative symptoms and disorders among psychiatrists has led to the identification of cases that were previously misdiagnosed. Other therapists maintain that dissociative disorders are overdiagnosed in people who are extremely vulnerable to suggestion.

It should be noted that psychiatrists in the U.S. and Canada have significantly different opinions of dissociative disorder diagnoses. On the whole, Canadian psychiatrists, both French- and English-speaking, have serious reservations about the scientific validity and diagnostic status of dissociative amnesia and dissociative identity disorder. Only 30% of Canadian psychiatrists think that these two dissociative disorders should be included in the *DSM-IV-TR* without reservation; and only 13% think that there is strong scientific support for the validity of these diagnoses.

Diagnosis

The diagnosis of dissociative amnesia is usually a diagnosis of exclusion. The doctor will take a detailed medical history, give the patient a physical examination, and order blood and urine tests, as well as an electroencephalogram (EEG) or head x ray in order to rule out memory loss resulting from seizure disorders, substance abuse (including abuse of inhalants), head injuries, or medical conditions, such as **Alzheimer's disease** or delirium associated with fever.

Some conditions, such as age-related memory impairment (AAMI), may be ruled out on the basis of the patient's age. **Malingering** can usually be detected in patients who are faking amnesia because they typically exaggerate and dramatize their symptoms; they have obvious financial, legal, or personal reasons (such as draft evasion) for pretending loss of memory. In addition, patients with genuine dissociative amnesia usually score high on tests of hypnotizability. The examiner may administer the Hypnotic Induction Profile (HIP) or a similar measure that evaluates whether the patient is easily hypnotized. This enables the examiner to rule out malingering or **factitious disorder**.

There are several standard diagnostic questionnaires that may be given to evaluate the presence of a dissociative disorder. The Dissociative Experiences Scale, or DES, is a frequently administered self-report screener for all forms of dissociation. The Structured Clinical Interview for the *DSM-IV-TR* Dissociative Disorders, or SCID-D, can be used to make the diagnosis of dissociative amnesia distinct from the other dissociative disorders defined by the *DSM-IV-TR*. The SCID-D is a semi-structured interview, which means that the examiner's questions are open-ended and allow the patient to describe experiences of amnesia in some detail, as distinct from simple "yes" or "no" answers.

As of 2002, there are no widely used screeners or diagnostic questionnaires specifically for dissociative amnesia.

Diagnosis of dissociative amnesia in children before the age of puberty is complicated by the fact that inability to recall the first four to five years of one's life is a normal feature of human development. As part of the differential diagnosis, a physician who is evaluating a child in this age group will rule out inattention, **learning disorders**, oppositional behavior, and **psychosis**, and seizure disorders or head trauma. To make an accurate diagnosis, several different people (i.e., teachers, therapists, **social workers**, the child's primary care physician) may be asked to observe or evaluate the child.

Treatments

Treatment of dissociative amnesia usually requires two distinct periods or phases.

Psychotherapy

Psychotherapy for dissociative amnesia is supportive in its initial phase. It begins with creating an atmosphere of safety in the treatment room. Very often, patients gradually regain their memories when they feel safe with and supported by the therapist. This rapport does not mean that they necessarily recover their memories during therapy sessions; one study of 90 patients with dissociative amnesia found that most of them had their memories return while they were at home alone or with family or close friends. The patients denied that their memories were derived from a therapist's suggestions, and a majority of them were able to find independent evidence or corroboration of their childhood abuse.

If the memories do not return spontaneously, hypnosis or sodium amytal (a drug that induces a semi-hypnotic state) may be used to help recover them.

After the patient has recalled enough of the missing past to acquire a stronger sense of self and continuity in their life history, the second phase of psychotherapy commences. During this phase, the patient deals more directly with the traumatic episode(s), and recovery from its aftereffects. Studies of the treatments for dissociative amnesia in combat veterans of World War I (1914–1918) found that recovery and cognitive integration of dissociated traumatic memories within the patient's overall personality were more effective than treatment methods that focused solely on releasing feelings.

Medications

At present, there are no therapeutic agents that prevent amnestic episodes or that cure dissociative amnesia itself. Patients may, however, be given antidepressants or other appropriate medications for treatment of the depression, anxiety, **insomnia**, or other symptoms that may accompany dissociative amnesia.

Legal implications

Dissociative amnesia poses a number of complex issues for the legal profession. The disorder has been cited by plaintiffs in cases of recovered memories of abuse leading to lawsuits against the perpetrators of the abuse. Dissociative amnesia has also been cited as a defense in cases of murder of adults as well as in cases of neonatricide (murder of an infant shortly after birth). Part of the problem is the adversarial nature of courtroom procedure in the U.S., but it is generally agreed that judges and attorneys need better guidelines regarding dissociative amnesia in defendants and plaintiffs.

Prognosis

The prognosis for recovery from dissociative amnesia is generally good. The majority of patients eventually recover the missing parts of their past, either by spontaneous re-emergence of the memories or through hypnosis and similar techniques. A minority of patients, however, are never able to reconstruct their past; they develop a chronic form of dissociative amnesia. The prognosis for a specific patient depends on a combination of his or her present life circumstances; the presence of other mental disorders; and the severity of stresses or conflicts associated with the amnesia.

Prevention

Strategies for the prevention of child abuse might lower the incidence of dissociative amnesia in the general population. There are no effective preventive strategies for dissociative amnesia caused by traumatic experiences in adult life in patients without a history of childhood abuse.

See also Abuse

Resources

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International Society for the Study of Dissociation (ISSD). 60 Revere Drive, Suite 500, Northbrook, IL 60062. (847) 480-0899. Fax: (847) 480-9282. <www.issd.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

National Organization for Rare Disorders, Inc. P. O. Box 8923, New Fairfield, CT 06812-8923. (203) 746-6518. <www.rarediseases.org>.

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Dissociative fugue

Definition

Dissociative fugue is a rare condition in which a person suddenly, without planning or warning, travels far from home or work and leaves behind a past life. Patients show signs of **amnesia** and have no conscious understanding or knowledge of the reason for the flight. The condition is usually associated with severe **stress** or trauma. Because persons cannot remember all or part of their past, at some point they become confused about their identity and the situations in which they find themselves. In rare cases, they may take on new identities. The American Psychiatric Association (APA) classifies disassociative fugue as one of four dissociative disorders, along with **dissociative amnesia**, **dissociative identity disorder**, and **depersonalization disorder**.

Description

The key feature of dissociative fugue is "sudden, unexpected travel away from home or one's customary place of daily activities, with inability to recall some or all of one's past," according to the APA. The travels associated with the condition can last for a few hours or as long as several months. Some individuals have traveled thousands of miles from home while in a state of dissociative fugue. (The word *fugue* stems from the Latin word for flight—*fugere*.) At first, a person experiencing the condition may appear completely normal. With time, however, confusion appears. This confusion may result from the realization that the person can not remember the past. Victims may suddenly realize that they do not belong where they find themselves.

During an episode of dissociative fugue, a person may take on a new identity, complete with a new name and even establish a new home and ties to their his/her community. More often, however, the victim realizes something is wrong not long after fleeing—in a matter of hours or days. In such cases, the victim may phone home for help, or come to the attention of police after becoming distressed at finding himself/herself unexplainably in unfamiliar surroundings.

Dissociative fugue is distinct from Dissociative Identity disorder (DID). In cases of DID, which previously was called Multiple Personality Disorder (MPD), a person loses memory of events that take place when one of several distinct identities takes control of the person. If a person with dissociative fugue assumes a new identity, it does not co-exist with other identities, as is typical of DID. Repeated instances of apparent dissociative fugue are more likely a symptom of DID, not true dissociative fugue.

Causes and symptoms

Causes

Episodes of dissociative fugue are often associated with very stressful events. Traumatic experiences such a war, or natural disasters, seem to increase the incidence of the disorder. Other, more personal types of stress might also lead to the unplanned travel and amnesia characteristic of dissociative fugue. The shocking death of a loved one or seemingly unbearable pressures at work or home, for example, might cause some people to run away for brief periods and blank out their pasts.

Symptoms

A person in the midst of a dissociative fugue episode may appear to have no psychiatric symptoms at all or to be only slightly confused. Therefore, for a time, it may be very difficult to spot someone experiencing a fugue. After a while, however, the patient shows significant signs of confusion or distress because he or she cannot remember recent events, or realizes a complete sense of identity is missing. This amnesia is a characteristic symptom of the disorder.

Demographics

Dissociative fugue is a rare disorder estimated to affect just 0.2% of the population, nearly all of them

KEY TERMS

Amnesia—A general medical term for loss of memory that is not due to ordinary forgetfulness. Amnesia can be caused by head injuries, brain disease, or epilepsy as well as by dissociation.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

adults. More people may experience dissociative fugue, however, during or in the aftermath of serious accidents, wars, natural disasters, or other highly traumatic or stressful events.

Diagnosis

The ***Diagnostic and Statistical Manual of Mental Disorders***, 4th Edition, Text Revision, also known as the *DSM-IV-TR* lists four criteria for diagnosing dissociative fugue:

- Unexplained and unexpected travel from a person's usual place of living and working along with partial or complete amnesia.
- Uncertainty and confusion about one's identity, or in rare instances, the adoption of a new identity.
- The flight and amnesia that characterize the fugue are not related exclusively to DID, nor is it the result of substance abuse or a physical illness.
- An episode must result in distress or impairment severe enough to interfere with the ability of the patient to function in social, work or home settings.

Accurate **diagnosis** typically must wait until the fugue is over and the person has sought help or has been brought to the attention of mental health care providers. The diagnosis can then be made using the patient's history and reconstruction of events that occurred before, during, and after the patient's excursion.

Treatments

Psychotherapy, sometimes involving hypnosis, is often effective in the treatment of dissociative fugue. Patients, with support from therapists, are encouraged to remember past events by learning to face and cope with the stressful experiences that precipitated the fugue. Since the cause of the fugue is usually a traumatic event, it is often necessary to treat disturbing feelings and emotions that emerge when the patient finally faces the trauma. The troubling events that drove a person to run and forget about his or her past may, when remembered, result in **grief**, depression, fear, anger, remorse, and other psychological states that require therapy.

Prognosis

The prognosis for dissociative fugue is often good. Not many cases last longer than a few months and many people make a quick recovery. In more serious cases, the patient may take longer to recover memories of the past.

See also Dissociative identity disorder

Resources

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ORGANIZATIONS

International Society for the Study of Dissociation. 60 Revere Dr., Suite 500, Northbrook, IL 60062. <http://www.issd.org/>.

National Alliance for the Mentally Ill. Colonial Place Three, 2107 Wilson Blvd., Suite 300, Arlington,VA 22021. <http://www.nami.org/index.html>.

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Dissociative identity disorder

Definition

Previously known as multiple personality disorder, dissociative identity disorder (DID) is a condition in which a person has more than one distinct identity or personality state. At least two of these personalities repeatedly assert themselves to control the affected person's

KEY TERMS

Alter—An alternate or secondary personality in a person with dissociative identity disorder. Each alter has a unique way of looking at and interacting with the world.

Amnesia—A general medical term for loss of memory that is not due to ordinary forgetfulness. Amnesia can be caused by head injuries, brain disease, or epilepsy as well as by dissociation.

Borderline personality disorder—A severe and usually life-long mental disorder characterized by violent mood swings and severe difficulties in sustaining interpersonal relationships.

Depersonalization—A dissociative symptom in which the patient feels that his or her body is unreal, is changing, or is dissolving.

Derealization—A dissociative symptom in which the external environment is perceived as unreal.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Dissociative identity disorder (DID)—Term that replaced Multiple personality disorder. A condition in which two or more distinctive identities or personality states alternate in controlling a person's consciousness and behavior.

Host—The dominant or main alter in a person with DID.

Hypnosis—The means by which a state of extreme relaxation and suggestibility is induced: used to treat amnesia and identity disturbances that occur in dissociative disorders.

Malingering—Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

Multiple personality disorder (MPD)—An older term for dissociative identity disorder (DID).

Panic disorder—An anxiety disorder in which an individual experiences sudden, debilitating attacks of intense fear.

Post-traumatic stress disorder—A disorder caused by an extremely stressful or traumatic event (such as rape, act of war, or natural disaster) in which the trauma victim is haunted by flashbacks. In the flashbacks, the event is re-experienced in the present. Other symptoms include nightmares and feelings of anxiety.

Primary personality—The core personality of a DID patient. In women, the primary personality is often timid and passive, and may be diagnosed as depressed.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Shift—The transition of control from one alter to another in a person with DID. Usually shifts occur rapidly, within seconds, but in some cases a more gradual change over is observed. Also referred to as a switch.

Somatization disorder—A type of mental disorder in which the patient suffers from physical complaints that serve as coping strategies for emotional distress.

Trauma—A disastrous or life-threatening event that can cause severe emotional distress. DID is associated with trauma in a person's early life or adult experience.

behavior. Each personality state has a distinct name, past, identity, and self-image.

Psychiatrists and psychologists use a handbook called the *Diagnostic and Statistical Manual of Mental Disorders* , fourth edition text revision or *DSM-IV-TR*, to diagnose mental disorders. In this handbook, DID is classified as a dissociative disorder. Other mental disorders in this category include **depersonalization disorder**, **dissociative fugue**, and **dissociative amnesia**. It should be noted, however, that the nature of DID and even its existence is debated by psychiatrists and psychologists.

Description

"Dissociation" describes a state in which the integrated functioning of a person's identity, including consciousness, memory and awareness of surroundings, is disrupted or eliminated. Dissociation is a mechanism that allows the mind to separate or compartmentalize certain

memories or thoughts from normal consciousness. These memories are not erased, but are buried and may resurface at a later time. Dissociation is related to hypnosis in that hypnotic trance also involves a temporarily altered state of consciousness. Dissociation occurs along a continuum or spectrum, and may be mild and part of the range of normal experience, or may be severe and pose a problem for the individual experiencing the dissociation. An example of everyday, mild dissociation is when a person is driving for a long period on the highway and takes several exits without remembering them. In severe, impairing dissociation, an individual experiences a lack of awareness of important aspects of his or her identity.

The phrase "dissociative identity disorder" replaced "multiple personality disorder" because the new name emphasizes the disruption of a person's identity that characterizes the disorder. A person with the illness is consciously aware of one aspect of his or her personality or self while being totally unaware of, or dissociated from, other aspects of it. This is a key feature of the disorder. It only takes two distinct identities or personality states to qualify as DID but there have been cases in which 100 distinct alternate personalities, or alters, were reported. Fifty percent of DID patients harbor fewer than 11 identities.

Because the alters alternate in controlling the patient's consciousness and behavior, the affected patient experiences long gaps in memory— gaps that far exceed typical episodes of forgetting that occur in those unaffected by DID.

Despite the presence of distinct personalities, in many cases one primary identity exists. It uses the name the patient was born with and tends to be quiet, dependent, depressed and guilt-ridden. The alters have their own names and unique traits. They are distinguished by different temperaments, likes, dislikes, manners of expression and even physical characteristics such as posture and body language. It is not unusual for patients with DID to have alters of different genders, sexual orientations, ages, or nationalities. Typically, it takes just seconds for one personality to replace another but, in rarer instances, the shift can be gradual. In either case, the emergence of one personality, and the retreat of another, is often triggered by a stressful event.

People with DID tend to have other severe disorders as well, such as depression, substance abuse, **borderline personality disorder** and eating disorders, among others. The degree of impairment ranges from mild to severe, and complications may include **suicide** attempts, self-mutilation, violence, or drug abuse.

Left untreated, DID can last a lifetime. Treatment for the disorder consists primarily of individual **psychotherapy**.

Causes and symptoms

Causes

The severe dissociation that characterizes patients with DID is currently understood to result from a set of causes:

- an innate ability to dissociate easily
- repeated episodes of severe physical or sexual **abuse** in childhood
- lack of a supportive or comforting person to counteract abusive relative(s)
- influence of other relatives with dissociative symptoms or disorders

The primary cause of DID appears to be severe and prolonged trauma experienced during childhood. This trauma can be associated with emotional, physical or sexual abuse, or some combination. One theory is that young children, faced with a routine of torture, sexual abuse or **neglect**, dissociate themselves from their trauma by creating separate identities or personality states. A manufactured alter may suffer while the primary identity "escapes" the unbearable experience. Dissociation, which is easy for a young child to achieve, thus becomes a useful defense. This strategy displaces the suffering onto another identity. Over time, the child, who on average is around six years old at the time of the appearance of the first alter, may create many more.

As stated, there is considerable controversy about the nature, and even the existence, of dissociative identity disorder. One cause for the skepticism is the alarming increase in reports of the disorder since the 1980s. An area of contention is the notion of suppressed memories, a crucial component in DID. Many experts in memory research say that it is nearly impossible for anyone to remember things that happened before the age three, the age when some DID patients supposedly experience abuse, but the brain's storage, retrieval, and interpretation of childhood memories are still not fully understood. The relationship of dissociative disorders to childhood abuse has led to intense controversy and lawsuits concerning the accuracy of childhood memories. Because childhood trauma is a factor in the development of DID, some doctors think it may be a variation of **post-traumatic stress disorder** (PTSD). In both DID and PTSD, dissociation is a prominent mechanism.

Symptoms

The major dissociative symptoms experienced by DID patients are **amnesia**, **depersonalization**, derealization, and identity disturbances.

AMNESIA. Amnesia in DID is marked by gaps in the patient's memory for long periods of their past, and, in some cases, their entire childhood. Most DID patients have amnesia, or "lose time," for periods when another personality is "out." They may report finding items in their house that they can't remember having purchased, finding notes written in different handwriting, or other evidence of unexplained activity.

DEPERSONALIZATION. Depersonalization is a dissociative symptom in which the patient feels that his or her body is unreal, is changing, or is dissolving. Some DID patients experience depersonalization as feeling to be outside of their body, or as watching a movie of themselves.

DEREALIZATION. Derealization is a dissociative symptom in which the patient perceives the external environment as unreal. Patients may see walls, buildings, or other objects as changing in shape, size, or color. DID patients may fail to recognize relatives or close friends.

IDENTITY DISTURBANCES. Persons suffering from DID usually have a main personality that psychiatrists refer to as the "host." This is generally not the person's original personality, but is rather one developed in response to childhood trauma. It is usually this personality that seeks psychiatric help. DID patients are often frightened by their dissociative experiences, which can include losing awareness of hours or even days, meeting people who claim to know them by another name, or feeling "out of body."

Psychiatrists refer to the phase of transition between alters as the "switch." After a switch, people assume whole new physical postures, voices, and vocabularies. Specific circumstances or stressful situations may bring out particular identities. Some patients have histories of erratic performance in school or in their jobs caused by the emergence of alternate personalities during examinations or other stressful situations. Each alternate identity takes control one at a time, denying control to the others. Patients vary with regard to their alters' awareness of one another. One alter may not acknowledge the existence of others or it may criticize other alters. At times during therapy, one alter may allow another to take control.

Demographics

Studies in North America and Europe indicate that as many as 5% of patients in psychiatric wards have undiagnosed DID. Partially hospitalized and out-patients may have an even higher incidence. For every one man diagnosed with DID, there are eight or nine women. Among children, boys and girls diagnosed with DID are pretty closely matched 1:1. No one is sure why this discrepancy between diagnosed adults and children exists.

Diagnosis

The *DSM-IV-TR* lists four diagnostic criteria for identifying DID and differentiating it from similar disorders:

- Traumatic stressor: The patient has been exposed to a catastrophic event involving actual or threatened death or injury, or a serious physical threat to him- or herself or others. During exposure to the trauma, the person's emotional response was marked by intense fear, feelings of helplessness, or horror. In general, stressors caused intentionally by human beings (genocide, rape, torture, abuse, etc.) are experienced as more traumatic than accidents, natural disasters, or "acts of God."
- The demonstration of two or more distinct identities or personality states in an individual. Each separate identity must have its own way of thinking about, perceiving, relating to and interacting with the environment and self.
- Two of the identities assume control of the patient's behavior, one at a time and repeatedly.
- Extended periods of forgetfulness lasting too long to be considered ordinary forgetfulness.
- Determination that the above symptoms are not due to drugs, alcohol or other substances and that they can't be attributed to any other general medical condition. It is also necessary to rule out fantasy play or imaginary friends when considering a **diagnosis** of DID in a child.

Proper diagnosis of DID is complicated because some of the symptoms of DID overlap with symptoms of other mental disorders. Misdiagnoses are common and include depression, **schizophrenia**, borderline personality disorder, **somatization disorder**, and **panic disorder**.

Because the extreme dissociative experiences related to this disorder can be frightening, people with the disorder may go to emergency rooms or clinics because they fear they are going insane.

When a doctor is evaluating a patient for DID, he or she will first rule out physical conditions that sometimes produce amnesia, depersonalization, or derealization. These conditions include head injuries, **brain** disease (especially seizure disorders), side effects from medications, substance abuse or intoxication, AIDS **dementia** complex, or recent periods of extreme physical **stress** and sleeplessness. In some cases, the doctor may give the patient an electroencephalograph (EEG) to exclude epilepsy or other seizure disorders. The physician also

must consider whether the patient is **malingering** and/or offering fictitious complaints.

If the patient appears to be physically healthy, the doctor will next rule out psychotic disturbances, including schizophrenia. Many patients with DID are misdiagnosed as schizophrenic because they may "hear" their alters "talking" inside their heads. If the doctor suspects DID, he or she can use a screening test called the Dissociative Experiences Scale (DES). If the patient has a high score on this test, he or she can be evaluated further with the Dissociative Disorders Interview Schedule (DDIS) or the Structured Clinical Interview for Dissociative Disorders (SCID-D).

Treatments

Treatment of DID may last for five to seven years in adults and usually requires several different treatment methods.

Psychotherapy

Ideally, patients with DID should be treated by a therapist with specialized training in dissociation. This specialized training is important because the patient's personality switches can be confusing or startling. In addition, many patients with DID have hostile or suicidal alter personalities. Most therapists who treat DID patients have rules or contracts for treatment that include such issues as the patient's responsibility for his or her safety. Psychotherapy for DID patients typically has several stages: an initial phase for uncovering and "mapping" the patient's alters; a phase of treating the traumatic memories and "fusing" the alters; and a phase of consolidating the patient's newly integrated personality.

Most therapists who treat multiples, or DID patients, recommend further treatment after personality integration, on the grounds that the patient has not learned the social skills that most people acquire in adolescence and early adult life. In addition, **family therapy** is often recommended to help the patient's family understand DID and the changes that occur during personality reintegration.

Many DID patients are helped by **group therapy** as well as individual treatment, provided that the group is limited to people with dissociative disorders. DID patients sometimes have setbacks in mixed therapy groups because other patients are bothered or frightened by their personality switches.

Medications

Some doctors will prescribe tranquilizers or antidepressants for DID patients because their alter personalities may have anxiety or mood disorders. However, other therapists who treat DID patients prefer to keep medications to a minimum because these patients can easily become psychologically dependent on drugs. In addition, many DID patients have at least one alter who abuses drugs or alcohol, substances which are dangerous in combination with most tranquilizers.

Hypnosis

While not always necessary, hypnosis (or **hypnotherapy**) is a standard method of treatment for DID patients. Hypnosis may help patients recover repressed ideas and memories. Further, hypnosis can also be used to control problematic behaviors that many DID patients exhibit, such as self-mutilation, or eating disorders like **bulimia nervosa**. In the later stages of treatment, the therapist may use hypnosis to "fuse" the alters as part of the patient's personality integration process.

Prognosis

Unfortunately, no systematic studies of the long-term outcome of DID currently exist. Some therapists believe that the prognosis for recovery is excellent for children and good for most adults. Although treatment takes several years, it is often ultimately effective. As a general rule, the earlier the patient is diagnosed and properly treated, the better the prognosis. Patients may find they are bothered less by symptoms as they advance into middle age, with some relief beginning to appear in the late 40s. Stress or substance abuse, however, can cause a relapse of symptoms at any time.

Prevention

Prevention of DID requires **intervention** in abusive families and treating children with dissociative symptoms as early as possible.

See also Dissociation and dissociative disorders

Resources

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ORGANIZATIONS

International Society for the Study of Dissociation, 60 Revere Dr., Suite 500, Northbrook, IL 60062. <http://www.issd.org/>.

National Alliance for the Mentally Ill. Colonial Place Three, 2107 Wilson Blvd., Suite 300, Arlington,VA 22021. <http://www.nami.org/helpline/did.html>.

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Disulfiram

Definition

Disulfiram is an aldehyde dehydrogenase inhibitor. It prohibits the activity of aldehyde dehydrogenase, an enzyme found in the liver. In the United States, disulfiram is sold under brand name Antabuse.

Purpose

Disulfiram is used as a conditioning treatment for alcohol dependence. When taken with alcohol, disulfiram causes many unwanted and unpleasant effects, and the fear of these is meant to condition the patient to avoid alcohol.

Description

Two Danish physicians who were investigating disulfiram for its potential benefits to destroy parasitic worms took disulfiram and became sick at a cocktail party. After a series of pharmacological and clinical studies, it was determined that disulfiram interacts with alcohol.

Disulfiram by itself is not toxic. If taken with alcohol, however, it alters certain steps in the breakdown of alcohol. When alcohol is ingested, it is converted first to a chemical called acetaldehyde. Acetaldehyde is further broken down into acetate. In order for acetaldehyde to be broken down into acetate, aldehyde dehydrogenase needs to be active. Disulfiram is an aldehyde dehydrogenase inhibitor. Since disulfiram blocks the activity of aldehyde dehydrogenase, acetaldehyde cannot be broken down and the levels of acetaldehyde become five to ten times higher than the normal levels. This causes uncomfortable effects that encourage the person to avoid alcohol.

Disulfiram comes in a 250- and 500-mg tablet.

Recommended dosage

Disulfiram therapy should be started only after the patient has abstained from alcohol for at least 12 hours. The initial dose may be as high as 500 mg taken once daily. If the medication is sedating, the dose can be administered in the evening. Ideally, though, the daily dose should be taken in the morning—the time the resolve not to drink may be strongest. The initial dosing period can last for one to two weeks.

Maintenance dose can range anywhere from 125–500 mg daily with the average dose being 250 mg daily. Disulfiram therapy should continue until full recovery. This may take months to years, depending upon patient's response and motivation to stop using alcohol. The duration of disulfiram's activity is 14 days after discontinuation, and patients need to avoid alcohol for this period of time.

Precautions

Before beginning therapy, patients should be carefully evaluated for their intellectual capacity to understand the goal of therapy, behavioral modification with negative **reinforcement**. Patients with history of **psychosis**, severe myocardial disease, and coronary occlusion should not take disulfiram. People with diabetes taking disulfiram are at an increased risk for complications. Severe liver failure has been associated with the use of disulfiram in patients with or without a prior history of liver problems. People with advanced or severe liver disease should not take disulfiram. Disulfiram should never be given to patients who are in a state of alcohol intoxication or without the patient's knowledge. Those patients with history of **seizures**, hypothyroidism, or nephritis need to use disulfiram with caution and close monitoring.

Besides avoiding alcohol, patients should also avoid any products containing alcohol. This includes many cold syrups, tonics, and mouthwashes. Patients should

KEY TERMS

Coronary occlusion—Blockage of the arteries supplying the blood to the heart.

Hypothyroidism—Thyroid gland that is abnormally low-functioning. A lowered metabolic rate results.

Jaundice—A yellowing of the skin caused by excess bilirubin in the blood; a liver disorder.

MAO inhibitors—A group of antidepressant drugs that decrease the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Myocardial disease—Disease of the muscular layer of the heart wall.

Nephritis—Inflammation of the kidney.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

not even use topical preparations that contain alcohol such as perfume and after-shave lotion.

Side effects

The most common side effect of disulfiram includes drowsiness and **fatigue**. Many patients experience metallic or garlic-like aftertaste, but most patients develop tolerance to this effect.

In addition, disulfiram is also associated with impotence. This is most common in doses of 500 mg daily. Disulfiram can also cause blurred vision, skin discoloration, inflammation of the skin, increased heart rate, and mental changes.

During the first three months of therapy, patients should have their liver function evaluated. Patients need to be monitored for the signs of jaundice, nausea, vomiting, abdominal pain, light stools and dark urine as these may be the signs of liver damage due to disulfiram. The signs of alcohol ingestion include flushing, headache, nausea, vomiting and abdominal pain.

Interactions

Disulfiram can make cisapride, benzodiazepines, astemizole, cyclosporine, erythromycin, and cholesterol-lowering drugs called statins more toxic. Disulfiram in combination with isoniazid, MAO inhibitors (such as phenelzide and **tranylcypromine**), metronidazole, omeprazole and tricyclic antidepressants may cause adverse central nervous system effects.

In addition, disulfiram may raise the concentrations of the medications theophylline and phenytoin in the body. Disulfiram may put patients on warfarin (a blood-thinning drug) at an increased risk of bleeding. Disulfiram should never be used with tranylcypromine and amprenavir oral solution.

Disulfiram may react even with small amounts of alcohol found in over-the-counter cough and cold preparations and any medication that comes in an elixir form.

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Divalproex sodium

Definition

Divalproex sodium is an anticonvulsant (anti-seizure) drug. It is also used to treat mania and to help prevent migraine headaches. It is sold under multiple brand names in the United States, including Depacon, Depakene, Depakote, and Depakote sprinkle.

Purpose

Divalproex sodium is effective in the treatment of epilepsy, particularly for preventing simple, complex (petit mal), absence, mixed, and tonic-clonic (grand mal) **seizures**. Divalproex sodium is also used to treat the manic phase of **bipolar disorder** (also called manic-depressive disorder) in adults, and to prevent migraine headache in adults.

KEY TERMS

Absence seizure—An epileptic seizure characterized by a sudden, momentary loss of consciousness, occasionally accompanied by some minor, jerky movements in the neck or upper arms, a twitching of the face, or a loss of muscle tone.

Addison's disease—Disease caused by malfunctioning adrenal glands that can be treated with cortisol replacement therapy. Symptoms include anemia, low blood pressure, digestive complaints, and diarrhea.

Alzheimer's disease—An incurable dementia marked by the loss of cognitive ability and memory over a period of 10–15 years. Usually affects elderly people.

Complex seizure—In complex seizures, the person experiences impaired consciousness.

Huntington's disease—A hereditary disorder that appears in middle age and is characterized by gradual brain deterioration, progressive dementia, and loss of voluntary movement. It is sometimes called Huntington's chorea.

Multiple sclerosis—A disease characterized by patches of hardened tissue in the brain or spinal cord, paralysis, and/or muscle tremors.

Neurons—Nerve cells in the brain that produce nerve impulses.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness.

Simple seizure—Simple partial seizures occur in patients who are conscious.

Tonic-clonic (grand mal) seizure—This is the most common type of seizure and is categorized into several phases beginning with vague symptoms hours or days before an attack. During the seizure, there is abnormal muscle contraction and relaxation and the individual may lose consciousness.

Description

Divalproex sodium is chemically compounded from sodium valproate and **valproic acid** in a 1:1 ratio.

Divalproex sodium is thought to work by increasing the levels of a **brain** neurotransmitter called gamma-aminobutyric acid (GABA). GABA is an inhibitory neurotransmitter, which means that its presence makes it harder for nerve cells (neurons) in the brain to become activated (fire). It is believed that increasing GABA's inhibitory action on brain neurons accounts for the ability of divalproex sodium to decrease seizures, curb manic behaviors, and decrease the frequency of migraine headaches.

Divalproex sodium was discovered to decrease the likelihood of seizure in 1963. In 1978, the United States Food and Drug Administration approved it for this use. Other uses for divalproex sodium were researched and approved subsequently, including use against mania (1995) and use to decrease migraine headache frequency. Divalproex sodium's 1995 approval as an anti-mania medication was considered an exciting advance, since it represented the first new drug introduced for this use in 25 years.

Recommended dosage

Divalproex sodium is available in tablets of 125 mg, 250 mg, and 500 mg. Divalproex sodium is also available in 125-mg capsules, and in a 500-mg extended release tablet. A syrup is also available, containing 250 mg active drug per 5 mL.

Divalproex sodium therapy is usually started at 10–15 mg per kg of body weight per day. Dosages are then increased until seizures seem to be well controlled. This is usually achieved at averages under 60 mg per kg per day.

To treat mania, divalproex sodium is usually started at a daily dose of about 750 mg.

For migraine prevention, divalproex sodium is started at 250 mg, twice per day. In some patients, this dose will have to be raised to a total of 1,000 mg per day.

Precautions

A greater risk of liver damage exists in patients with kidney disease, known liver disease, Addison's disease, blood diseases, children under the age of two, patients with organic brain diseases (such as Alzheimer's, Parkinson's, slow virus infections, Huntington's chorea, multiple sclerosis, etc.), patients with metabolic disorders present at birth, patients with severe seizure disorders and accompanying **mental retardation**, and patients who are taking several other anticonvulsant drugs.

Because divalproex sodium can affect a patient's blood by dropping the platelet (a type of blood cell that

affects clotting) count and interfering with coagulation (clotting) capability, both platelet count and coagulation parameters should be verified before starting the medication and at intervals throughout its use.

Divalproex sodium is known to cause an increased risk of birth defects when taken during pregnancy. An individual and her health care provider must weigh the potential risks and benefits of using this medication during pregnancy. Women who take this medicine should not breast-feed, since a small amount will pass into the breast milk

Divalproex sodium causes drowsiness and impairs alertness in some individuals. Patients just beginning to use the medication should avoid driving and using dangerous machinery until they determine how the drug affects them. The sedative effects are increased in the presence of alcohol, so patients should avoid drinking while taking medicines containing divalproex sodium.

Side effects

Some of the more common side effects of divalproex sodium include mild stomach cramps, change in menstrual cycle, diarrhea, loss of hair, indigestion, decreased appetite, nausea and vomiting, trembling in the hands and arms, and weight loss or weight gain. These side effects usually go away as the patient's body becomes accustomed to the medication.

Less common side effects include severe stomach cramps or continued nausea and vomiting, changes in mood, behavior, or thinking, double vision or seeing spots, severe **fatigue**, easy bruising or unusual bleeding, yellow cast to the skin or the whites of the eyes (jaundice), odd eye movements, and increased seizures. Patients who notice these symptoms should check with their doctor to see if their dosage or medication needs to be adjusted.

Rare side effects that should be checked out by a doctor include clumsiness, difficulty with balance, constipation, dizziness, drowsiness, headache, skin rash, agitation, restlessness, or irritability.

Interactions

Divalproex sodium is broken down (metabolized) in the liver. Other drugs that are metabolized in the liver can have too low or too high concentrations in the body when taken with divalproex sodium. Levels of divalproex sodium may be increased when taken with felbamate, isoniazid, salicylates (aspirin-containing medications), clarithromycin, erythromycin, and troleandomycin. Divalproex sodium may increase levels of **carbamazepine**, phenytoin, **lamotrigine**, nimodipine, phenobarbital, and zidovudine. Use with **clonazepam** may cause absence seizures. Cholestyramine and colestipol may reduce the absorption and the blood levels of divalproex sodium.

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Rosalyn Carson-DeWitt, M.D.

Dolophine *see* **Methadone**

Domestic abuse or domestic violence *see* **Abuse**

Donepezil

Definition

Donepezil is a drug used to treat **dementia** associated with **Alzheimer's disease**. In the United States, donepezil is sold under the brand name Aricept.

Purpose

Donepezil is used to help treat symptoms of Alzheimer's disease in individuals with mild to moderate illness. The drug may cause small improvements in dementia for a short period of time, but donepezil does not stop the progression of Alzheimer's disease.

Description

The Food and Drug Administration has approved donepezil for treatment of the symptoms of Alzheimer's disease. In Alzheimer's disease, some cells in specific regions of the **brain** die. Because of this cell death, these brain cells lose their ability to transmit nerve impulses. Brain cells normally transmit nerve impulses by secreting various chemicals known as **neurotransmitters**.

Brain cells that make and secrete a neurotransmitter called acetylcholine are affected early in the course of Alzheimer's disease. Donepezil helps prevent the breakdown of acetylcholine in the brain, thus temporarily increasing its concentration. In doing so, donepezil may improve the thinking process by facilitating nerve impulse transmission within the brain.

Donepezil is available as tablets in two different strengths. It is broken down by the liver.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Recommended dosage

The initial dosage of donepezil is 5 mg taken at bedtime. This dose should be continued for four to six weeks. The dosage may then be increased to 10 mg at bedtime, but there is no clear evidence that the higher dosage is more beneficial. However, the higher dosage is likely to cause more side effects.

Precautions

Donepezil may slow heart rate, increase acid in the stomach, make urination difficult, cause breathing difficulties, and may make it more likely for people to have **seizures**. As a result, it should be used carefully with close physician supervision by people with certain heart conditions, those who are prone to stomach ulcers, people with bladder obstruction, individuals with asthma or chronic obstructive pulmonary disease, and people with a history of seizure disorders.

People taking donepezil should be reassessed periodically to determine whether the drug is providing any benefits. When caregivers feel the drug is no longer beneficial, it may be stopped.

Side effects

More than 5% of people taking donepezil experience difficulty sleeping, dizziness, nausea, diarrhea, muscle cramps, headache, or other pains.

Diarrhea, nausea, and vomiting occur more often with the 10-mg dose than the 5-mg dosage. These adverse effects are usually mild, short-lived, and typically subside when the drug is stopped. Other, less common, side effects are abnormal dreams, depression, drowsiness, fainting, loss of appetite, weight loss, frequent urination, arthritis, and easy bruising.

Interactions

Many drugs may alter the effects of donepezil; likewise, donepezil may alter the action of other drugs. Drugs such as dicylomine, phenytoin, **carbamazepine**, dexamethasone, rifampin, or phenobarbital may lessen the effects of donepezil. Other drugs such as bethanechol, ketoconazole, or quinidine may increase some of the side effects associated with donepezil. When donepezil and nonsteroidal anti-inflammatory drugs such as ibuprofen (Advil) or naproxen are used together, there may be an increased tendency to develop stomach ulcers. Donepezil may increase the side effects associated with use of **fluvoxamine**, an antidepressant. If succinylcholine, a drug commonly used during anesthesia, is used with donepezil, prolonged muscle paralysis may result.

Resources

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Kelly Karpa, RPh, Ph.D.

Dopamine *see* **Neurotransmitters**

Doral *see* **Quazepam**

Doxepin

Definition

Doxepin is an oral antidepressant. It is sold in the United States under the brand name Sinequan and is also available under its generic name.

Purpose

Doxepin is used primarily to treat depression and to treat the combination of symptoms of anxiety and depression. Like most antidepressants, doxepin has also been used to treat **panic disorder**, **obsessive-compulsive disorder**, **attention-deficit/hyperactivity disorder**, **enuresis** (bed-wetting), eating disorders such as **bulimia nervosa**, cocaine dependency, and the depressive phase of bipolar (manic-depressive) disorder. It has also been used to support smoking cessation programs.

Description

Doxepin acts to change the balance of naturally occurring chemicals in the **brain** that regulate the transmission of nerve impulses between cells. Its action primarily increases the concentration of norepinephrine and serotonin (both chemicals that stimulate nerve cells) and, to a lesser extent, blocks the action of another brain chemical, acetylcholine. Although not technically a tricyclic antidepressant, doxepin shares most of the properties of these drugs, which include **amitriptyline**, **clomipramine**, **desipramine**, **imipramine**, **nortriptyline**, **protriptyline**, and **trimipramine**. Studies comparing doxepin with these other drugs have shown that doxepin is no more or less effective than other antidepressants of its type. Its choice for treatment is as much a function of physician preference as any other factor.

The therapeutic effects of doxepin, like other antidepressants, appear slowly. Maximum benefit is often not evident for at least two weeks after starting the drug. People taking doxepin should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Recommended dosage

As with any antidepressant, doxepin must be carefully adjusted by the physician to produce the desired therapeutic effect. Doxepin is available as 10-mg, 25-mg, 50-mg, 75-mg, 100-mg, and 150-mg oral capsules as well as an oral concentrate solution containing 10 mg of drug in each milliliter of solution.

Therapy is usually started at 30 to 150 mg per day and gradually increased to 300 mg daily if needed. There is little evidence that doses above 300 mg daily provide any additional benefits. Amounts up to 150 mg may be taken as a single dose at bedtime to decrease daytime sleepiness. Doses of more than 150 mg per day should be divided into two or three doses and taken throughout the day.

In patients over age 60, therapy should be maintained at the low end of the dosing range and increased cautiously and with physician supervision. Patients with organic brain syndrome (psychiatric symptoms of **dementia** often seen in elderly patients) generally require daily doses of only 25 to 50 mg.

If the oral concentrate of doxepin is used, each dose should be diluted in at least 4 ounces (120 mL) of milk, orange, prune, tomato, pineapple, or grapefruit juice just before administration. Doxepin is not compatible with many carbonated beverages and should not be diluted in them.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Benign prostate hypertrophy—Enlargement of the prostate gland.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Organic brain syndrome—A class of disorders characterized by progressive deterioration of mental processes caused by temporary brain dysfunction or permanent brain damage. Symptoms include delusions, dementia, amnesia, and delirium that are not caused by drugs, alcohol, or as a side effect of medication.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Precautions

As with tricyclic antidepressants, doxepin should be used cautiously and with close physician supervision in people, especially the elderly, who have benign prostatic hypertrophy, urinary retention, and glaucoma, especially angle-closure glaucoma (the most severe form). Before starting treatment, people with these conditions should discuss the relative risks and benefits of treatment with their doctors to help determine if doxepin is the right antidepressant for them.

A common problem with antidepressants is sedation (drowsiness, lack of physical and mental alertness). This side effect is especially noticeable early in therapy. In most patients, sedation decreases or disappears entirely with time, but until then, patients taking doxepin should not perform hazardous activities requiring mental alertness or coordination. The sedative effect is increased when doxepin is taken with other central nervous system depressants, such as alcoholic beverages, sleeping medications, other sedatives, or antihistamines. It may be dangerous to take doxepin in combination with these substances. Doxepin may increase the possibility of having **seizures**. Patients should tell their physician if they have a history of seizures, including seizures brought on by the abuse of drugs or alcohol. These people should use doxepin only with caution and be closely monitored by their physician.

Doxepin may increase heart rate and **stress** on the heart. It may be dangerous for people with cardiovascular disease, especially those who have recently had a heart attack, to take this drug or other antidepressants in the same pharmacological class. In rare cases where patients with cardiovascular disease must receive doxepin, they should be monitored closely for cardiac rhythm disturbances and signs of cardiac stress or damage.

Doxepin should not be taken by nursing mothers because it is secreted into breast milk and may cause side effects in the nursing infant.

Side effects

Doxepin shares the side effects of tricyclic antidepressants. The most frequent of these are dry mouth, constipation, urinary retention, increased heart rate, sedation, irritability, dizziness, and decreased coordination. As with most side effects associated with tricyclic antidepressants, the intensity is highest at the beginning of therapy and tends to decrease with continued use.

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Patients may also chew sugarless gum or suck on sugarless candy in order to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement who take doxepin may be especially likely to have problems with urinary retention. Symptoms include having difficulty starting a urine flow and more difficulty than usual passing urine. In most cases, urinary retention is managed with dose reduction or by switching to another type of antidepressant. In extreme cases, patients may require treatment with bethanechol, a drug that reverses this particular side effect. People who think they may be experiencing any side effects from this or any other medication should tell their physicians.

Interactions

Dangerously high blood pressure has resulted from the combination of antidepressants such as doxepin and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Because of this, doxepin should never be taken in combination with MAO inhibitors. Patients taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting doxepin or any tricyclic antidepressant. The same holds true when discontinuing doxepin and starting an MAO inhibitor.

Doxepin may decrease the blood pressure–lowering effects of **clonidine**. Patients who take both drugs should be monitored for loss of blood-pressure control and the dose of clonidine increased as needed.

The sedative effects of doxepin are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or medications used for other mental disorders such as **schizophrenia**. The anticholinergic effects of doxepin are additive with other anticholinergic drugs such as **benztropine**, **biperiden**, **trihexyphenidyl**, and antihistamines.

See also Neurotransmitters

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Jack Raber, Pharm.D.

Draw-a-person test *see* **Figure drawings**

DSM *see* ***Diagnostic and Statistical Manual of Mental Disorders***

Dual diagnosis

Definition

Dual **diagnosis** is a term that refers to patients who have both a mental health disorder and substance use disorder. It may be used interchangeably with "co-occurring disorders" or "comorbidity." According to the U.S. Substance Abuse and Mental Health Services Administration (SAMHSA), an estimated 10 million people in the United States will have a combination of at least one mental health and one substance abuse disorder in any twelve-month period. Substance abuse is the most common and significant co-occurring disorder among adults with such severe mental illnesses as **schizophrenia** or **bipolar disorder**. It may also be observed in individuals with mental health diagnoses that include depression, anxiety, **post-traumatic stress disorder**, or eating disorders. The term "substance abuse" refers to substance use disorders that range along a continuum from abuse to dependence or **addiction**.

The term "dual diagnosis" is considered to be misleading by some professionals because most people with this diagnosis actually have many problems rather than just two discrete illnesses. Occasionally, the term is used to describe a person with developmental disabilities and/or a mental health disorder or substance abuse disorder. More commonly, dual diagnosis refers to those with severe mental illness and a drug or alcohol abuse disorder, and who receive therapy in the public treatment systems.

Description

The prevalence of people with dual diagnoses became fully apparent to clinicians in the early 1980s. Initially, dual diagnoses were thought to be most likely in young adults with schizophrenia or bipolar disorder who also had extensive histories of drug or alcohol abuse. There was a widespread belief, often shared by family members of affected patients, that a young person's initiation into illegal drug use actually caused a subsequent mental illness. It is now more commonly thought that symptoms of the mental disorder generally appear first, and that the abuse of drugs or alcohol may represent the patient's attempt to self-medicate and alleviate the troublesome symptoms that accompany mental health disorders.

Today it is clear that the co-occurrence of mental illness and substance abuse is common: about 50% of individuals with severe mental illnesses are affected by substance abuse. A dual diagnosis is also associated with a host of negative outcomes that may include higher rates of relapse, **hospitalization**, incarceration, violence, **homelessness**, and exposure to such serious infections as HIV and hepatitis.

Despite almost twenty years of evidence regarding the prevalence and serious illnesses of people with dual diagnoses, the United States mental health and substance abuse systems continue to operate on parallel tracks, causing additional confusion to those with concurrent disorders. Refusal to combine services to provide better coordinated treatment has meant unnecessary suffering and expense for thousands of patients and their families.

For many people with dual diagnoses, the criminal justice system—juvenile as well as adult— becomes their *de facto* treatment system. Nearly two-thirds of incarcerated youth with substance abuse disorders have at least one other mental health disorder. The common association between **conduct disorder** or **attention-deficit/hyperactivity disorder** and substance abuse are two examples of combinations of serious and disabling disorders. A person in need of treatment for dual diagnoses who is in the current criminal justice system may not be evaluated or assessed, let alone provided with appropriate treatment.

Demographics

Children of alcohol or other drug-addicted parents are at increased risk for developing substance abuse and mental health problems. Disruptive behavior disorders coexist with adolescent substance abuse problems more often than not. Other special groups that may be affected include older adults with mood or anxiety disorders, especially those who are grieving numerous losses. They may drink or misuse or abuse prescription drugs to cope with their lowered quality of life. These factors can often complicate treatment of hypertension, diabetes, arthritis, and other health-related problems that affect the elderly as well.

Abuse of alcohol or other drugs may occur in persons with eating disorders in an effort to deal with guilt, shame, anxiety, or feelings of self-loathing as a result of bingeing and purging food. Many military veterans suffer from anxiety, depression or post-traumatic stress disorder and have histories of substance abuse. Services for veterans are woefully inadequate, adding to the chronic nature of dual diagnosis among them.

Treatment

One of the difficulties in treating patients with dual diagnoses is that most treatments for mental illness are usually developed for and validated by studies of patients with single diagnoses; therefore, many cases of comorbidity may not be well treated by these approaches. Recent research on services provided to people with dual diagnoses, however, indicates that treatment can be successful, provided certain specific components are included in the treatment process. The critical elements identified as part of treatment programs with the most successful outcomes are:

- Staged interventions that begin with engaging the client; persuading him or her to become involved in recovery-focused activities; acquiring skills and support to control the illnesses; and then helping the patient with relapse prevention.
- Assertive outreach that may involve intensive **case management** and meetings in the person's home.
- Motivational interventions to help the client become committed to self-management of their illnesses.
- Counseling that includes cognitive and behavioral skills.
- Social network support and/or family interventions.
- An understanding of the long-term nature of recovery.
- Comprehensive scope to treatment that includes personal habits, stress management, friendship networks, housing, and many other aspects of a person's life.
- Cultural sensitivity and competence.

The success of 12-step programs in the treatment of substance abuse is well-established. Nevertheless, the level of confrontation sometimes found in a traditional 12-step group may feel overwhelming to people with mental illnesses. In addition, the use of psychotropic (mood- or behavior-altering) medications is controversial in some areas of the substance abuse recovery community. As a result, other models of consumer-led **support groups** specifically for people with concurrent disorders, such as Dual Recovery Anonymous and Double Trouble, are being developed.

Resources

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ORGANIZATIONS

National Alliance for the Mentally Ill (NAMI). 2107 Wilson Blvd., Suite 300, Arlington, VA 22201. <http://www.nami.org>.

National Mental Health Association. 1021 Prince St., Alexandria, VA 22314 <http://www.nmha.org>.

National Mental Health Consumers' Self-Help Clearinghouse. 1211 Chestnut St, Suite 1207, Philadelphia, PA 19107. <http://www.mhselfhelp.org>.

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Dyslexia *see* **Reading disorder**

Dyspareunia

Definition

Dyspareunia is painful sexual intercourse. The same term is used whether the pain results from a medical or a psychosocial problem. Dyspareunia may be diagnosed in men and women, although the **diagnosis** is rare in men; when it does occur in men, it is almost always caused by a medical problem.

This discussion focuses only on pain with intercourse caused by psychosocial problems; therefore, only women's experiences are emphasized in this entry.

The professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***, Fourth edition, text revised (known as the *DSM-IV-TR*) classifies this condition as a sexual dysfunction.

Description

Dyspareunia is any pain experienced any time before, during, or following sexual intercourse. The pain may be located in the genitals or within the pelvis. It is not unusual for women occasionally to experience pain during intercourse. This is not true dyspareunia.

A woman who has dyspareunia often also has **vaginismus**. This is an involuntary tightening of the vaginal muscles in response to penetration. It can make intercourse painful, or impossible.

KEY TERMS

Dyspareunia—Painful sexual intercourse.

Vaginismus—An involuntary tightening of the vaginal muscles that makes sexual intercourse painful, difficult, or impossible.

Vulvar vestibulitis syndrome (VVS)—Vulvar vestibulitis syndrome is thought to be the most frequent cause of dyspareunia in premenopausal women. A chronic, persistent clinical syndrome, vulvar vestibulitis is characterized by severe pain on vestibular touch or attempted vaginal entry.

Causes and symptoms

Causes

Psychosocial causes of dyspareunia include:

- Prior sexual trauma. Many women who have been raped or sexually abused as children have dyspareunia. Even when a woman wishes to have sex with someone later, the act of intercourse may trigger memories of the trauma and interfere with her enjoyment of the act. Vaginismus also often occurs in such women.
- Guilt, anxiety, or tension about sex. Any of these can cause tense vaginal muscles and also prevent arousal from occurring. People who were raised with the idea that sex is bad may be more prone to have this problem. Fear of pregnancy may make arousal difficult.
- Prior physical trauma to the vaginal area. Women who have had an accidental injury or surgery in the vaginal area may become sensitive to penetration. Vaginismus is common in these cases, as well.
- Depression or anxiety in general. Either of these can lead to loss of interest in sex. This can be experienced by either sex.
- Problems in a relationship. Dyspareunia may occur when a woman feels her sexual partner is abusive or emotionally distant, she is no longer attracted to her partner, or she fears her partner is no longer attracted to her. Men, too, can lose interest in sex because of prior emotional trauma in a relationship; however, the result is usually impotence, rather than dyspareunia.
- Vasocongestion. Vasocongestion can occur when either partner frequently becomes aroused but does not reach orgasm. Vasocongestion is a pooling of blood in dilated blood vessels. Normally, the pelvic area becomes congested with blood when a person becomes sexually aroused. This congestion goes away quickly after orgasm. If there is no orgasm, the congestion takes much longer to resolve.

Any of these factors may cause painful sex. The affected person may then associate pain with sex and find it even harder to relax and become aroused in future.

Symptoms

The *DSM-IV-TR* diagnostic criteria for dyspareunia are as follows:

- Recurrent or persistent genital pain related to sexual intercourse that may occur before, during, or after intercourse.
- The affected person is distressed by the pain, or experiences relationship problems as a result of the pain.
- The pain is not caused exclusively by vaginismus or lack of lubrication, is not better accounted for by another disorder, and is not due exclusively to the direct effects of a drug, medication, or a general medical condition. Dyspareunia can occur with other sexual dysfunctions.

The most common symptom of dyspareunia from psychosocial causes is pain at the vaginal opening as the penis enters the vagina. Entry may be difficult, and the pain may be burning, or sharp. The woman may have a sense of being "dry." Pain may continue or ease as thrusting continues.

Vasocongestion can cause an aching pain in the pelvic area that persists for hours after intercourse. Pain with orgasm, or pain deep in the pelvis with thrusting, is more likely to be a sign of a medical problem, but can result from lack of arousal and tension.

A person who experiences pain during sex may feel embarrassed or ashamed. Dyspareunia can cause problems in relationships or lead to the affected person avoiding relationships altogether.

Demographics

About 15% of women may have pain with intercourse at some point in their lives. About 1–2% have true dyspareunia. The incidence is much higher in women who have been raped or otherwise sexually abused. As stated, dyspareunia in men is rare and is almost always caused by a medical problem.

Diagnosis

About 30% to 40% of all women who seek help from a sexual counselor for dyspareunia turn out to have a medical problem that is causing their pain. A full medical examination is necessary to rule out a possible medical cause. This includes a pelvic exam and may also include an ultrasound, as well as other diagnostic tests. Examples of possible physical causes are infections, sexually transmitted diseases (STDs), estrogen deficiencies, and vulvar vestibulitis.

Once a medical cause is ruled out, a full family and sexual history can help pinpoint possible psychosocial causes. A psychological evaluation can determine the cause of the problem. Women who have been raped or abused may also suffer from **post-traumatic stress disorder** (PTSD) or **generalized anxiety disorder**.

There are two types of dyspareunia. Lifelong or primary dyspareunia means that the condition has been present for the entire sexual life of the affected person. This type is usually associated with being raised to believe that sex is bad, sexual abuse, fear of sex, or a painful first sexual experience. Acquired or secondary dyspareunia begins after a period of normal sexual function. It often has a medical cause, but may be a result of some sort of trauma, such as rape.

Treatments

Counseling is often helpful to identify and reframe negative feelings about sex. **Couples therapy** can help improve communication between partners and resolve problems that may be a factor in the sexual relationship. Women who have been abused or raped may benefit from counseling techniques designed to help overcome fears and issues caused by traumatic experiences.

Sex therapy may be offered to provide information about the physical aspects of arousal and orgasm. A sex therapist will also offer suggestions for how to improve sexual technique. For example, increasing time for foreplay and allowing the woman to control when and how penetration occurs can help her to relax and become aroused more easily.

Women who also have vaginismus may be given a set of devices they can use at home to dilate the opening of the vagina. Affected women start with a very small device and gradually work up to a penis-sized device, proceeding to a larger size only when they can use the smaller one without pain or fear. This retrains the vaginal muscles and helps the involuntary muscle tightening of vaginismus.

Use of a vaginal lubricant, at least temporarily, may be helpful in some women to reduce anxiety about possible pain.

There are no specific medications that treat dyspareunia. Medications that increase blood flow or relax muscles may be helpful in some cases.

Prognosis

With treatment, the chance of overcoming dyspareunia and having an enjoyable sexual life is good. Treatment can take several months, particularly in the case of survivors of a violent trauma such as rape.

See also Erectile dysfunction; Post-traumatic stress disorder

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Dysthymic disorder

Definition

Dysthymic disorder is defined as a mood disorder with chronic (long-term) depressive symptoms that are present most of the day, more days than not, for a period of at least two years.

Description

Everyone experiences feelings of unhappiness and sadness occasionally. When these depressed feelings start to dominate everyday life and cause physical and mental deterioration, the feelings become known as depressive disorders. Depressive disorders can be categorized as **major depressive disorder** or dysthymic disorder. Individuals who suffer from dysthymic disorder have had their depressive symptoms for years— they often cannot pinpoint exactly when they started to feel depressed. People suffering from dysthymic disorder may describe to their doctor feelings of hopelessness, lowered self-esteem, poor concentration, indecisiveness, decreased

motivation, sleeping too much or too little, or eating too much or too little. Symptoms are present often and for the whole day, and are typically present for at least two years.

Causes and symptoms

Causes

The causes of depression are complex and not yet completely understood. Sleep abnormalities, hormones, **neurotransmitters** (chemicals that communicate impulses from one nerve cell to another), upbringing, heredity, and stressors (significant life changes or events that cause **stress**) all have been implicated as causes of depression.

Dysthymic disorder occurs in approximately 25% to 50% of persons who have sleep abnormalities that include reduced rapid eye movement (REM) sleep and impaired sleep continuity. Rapid eye movement sleep is an essential component of the sleep cycle and quality of sleep.

There is some evidence that suggests a correlation with hormonal imbalances of cortisol or thyroid hormones. In many adults, levels of cortisol (a stress hormone) are elevated during acute depressive periods and return to normal when the person is no longer depressed. In children and adolescents, results have been quite inconsistent, although there is some evidence that hypersecretion of cortisol is associated with more severe depressive symptoms and with a higher likelihood of recurrence of depression. A lack of thyroid hormone mimics depression quite well and is routinely checked in patients with recent onset depression.

In depression, there appears to be abnormal excess or inhibition of signals that control mood, thoughts, pain, and other sensations. Some studies suggest an imbalance of the neurotransmitter called serotonin. It is assumed that the reason antidepressants are effective is that they correct these chemical imbalances. For example, the selective serotonin reuptake inhibitors (SSRIs), one class of antidepressant medications that includes **fluoxetine** (Prozac), appears to establish a normal level of serotonin. As the name implies, the drug inhibits the re-uptake of serotonin neurotransmitter from the gaps between the nerve cells, thus increasing neurotransmitter action, alleviating depressive symptoms.

A child's upbringing may also be key in the development of dysthymic disorder. For example, it is speculated that if a person is abused and neglected throughout childhood and adolescence, a pattern of low self-esteem and negative thinking may emerge, and, from that, a lifelong pattern of depression may follow.

Heredity seems to play a role in the development of depressive disorders. People with major depression in their immediate family are up to three times more likely to have the disorder themselves. It would seem that biological and genetic factors may make certain individuals more prone to depressive disorders, but that environmental circumstances, or stressors, may then trigger the disorder.

KEY TERMS

Affect—The expression of emotion displayed to others through facial expressions, hand gestures, tone of voice, etc. Types of affect include: flat (inanimate, no expression), blunted (minimally responsive), inappropriate (incongruous expressions of emotion relative to the content of a conversation), and labile (sudden and abrupt changes in type and intensity of emotion).

Cortisol—A steroid hormone released by the cortex (outer portion) of the adrenal gland when a person is under stress.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Thyroid hormone—A complex hormone that regulates metabolic rate of all cells.

Symptoms

The mental health professional's handbook to aid patient **diagnosis** is the *Diagnostic and Statistical Manual of Mental Disorders,* also called the *DSM.* The 2000 edition of this manual is known as the *DSM-IV-TR* (fourth edition, text revised). The *DSM-IV-TR* has established a list of criteria that can indicate a diagnosis. These criteria include:

- Depressed mood for most of the day, more days than not.
- When depressed, two (or more) of the following are also present: decreased appetite or overeating, too much or too little sleep, low energy level, low self-esteem, decreased ability to concentrate, difficulty making decisions, and/ or feelings of hopelessness.

- During the two years of the disorder, the patient has never been without symptoms listed for more than two months at a time.
- No major depressive episode (a more severe form of depression) has been present during the first two years of the disorder.
- There has never been a manic disorder, and criteria for a less severe depression called **cyclothymic disorder** has never been established.
- The disorder does not exclusively occur with **psychosis**, **schizophrenia** or delusional illnesses.
- The symptoms of depression cause clinically significant impairment and distress in occupational, social, and general functioning. Dysthymic disorder can be described as "early onset" (onset before age 21 years), "late onset" (onset is age 21 years or older), and "with atypical features" (features that are not commonly observed).

Demographics

The lifetime prevalence has been estimated to be 4.1% for women and 2.2% for men. In adults, dysthymic disorder is more common in women than in men and research suggests that the prevalence in the age group 25 to 64 years is 6% for women. In children, dysthymic disorder can occur equally among both genders.

Diagnosis

To diagnose a patient with this disorder, the *DSM-IV-TR* criteria must be established, and this is accomplished through an extensive psychological interview and evaluation. The affected person seeking the clinician's help usually exhibits symptoms of irritability, feelings of worthlessness and hopelessness, crying spells, decreased sex drive, agitation, and thoughts of death. The clinician must rule out any possible medical conditions that can cause depressed **affect**. (Affect can be defined as the expression of emotion displayed to others through facial expressions, hand gestures, tone of voice, etc.) The diagnosis cannot be made if depression occurs during an active course of psychosis, **delusions**, schizophrenia, or **schizoaffective disorder**. If substance abuse is determined as the cause of depression, then a diagnosis of substance-induced mood disorder can be established.

Further psychological tests that can be administered to help in the diagnostic process include the **Beck Depression Inventory** and the **Hamilton Depression Scale**.

Treatments

The goals of treatment include remission of symptoms and psychological and social recovery.

Medications

Studies suggest some treatment success with medications such as tricyclic antidepressants (TCAs) or monoaminoxidase inhibitors (MAOIs). Medications can be effective in patients who have depression due to sleep abnormalities. Some tricyclic antidepressants include **amitriptyline** (Elavil), **imipramine** (Tofranil), and **nortriptyline** (Aventyl, Pamelor), and some MAOIs include **tranylcypromine** (Parnate) and **phenelzine** (Nardil). Selective serotonin reuptake inhibitors (SSRIs) are recommended during initial treatment planning after a definitive diagnosis is well established. The most commonly prescribed SSRIs are fluoxetine (Prozac), **sertraline** (Zoloft), **paroxetine** (Paxil), **fluvoxamine** (Luvox), and **citalopram** (Celexa).

Psychological therapies

Clinical reports suggest that **cognitive-behavioral therapy**, interpersonal **psychotherapy**, or **family therapy** can be effective with concurrent antidepressant medication to treat the symptoms of depression. In these therapies, the goal is to help the patient develop healthy problem-solving and coping skills.

Prognosis

Dysthymic disorder often begins in late childhood or adolescence. The disorder follows a chronic (long-term) course. The development of a more major form of clinical depression called major depressive disorder among children with dysthymic disorder is significant. In other words, childhood onset of dysthymic disorder is considered an early indicator for recurrent mood disorder that may even have more severe clinical symptoms in the patient's future.

Patients with this disorder usually have impaired emotional, social, and physical functioning.

In general, the clinical course of dysthymic disorder is not promising. Causes of a poorer outcome include not completing treatment, noncompliance with medication intake, and lack of willingness to change behaviors that promote a depressed state. However, patients can do very well with a short course of medications if they have a desire to follow psychotherapy treatment recommendations.

If left untreated, dysthymic disorder can result in significant financial and occupational losses. People with

this disorder tend to isolate themselves by restricting daily activities and spending days in bed. Patients often complain of poor health and incur more disability days when compared to the general population. Higher rates of successful outcome occur in people who undergo psychotherapy and treatment with appropriate medications.

Prevention

There is no known prevention for dysthymic disorder. Early **intervention** for children with depression may be effective in arresting the development of more severe problems.

See also Neurotransmitters; Origin of mental illness

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Linda Hesson, M.A., LLP, CAC

E

Eating disorders *see* **Anorexia nervosa and Bulimia nervosa**

EEG *see* **Electroencephalography**

Effexor *see* **Venlafaxine**

Elavil *see* **Amitriptyline**

Electroconvulsive therapy

Definition

Electroconvulsive therapy (ECT) is a medical procedure in which a small, carefully controlled amount of electric current is passed through the **brain** to treat symptoms associated with certain mental disorders. The electric current produces a convulsion for the relief of symptoms associated with such mental illnesses as **major depressive disorder**, **bipolar disorder**, acute **psychosis**, and **catatonia**.

Purpose

Also known as electroconvulsive shock therapy or electroshock therapy, ECT is used together with anesthesia, muscle relaxants and oxygen to produce a mild generalized seizure or convulsion. With repeated administration, usually over a period of weeks, ECT is highly effective in relieving symptoms of several mental illnesses.

The American Psychiatric Association's *Practice Guidelines for the Treatment of Psychiatric Disorders* discusses the use of ECT in the treatment of major depressive disorder, bipolar disorder and **schizophrenia**. Electroconvulsive therapy is administered to provide relief from the signs and symptoms of these and occasionally other mental illnesses. ECT is used routinely to treat patients with major depression, delusional depression, mania, and depression associated with bipolar disorder and schizophrenia. It is most closely associated with the treatment of severe depression, for which it provides the most rapid relief available as of 2002. In addition, patients suffering from catatonia, neuroleptic malignant syndrome, and parkinsonism may also benefit from the procedure.

ECT may become the treatment of first choice for depression if a patient with severe depression or psychotic symptoms is at increased risk of committing **suicide** and has not responded to other treatments. Although antidepressant medications are effective in many cases, they may take two to six weeks to begin to work. Some patients with mania and schizophrenia may not be able to tolerate the side effects of the antipsychotic medications used to treat these disorders. In addition, some patients may be unable to take their prescribed medications. For these individuals, ECT is an important option. ECT is also indicated when patients need a treatment that brings about rapid improvement because they are refusing to eat or drink, or presenting some other danger to themselves.

ECT is also recommended for certain subgroups of patients diagnosed with depression. Many elderly patients, for example, respond better to ECT than to antidepressant medications. Pregnant women are another subgroup that may benefit from ECT. Because ECT does not harm a fetus as some medications might, pregnant women suffering from severe depression can safely choose ECT for relief of their depressive symptoms.

Precautions

Candidates for ECT must be carefully screened. Prior to receiving this treatment, patients receive a thorough evaluation to identify any medical conditions they may have that might complicate their response to the procedure. This evaluation includes a complete medical history, a physical examination, and routine laboratory tests. In addition to standard blood tests, the patient should receive an electrocardiogram (EKG) to test for heart abnormalities. Evidence of a recent heart attack would

KEY TERMS

Acute psychosis—A severe mental disorder marked by delusions, hallucinations, and other symptoms that indicate that the patient is not in contact with reality.

Catatonia—Disturbance of motor behavior with either extreme stupor or random, purposeless activity.

Electroencephalography—The measurement and recording of the brain's electrical activity.

Informed consent—A person's agreement to undergo a medical or surgical procedure, or to participate in a clinical study, after being properly advised of the medical facts related to the procedure or study and the risks involved.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Neuroleptic—Another name for the older antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

Neuroleptic malignant syndrome (NMS)—An unusual but potentially serious complication that develops in some patients who have been treated with antipsychotic medications. NMS is characterized by changes in blood pressure, altered states of consciousness, rigid muscles, and fever. Untreated NMS can result in coma and death.

Parkinsonism—A condition caused by the destruction of the brain cells that produce dopamine (a neurotransmitter), and characterized by tremors of the fingers and hands, a shuffling gait, and muscular rigidity.

Psychomotor—Referring to a response or reaction that involves both the brain and muscular movements.

Psychotropic—Having an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

Relapse—A person experiences a relapse when he or she re-engages in a behavior that is harmful and that he or she was trying to change or eliminate. Relapse is a common occurrence after treatment for many disorders, including addictions and eating disorders.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tourniquet—A rubber tube or length of cloth that is used to compress a blood vessel in order to stop bleeding or to shut off circulation in a part of the body. The tourniquet is wrapped around the arm (or other limb) and tightened by twisting.

disqualify a patient from receiving ECT. Spinal and chest x rays can identify other physical conditions that might complicate a patient's response. Finally, a **computed tomography** (CT) scan should be performed to rule out any structural abnormalities in the brain that might be made worse by the electrical stimulation and resulting convulsions associated with ECT. Signs of a recent **stroke** or a tumor in the brain, for instance, would disqualify a patient as a candidate for ECT therapy.

The doctors who are administering the procedure must receive the **informed consent** of the patient a day before the first treatment is given. In addition, at least two psychiatrists should confirm that ECT is the proper treatment for a specific patient. One of these physicians should serve as the source of a "second opinion" and not be actively involved in treating the patient on a daily basis. This second, or outside, medical consultant should independently determine that ECT is appropriate for a particular patient after conducting a physical examination. The second physician should also confirm that the patient is mentally sound enough to give informed consent to the procedure.

Patients in any age group are eligible for treatment with ECT; however, informed consent for patients under 18 must be given by a parent or legal guardian.

Description

Early history of ECT

Ugo Cerletti and Lucio Bini, who were two Italian physicians working in the 1930s, were the first to use electroconvulsive therapy to treat patients with severe

mental illnesses. Their first patient was a 39-year-old unidentifiable homeless man who had been found wandering through the railroad station in Rome, mumbling incoherently. The doctors were inspired to try the new method by a notion that intrigued psychiatrists in this period, who were desperate for useful therapies— namely, that epilepsy and schizophrenia never appeared in the same person at the same time. (It was later shown, however, that it is possible for the same individual to suffer from both disorders at the same time.) Since epilepsy causes **seizures**, psychiatrists in the 1930s reasoned that artificially induced seizures might cure schizophrenia. Some in the medical community were receptive to this approach because physicians were already using a variety of chemicals to produce seizures in patients. Unfortunately, many of their patients died or suffered severe injuries because the strength of the convulsions could not be well controlled.

As ECT became more widely used, many members of the general public and some in the psychiatric profession were opposed to its use. To them it seemed barbaric and crude. ECT joined **psychosurgery** as one of the most intensely distrusted psychiatric and neurological practices. Many people were frightened simply because ECT was called "shock treatment." Many assumed the procedure would be painful; others thought it was a form of electrocution; and still others believed it would cause brain damage. Unfavorable publicity in newspapers, magazines and movies added to these fears. Indeed, from the 1930s up through the 1960s, doctors and nurses did not explain either ECT or other forms of psychiatric treatment to patients and their families very often. Moreover, many critics had good reasons for opposing the procedure before it was refined. Neither anesthesia nor muscle relaxants were used in the early days of ECT. As a result, patients had violent seizures, and even though they did not remember them, the thought of the procedure itself seemed frightening. Even more unfortunately, this crude, early version of ECT was applied sometimes to patients who could never have benefited from ECT under any conditions.

As the procedures used with ECT became more refined, psychiatrists found that ECT was an effective treatment for schizophrenia and soon after, depression and bipolar disorder. The use of ECT, however, was phased out when antipsychotic and antidepressant drugs were introduced during the 1950s and 1960s. The psychiatric community reintroduced ECT several years later when patients who didn't respond to the new drugs stimulated a search by mental health professionals for effective, and if necessary, non-drug treatments. While the new psychotropic medications provided relief for untold thousands of patients who suffered greatly from their illnesses and would otherwise have been condemned to mental hospitals, the drugs unfortunately produced a number of side effects, some of which are irreversible. Another drawback is that some medications do not have a noticeable effect on the patient's mood for two to six weeks. During this time, the patient may be at risk for suicide. In addition, there are patients who do not respond to any medications or who have severe allergic reactions to them. For these individuals, ECT may be the only treatment that will help.

ECT in contemporary practice

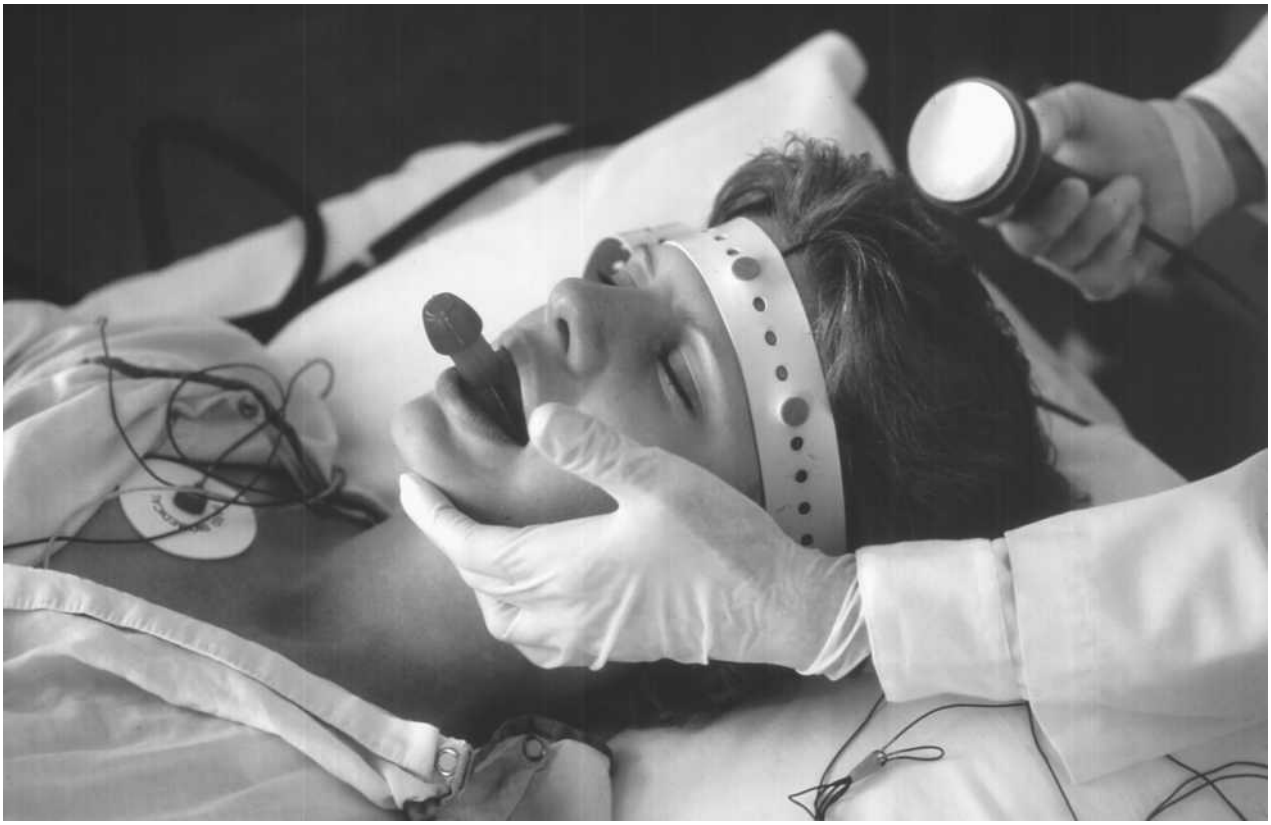
Today, with the introduction of improved safety procedures, ECT is a remarkably safe and highly effective procedure. It is performed in both inpatient and outpatient facilities in specially equipped rooms with oxygen, suction, and cardiopulmonary resuscitation equipment readily available to deal with the rare emergency. A team of health care professionals, including a **psychiatrist**, an anesthesiologist, a respiratory therapist, and other assistants, is present throughout the entire procedure.

As of 2000, the American Psychiatric Association has renewed its set of guidelines, first published in 1990, for determining the appropriate use of ECT in patients suffering from depression. They state that patients qualify for ECT if they:

- cannot tolerate, or receive no significant benefit from, antidepressant medications
- have responded well to ECT treatments during past depressive episodes
- face a greater risk from taking antidepressant drugs than from undergoing ECT
- need treatment without delay to avoid suicide or other self-destructive acts

Administration of ECT

ECT is performed while the patient is unconscious. Unconsciousness is induced by a short-acting barbiturate such as methohexital (Brevital sodium), or another appropriate anesthetic drug. The drug is given intravenously. To prevent the patient from harming themselves during the convulsions or seizures induced by ECT, he or she is given succinylcholine (Anectine) or a similar drug that temporarily paralyzes the muscles. Because the patient's muscles are relaxed, the seizures will not produce any violent contractions of the limbs and torso. Instead, the patient lies quietly on the operating table. One of the patient's hands or feet, however, is tied off with a tourniquet before the muscle relaxant is given. The tourniquet prevents the muscles in this limb from being paralyzed like the muscles in other parts of the



This woman has been prepared to receive electroconvulsive therapy— an effective treatment for depression. This patient has been given a short-acting medication that induces unconsciousness, and another medication was given that relaxes her muscles so that the induced seizures will not produce any violent contractions. Instead, the patient lies quietly on the operating table. The rubber mouthpiece keeps her from biting down on teeth or her tongue during the seizure. *(Photo Reasearchers, Inc. Reproduced by permission.)*

patient's body. The hand or foot is used to monitor muscle movement induced by the electrical current applied to the brain.

A breathing tube is then inserted into the unconscious patient's airway and a rubber mouthpiece is inserted into the mouth to prevent him or her from biting down on teeth or tongue during the electrically induced convulsion. As the current is applied, brain activity is monitored using **electroencephalography**. These brain wave tracings tell the medical team exactly how long the seizure lasts. The contraction of muscles in the arm or leg not affected by the muscle relaxant also provides an indication of the seizure's duration.

The electrodes for ECT may be placed on both sides of the head (bilateral) or one side (unilateral). Physicians often use bilateral electrode placement during the first week or so of treatments. An electric current is passed through the brain by means of a machine specifically designed for this purpose. The usual dose of electricity is 70–150 volts for 0.1–0.5 seconds. In the first stage of the seizure (tonic phase), the muscles in the body that have not been paralyzed by medication contract for a period of 5–15 seconds. This is followed by the second stage of the seizure (clonic phase) that is characterized by twitching movements, usually visible only in the toes or in a non-paralyzed arm or leg. These are caused by alternating contraction and relaxation of these same muscles. This stage lasts approximately 10–60 seconds. The physician in charge will try to induce a seizure that lasts between one-half and two minutes. If the first application of electricity fails to produce a seizure lasting at least 25 seconds, another attempt is made 60 seconds later. The session is stopped if the patient has no seizures after three attempts. The entire procedure, from beginning to end, lasts about 30 minutes.

The absence of seizures is most commonly caused either by the patient's physical condition at the time of treatment or by the individual nature of human responses to drugs and other treatment procedures. Just as there are some patients who do not respond to one type of antidepressant medication but do respond to others, some patients do not respond to ECT.

The total number of ECT treatments that will be given depends on such factors as the patient's age, **diagnosis**, the history of illness, family support and response to therapy. Treatments are normally given every other day with a total of two to three per week. The ECT treatments are stopped when the patient's psychiatric symptoms show significant signs of improvement. Depending on the patient's condition, this improvement may happen in a few weeks or, rarely, over a six-month period. In most cases, patients with depression require between six and twelve ECT sessions.

Only rarely is ECT treatment extended beyond six months. In such infrequent cases, treatments are decreased from two to four per week after the first month to one treatment every month or so.

No one knows for certain why ECT is effective. Because the treatment involves passing an electric current through the brain, which is electrically excitable tissue, it is not surprising that ECT has been shown to affect many neurotransmitter systems. **Neurotransmitters** are chemical messengers in the nervous system that carry signals from nerve cell to nerve cell. The neurotransmitters affected by ECT include dopamine, norepinephrine, serotonin and GABA (gamma-aminobutyric acid).

Preparation

Patients and their relatives are prepared for ECT by viewing a videotape that explains both the procedure and the risks involved. The physician then answers any questions these individuals might have, and the patient is asked to sign an informed consent form. This form gives the doctor and the hospital legal permission to administer the treatment.

After the form has been signed, the doctor performs a complete physical examination and orders a number of tests that can help identify any potential problem. These tests may include a chest x ray; an electrocardiogram (EKG); a CT scan; a urinalysis; a spinal x ray; a brain wave tracing (EEG); and a complete blood count (CBC).

Some medications, such as lithium and a class of antidepressants known as monoamine oxidase inhibitors (MAOIs), should be discontinued for some time before ECT administration. Patients are instructed not to eat or drink for at least eight hours prior to the procedure in order to reduce the possibility of vomiting and choking. During the procedure itself, the members of the health care team closely monitor the patient's vital signs, including blood pressure, heart rate and oxygen content.

Aftercare

The patient is moved to a recovery area after an ECT treatment. Vital signs are recorded every five minutes until the patient is fully awake, which may take 15–30 minutes. The patient may experience some initial confusion, but this feeling usually disappears in a matter of minutes. The patient may complain of headache, muscle pain, or back pain, which can be quickly relieved by aspirin or another mild medication.

Following successful ECT treatments, patients with bipolar disorder may be given maintenance doses of lithium. Similarly, patients with depression may be given antidepressant drugs. These medications are intended to reduce the chance of relapse or the recurrence of symptoms. Some studies have estimated that approximately one-third to one-half of patients treated with ECT relapse within 12 months of treatment. After three years, this figure may increase to two-thirds. Follow-up care with medications for bipolar disorder or depression can reduce the relapse rate in the year following ECT treatment from 50% to 20%. Some patients might relapse because they do not respond well to the medications they take after their ECT sessions are completed. In some cases, patients who relapse may suffer from severe forms of depression that are especially difficult to treat by any method.

Risks

Recent advances in medical technology have substantially reduced the complications associated with ECT. These include memory loss and confusion. Persons at high risk of having complications following ECT include those with a recent heart attack, uncontrolled high blood pressure, brain tumors, and previous spinal injuries.

One of the most common side effects of electroconvulsive therapy is memory loss. Patients may be unable to recall events that occurred before and after treatment. Elderly patients, for example, may become increasingly confused and forgetful as the treatments continue. In a minority of individuals, memory loss may last for months. For the majority of patients, however, recent memories return in a few days or weeks.

Elderly patients receiving ECT may experience disturbances in heart rhythm; slow heartbeat (bradycardia); or rapid heartbeat (tachycardia); and an increased number of falls. As many as one-third of elderly patients may experience such complications following the procedure.

Normal results

ECT often produces dramatic improvement in the signs and symptoms of major depression, especially in

elderly patients. Sometimes the benefits are evident even during the first week of treatment.

A remarkable 90% of patients who receive ECT for depression respond positively. By contrast, only 70% respond as well when treated with antidepressant medications alone. While it is estimated that as many as 50% of successfully treated patients will have future episodes of depression, the prognosis for each episode of illness is good. Mania also often responds well to treatment with ECT. The picture is not as bright for schizophrenia, which is more difficult to treat and is characterized by frequent relapses.

Post-treatment confusion and forgetfulness are common, though disturbing, symptoms associated with ECT. Doctors and nurses must be patient and supportive by providing patients and their families with factual information about the nature and timeframe of the patient's recovery.

A few patients are placed on maintenance ECT. This term means that they must return to the hospital every one to two months as needed for an additional treatment. These persons are thus able to keep their illness under control and lead normal and productive lives.

Abnormal results

If an ECT-induced seizure lasts too long (more than two minutes) during the procedure, physicians will control it with an intravenous infusion of an anticonvulsant drug, usually **diazepam** (Valium).

Overall, ECT is a very safe procedure. The complications encountered are no different from those that may occur with the administration of anesthesia without ECT. There is no convincing evidence of long-term harmful effects from ECT. Researchers are continuing to explore its potential in treating other disorders.

See also Catatonic disorder; Neurotransmitters

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Electroencephalography

Definition

Electroencephalography (EEG) is a neurological diagnostic procedure that records the changes in electrical potentials (**brain** waves) in various parts of the brain.

Purpose

The EEG is an important aid in the **diagnosis** and management of epilepsy and other seizure disorders, as well as in the diagnosis of brain damage related to trauma and diseases such as strokes, tumors, encephalitis, and drug and alcohol intoxication. The EEG is also useful in monitoring brain wave activity and in the determination of brain death. Research is active in determining the role of EEG in the diagnosis and management of **mental retardation**, **sleep disorders**, degenerative diseases such as **Alzheimer's disease** and Parkinson's disease, and in certain mental disorders such as **autism** and **schizophrenia**.

Precautions

The EEG should be administered, monitored, and interpreted only by a specially trained health professional. It is important to recognize that diagnosis should not be based on the EEG alone—the EEG represents an adjunct to the neurological history, examination, and other specialized studies. The EEG is an extremely sensitive instrument, and tracings can be greatly influenced by the actions and the physiologic status of the patient. It is important that the patient be properly prepared physically and psychologically in order to obtain an accurate and reliable record. Medications such as anticonvulsants, tranquilizers, stimulants—including coffee, tea, cola drinks—and alcohol should be withheld for at least 24–48 hours prior to the test. Inasmuch as hypoglycemia affects brain wave patterns, the patient is told not to withhold any meals.

Description

Brain function is associated with electrical activity, which is always accompanied by an electrical field. This field consists of two parts, the electrical field and the magnetic field, and is called an electromagnetic field. The electrical field is measured by surface electrodes and is recorded by the electroencephalogram. Prior to the recording session, approximately 16–20 electrodes are attached to the patient's scalp with a conductive washable paste, or collodion. Depending on the purpose of the EEG, implantable needle electrodes may be utilized, in which case the patient should be informed that there will be mild discomfort.

The patient lies on a bed, padded table, or comfortable reclining chair and is asked to remain quiet and relaxed during the approximately one hour that is usually required. A sleep recording up to three hours in duration is usually obtained if the diagnosis is a seizure disorder. Under certain conditions, various stimuli such as flashing lights or deep breathing may be utilized. In an ambulatory EEG recording, the patient is attached to a portable cassette recorder and goes about regular activities, usually for up to 24 hours.

Magnetoencephalography

Magnetoencephalography, a supplement to EEG, also uses an electroencephalogram to measure the patient's electrical field. In addition, however, the patient's magnetic field is also recorded to measure electrical activity. Every electrical current generates a magnetic field. The magnetic field is detected by an instrument called a biomagnetometer and recorded as a magnetoencephalograph (MEG). The information provided by the MEG is entirely different from that provided by **computed tomography** (CT), topographic encephalography, or **magnetic resonance imaging** (MRI)—imaging instruments that provide still, structural, and anatomical information. The information recorded by the MEG provides important supplemental information to that recorded by the encephalogram and, used together and conjointly, they both provide a much more complete and comprehensive idea of cerebral events. Using MEG, the brain can be observed "in action" rather than just being viewed as a still image.

KEY TERMS

Encephalitis—Inflammation of the brain.

Occipital—The occipital bone forms the back part of the skull.

Magnetoencephalography has been used to map the sensory and motor cortices of the brain, to determine the organization of the auditory center of the brain, and to study cognitive functions such as speech, memory, attention and consciousness. This information is critical for neurosurgical planning such as the removal of brain lesions. Thus, preoperative MEG is valuable in planning the surgical treatment of tumors and malformations. MEG can provide surgeons with real-time computer-generated images of deep-seated lesions that are essential before surgery. The quantitative EEG is also known by the acronym BEAM (brain electrical activity mapping).

Preparation

Prior to the EEG, the patient is given full instructions in the procedure, particularly about the avoidance of certain medications and food. In cases where a sleep EEG is anticipated, the patient may be requested to minimize sleep or stay awake the night before the procedure. Sedatives to induce sleep should be avoided, if possible.

Aftercare

No specific procedures or aftercare are required. Patients are advised to resume their usual activities, especially the resumption of medications that had been temporarily discontinued.

Risks

The primary risk of EEG is the production of a seizure in an epileptic patient. This may result from the

This woman is undergoing an electroencephalogram (EEG) to diagnose Alzheimer's disease. On the computer screen at the right are the colored scans of the electrical activity in her brain. Alzheimer's patients show a specific abnormality in their EEGs. *(Catherine Pouedras. Science Photo Library, National Audubon Society Collection/Photo Researchers, Inc. Reproduced by permission.)*

temporary discontinuation of anticonvulsant medication or from the provocation of a seizure by an epileptogenic stimulus such as flashing lights or deep breathing. Although the provocation of a seizure may serve to substantiate the diagnosis, all potential seizure patients should be carefully monitored to avoid injury in case a seizure does result.

Normal results

The rate, height, and length of brain waves vary depending on the part of the brain being studied, and every individual has an unique and characteristic brainwave pattern. Age and state of consciousness also cause changes in wave patterns. Several wave patterns have been identified:

- Alpha waves: Most of the recorded waves in a normal adult's EEG are the occipital alpha waves, which are best obtained from the back of the head when the subject is resting quietly with the eyes closed but not asleep. These waves, occurring typically in a pattern of eight to 13 cycles per second, are blocked by excitement or by opening the eyes.
- Beta waves: These waves, obtained from the central and frontal parts of the brain, are closely related to the sensory-motor parts of the brain and are also blocked by opening the eyes. Their frequency is in the range of 8–30 hertz (cycles per second).
- Delta waves: These are irregular, slow waves of 2–3 hertz and are normally found in deep sleep and in infants and young children. They indicate an abnormality in an awake adult.
- Theta waves: These are characterized by rhythmic, slow waves of 4–7 hertz.

Abnormal results

EEG readings of patients with epilepsy or other seizure disorders display bursts, or spikes, of electrical activity. In focal epilepsy, spikes are restricted to one hemisphere of the brain. If spikes are generalized to both hemispheres, multifocal epilepsy may be indicated.

Diagnostic brain-wave patterns of other disorders varies widely. The appearance of excess theta waves (four to eight cycles per second) may indicate brain injury. Brain wave patterns in patients with brain disease, mental retardation, and brain injury show overall slowing. A trained medical specialist should interpret EEG results in the context of the patient's medical history and other pertinent medical test results.

See also Alcohol and related disorders; Sleep terror disorder; Sleepwalking disorder; Substance abuse and related disorders

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Elimination disorders

Definition

Elimination disorders are disorders that concern the elimination of feces or urine from the body. The causes of these disorders may be medical or psychiatric.

KEY TERMS

Constipation—Difficult bowel movements caused by the infrequent production of hard stools.

Feces—Waste products eliminated from the large intestine; excrement.

Incontinence—The inability to control the release of urine or feces.

Laxative—Substance or medication that encourages a bowel movement.

Stools—Feces, bowel movements.

Description

The American Psychiatric Association recognizes two elimination disorders, **encopresis** and **enuresis**. Encopresis is an elimination disorder that involves repeatedly having bowel movements in inappropriate places after the age when bowel control is normally expected. Encopresis is also called fecal incontinence. Enuresis, more commonly called bed-wetting, is an elimination disorder that involves release of urine into bedding, clothing, or other inappropriate places. Both of these disorders can occur during the day (diurnal) or at night (nocturnal). They may be voluntary or involuntary. Encopresis and enuresis may occur together, although most often they occur separately.

Elimination disorders may be caused by a physical condition, a side effect of a drug, or a psychiatric disorder. It is much more common for elimination disorders to be caused by medical conditions than psychiatric ones. In most cases in which the cause is medical, the soiling is unintentional. When the causes are psychiatric, the soiling may be intentional, but it is not always so.

Encopresis

Medical causes of encopresis are usually related to chronic constipation. As hard feces build up in the large intestine, the bowel is stretched out of shape. This allows liquid feces behind the hard stool to involuntarily leak out and stain clothing. Other medical causes of encopresis include malformations of the bowel and side effects of medication. Laxatives (medications that relieve constipation), drugs that kill some of the good bacteria in the intestines, and drugs that increase contractions in the intestines can all cause involuntary encopresis. Pediatricians or family physicians treat almost all cases of encopresis having medical causes. In cases of prolonged involuntary soiling,

children may develop feelings of shame and embarrassment, leading to low self-esteem.

Psychiatric causes of encopresis are not as clear. A few children may experience encopresis because of fear of the toilet or because their toilet training was either overly pressured or irregular and incomplete. Older children may soil intentionally, sometimes smearing the feces on wall or clothing or hiding feces around the house. Children who show this pattern of soiling behavior often have clinical behavior problems such as **conduct disorder** or **oppositional defiant disorder**. About one-quarter of children who soil intentionally also have enuresis.

Enuresis

Enuresis also has both medical and psychiatric causes. Primary enuresis occurs when a child has never established bladder control. Medical causes of primary enuresis are often related to malformations of the urinary system, developmental delays, and hormonal imbalances that affect the ability to concentrate urine. There appears to be a genetic component to primary enuresis, since the condition tends to run in families. Primary enuresis may also be caused by psychological stressors such as family instability or erratic toilet training.

Secondary enuresis occurs when a child has established good bladder control for a substantial period, then begins wetting again. Involuntary secondary enuresis is thought to be brought on by life stresses. For example, it is common for young children to begin wetting the bed after moving to a new house or having a new sibling enter the family. Voluntary enuresis is not common. Like voluntary encopresis, it is associated with psychiatric conditions such as conduct disorder and oppositional defiant disorder.

Treatment and prognosis

Most children outgrow their elimination disorders successfully by the time they are teens, with the exception of those children whose elimination disorders are symptoms of other psychiatric disturbances.

Encopresis is treated with stool softeners or laxatives and by instituting regular bowel evacuation patterns. Enuresis is treated by **behavior modification** including changing nighttime toileting habits. The least expensive and most effective method is by having the child sleep on a special pad that sets off an alarm when the pad becomes wet. This wakes the child and allows him to finish relieving in the toilet. Eventually he awakes without assistance before wetting. Drugs can also help in the treatment of enuresis, although relapse is common after they are stopped. Secondary enuresis caused by **stress** is treated by resolving the stress. **Psychotherapy** is usually not needed, although it may be helpful to children who develop feelings of shame associated with their elimination disorders. Adults can help children avoid shame and embarrassment by treating elimination accidents matter-of-factly and kindly.

Children with voluntary elimination disorders are treated for the diagnosed psychiatric problem associated with the elimination disorder using behavior modification, drugs, and other psychiatric interventions.

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Enabling behaviors *see* **Addiction**

Encopresis

Definition

Encopresis is an elimination disorder that involves repeatedly having bowel movements in inappropriate places after the age when bowel control is normally expected. Encopresis is also called "soiling" or "fecal incontinence."

Description

By four years of age, most children are toilet trained for bowel movements. After that age, when inappropriate bowel movements occur regularly over a period of several months, a child may be diagnosed with encopresis. Encopresis can be intentional on unintentional. Intentional soiling is associated with several psychiatric disorders. Involuntary or unintentional soiling is often the result of constipation.

Causes and symptoms

The only symptom of encopresis is that a person has bowel movements in inappropriate places, such as in clothing or on the floor. This soiling is not caused by taking laxatives or other medications, and is not due to a disability or physical defect in the bowel. There are two main types of encopresis, and they have different causes.

Involuntary encopresis

With involuntary encopresis, a person has no control over elimination of feces from the bowel. The feces is semi-soft to almost liquid, and it leaks into clothing without the person making any effort to expel it. Leakage usually occurs during the day when the person is active, and ranges from infrequent or almost continuous.

Involuntary soiling usually results from constipation. A hard mass of feces develops in the large intestine and is not completely expelled during a regular bowel movement in the toilet. This mass then stretches the large intestine out of shape, allowing liquid feces behind it to leak out. Up to 95% of encopresis is involuntary.

Although involuntary encopresis, called by the American Psychiatric Association (APA) encopresis with constipation and overflow incontinence, is caused by constipation, the constipation may be the result of psychological factors. Experiencing a stressful life event, harsh toilet training, toilet fear, or emotionally disturbing events can cause a child to withhold bowel movements or become constipated. Historically, children separated from their parents during World War II are reported to have shown a high incidence of encopresis, indicating that psychological factors play a role in this disorder.

Voluntary encopresis

A person with voluntary encopresis has control over when and where bowel movements occur and chooses to have them in inappropriate places. Constipation is not a factor, and the feces is usually a normal consistency. Often feces is smeared in an obvious place, although sometimes it is hidden around the house. The APA classifies voluntary encopresis as encopresis without constipation and overflow incontinence.

KEY TERMS

Feces—Waste products eliminated from the large intestine; excrement.

Incontinence—The inability to control the release of urine or feces.

Laxative—Substance or medication that encourages a bowel movement.

Stools—Feces; bowel movements.

In young children, voluntary encopresis may represent a power struggle between the child and the caregiver doing the toilet training. In older children, voluntary encopresis is often associated with **oppositional defiant disorder** (ODD), **conduct disorder**, sexual **abuse**, or high levels of psychological stressors.

Demographics

Encopresis occurs in 1–3% of children and is seen more often in boys than in girls. The frequency of encopresis appears to be independent of social class, and there is no evidence that it runs in families.

Diagnosis

To receive an APA **diagnosis** of encopresis, a child must have a bowel movement, either intentional or accidental, in an inappropriate place at least once a month for a minimum of three months. In addition, the child must be chronologically or developmentally at least four years old, and the soiling cannot be caused by illness, medical conditions (such as chronic diarrhea, spina bifida, anal stenosis, etc.), medications, or disabilities. However, it may be caused by constipation.

Treatments

Involuntary encopresis is treated by addressing the cause of the constipation and establishing soft, pain-free stools. This can include:

- increasing the amount of liquids a child drinks
- adding high-fiber foods to the diet
- short-term use of laxatives or stool softeners
- emptying the large intestine by using an enema
- establishing regular bowel habits

Once the constipation is resolved, involuntary encopresis normally stops.

Treatment of voluntary encopresis depends on the cause. When voluntary encopresis results from a power struggle between child and adult, it is treated with **behavior modification**. In addition to taking the steps listed above to ensure a soft, pain-free stool, the adult should make toileting a pleasant, pressure-free activity. Some experts suggest transferring the initiative for toileting to the child instead of constantly asking him/her to use the toilet. Others recommend toileting at scheduled times, but without pressure to perform. In either case, success should be praised and failure treated in a matter-of-fact manner. If opposition to using the toilet continues, the family may be referred to a child **psychiatrist** or a pediatric **psychologist**.

With older children who smear or hide feces, voluntary encopresis is usually a symptom of another more serious disorder. When children are successfully treated for the underlying disorder with psychiatric interventions, behavior modification, and education, the encopresis is often resolved.

Prognosis

Since 80–95% of encopresis is related to constipation, the success rate in resolving involuntary encopresis is high, although it may take time to establish good bowel habits and eliminate a reoccurrence of constipation. The success rate is also good for younger children in a power struggle with adults over toileting, although the results may be slow. The prognosis for older children with associated behavioral disorders is less promising and depends more on the success of resolving those problems than on direct treatment of the symptoms of encopresis.

Prevention

Power struggles during toilet training that lead to encopresis can be reduced by waiting until the child is developmentally ready and interested in using the toilet. Toilet training undertaken kindly, calmly, and with realistic expectations is most likely to lead to success. Successes should be rewarded and failures accepted. Once toilet training has been established, encopresis can be reduced by developing regular bowel habits and encouraging a healthy, high-fiber diet.

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Endep *see* **Amitriptyline**

Energy therapies

Definition

Energy therapies is a collective term used to refer to a variety of alternative and complementary treatments based on the use, modification, or manipulation of energy fields. Most energy therapies presuppose or accept the theory that matter and energy are not exclusive opposites, but that matter is simply a denser form of energy that is more easily perceived by the senses. Some energy therapies are associated with systems of traditional Indian or Chinese medicine that are thousands of years old; others draw upon contemporary scientific theories. Energy therapies can be divided for purposes of discussion into two groups: those that utilize energy fields located in, affecting, or emanating from the human body (biofield therapies); and those that use electromagnetic fields in unconventional ways. In addition, there are energy therapies that combine biofield therapy with some aspects of bodywork— Breema, polarity therapy, and qigong are examples of this combined approach.

Energy therapies vary widely in their understanding of qualifications to be a healer. Some have credentialing or training programs; others do not. Some practitioners of energy therapy believe that all or most people have the capacity to be healers; others regard the ability to use or direct healing energies as a gift or charism that is given only to people who are "chosen" or unusually spiritual.

Although energy therapies are often associated with either Eastern or so-called "New Age" belief systems, most do not expect people in need of healing to give up mainstream Western religious practice or allopathic medical/psychiatric treatments.

KEY TERMS

Aura—An energy field that is thought to emanate from the human body and to be visible to people with special psychic or spiritual powers.

Ayurvedic medicine—The traditional medical system of India. Ayurvedic treatments include diet, exercises, herbal treatments, meditation, massage, breathing techniques, and exposure to sunlight.

Biofield therapies—A subgroup of energy therapies that make use of energy fields (biofields) thought to exist within or emanate from the human body. Biofield therapies include such approaches as Reiki, therapeutic touch, qigong, and polarity balancing.

Bodywork—Any technique involving hands-on massage or manipulation of the body.

Breema—An alternative therapy that originated in California in the 1980s. Breema combines biofield therapy with certain elements of chiropractic and bodywork.

Chakra—One of the seven major energy centers in the body, according to traditional Indian yoga.

Endorphins—A group of peptide compounds released by the body in response to stress or traumatic injury. Endorphins react with opiate receptors in the brain to reduce or relieve pain.

Kundalini—In Indian yoga, a vital force or energy at the base of the spine that is activated or released by certain yoga postures or breathing techniques. This release is called the "awakening" of the kundalini. Some Westerners have had kundalini experiences that were diagnosed as psychotic episodes or symptoms of schizophrenia.

Meridians—In traditional Chinese medicine, a network of pathways or channels that convey qi, or vital energy, through the body.

Polarity therapy—A form of energy therapy influenced by Ayurvedic medicine that integrates bodywork with diet, home exercises, and self-awareness techniques. It is sometimes called polarity balancing.

Prana—The Sanskrit word for vital energy, roughly equivalent to qi in traditional Chinese medicine.

Qi—The traditional Chinese term for vital energy or the life force. The word is also spelled "ki" or "chi" in English translations of Japanese and Chinese medical books.

Qigong—A traditional form of Chinese energy therapy that includes physical exercises, breathing techniques, postures, and mental discipline. Internal qigong refers to exercises practiced to maintain one's own health and vitality; external qigong refers to the transfer of energy from a qigong master to another person for healing purposes. External qigong is also known as medical qigong.

Reiki—A form of energy therapy that originated in Japan. Reiki practitioners hold their hands on or slightly above specific points on the patient's body in order to convey universal life energy to that area for healing.

Therapeutic touch (TT)—An American form of energy therapy based on the ancient tradition of the laying-on of hands. TT is thought to work by removing energy blockages or disturbances from the patient's aura.

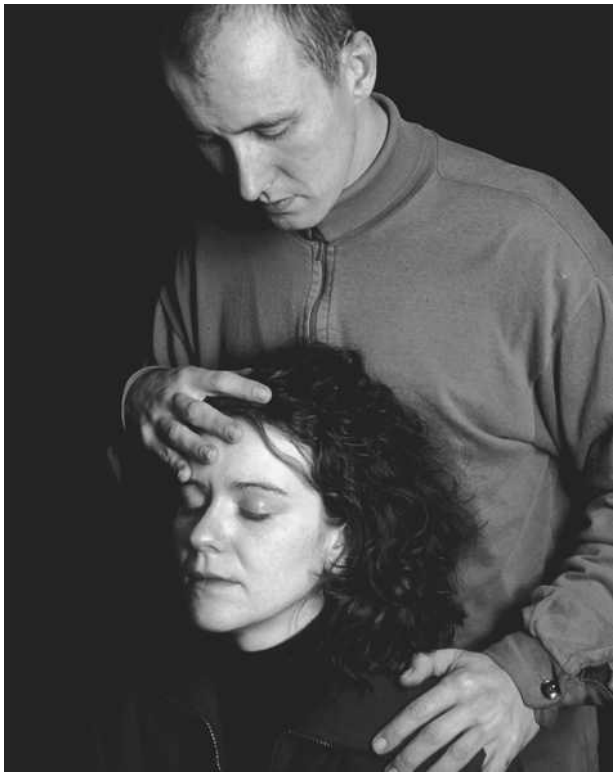
Purpose

The purpose of energy therapies can be broadly defined as the healing of mental or physical disorders by rebalancing the energy fields in the human body or by drawing upon spiritual energies or forces for such healing. Some energy therapies include internal **detoxification** or release of trauma-related memories as additional purposes.

Precautions

In general, persons who are interested in Breema, qigong, or any form of energy therapy that involves vigorous physical exercise or bodywork should seek the advice of a qualified medical practitioner before starting such a program. This precaution is particularly important for persons with chronic heart or lung disease, persons recovering from surgery or acute illness, or persons with arthritis or other disorders that affect the muscles and joints.

Some forms of energy therapy may produce unexpected or startling psychological reactions. For example, a type of psychospiritual energy referred to as Kundalini in Indian **yoga** sometimes produces experiences of spiritual crisis that may be interpreted by mainstream psychiatrists as symptoms of **schizophrenia** or another psychotic disorder. Practitioners of Reiki healing have reported instances of patients feeling tingling sensations, "spaciness," an "out of body" sensation, sudden warmth,



Woman undergoing therapeutic touch. In this form of healing, the therapist channels healing energy into the patient in order to cure the imbalances that cause physical or mental illness. *(Cordelia Molloy/ Science Photo Library. Photo Researchers, Inc. Reproduced by permission.)*

or similar experiences. As a rule, people in treatment for any mental condition or disorder should consult their therapist before beginning any form of energy treatment. This precaution is particularly important for patients diagnosed with **post-traumatic stress disorder** or a dissociative disorder, and for those who are easily hypnotized. It is also a good idea to find out as much as possible about the background and basic beliefs associated with a specific energy therapy, including the training or credentialing of its practitioners.

Description

Brief descriptions of some of the better known energy therapies follow.

Therapeutic touch

Therapeutic touch, or TT, is a form of energy therapy that developed in the United States. It is a noninvasive method of healing derived from an ancient laying-on of hands technique. In TT, practitioners alter the patient's energy field through a transfer of energy from their hands to the patient. Therapeutic touch was developed in 1972 by Dora Kunz, a psychic healer, and Dolores Krieger, a professor of nursing at New York University. The principle behind TT is restoration of balance or harmony to the human energy field, or aura, that is thought to extend several inches to several feet from the body. When illness occurs, it creates a disturbance or blockage in the vital energy field. The TT practitioner uses her/his hands to discern the blockage or disturbance. Although the technique is called "therapeutic touch," there is generally no touching of the client's physical body, only his or her energetic body or field. TT is usually performed on fully clothed patients who are either lying down on a flat surface or sitting up in a chair.

A therapeutic touch session consists of five steps or phases. The first step is a period of **meditation** on the practitioner's part, to become spiritually centered and energized for the task of healing. The second step is assessment or discernment of the energy imbalances in the patient's aura. In this step, the TT practitioner holds his or her hands about 2–3 inches above the patient's body and moves them in long, sweeping strokes from the patient's head downward to the feet. The practitioner may feel a sense of warmth, heaviness, tingling, or similar cues, as they are known in TT. The cues are thought to reveal the location of the energy disturbances or imbalances. In the third step, known as the unruffling process, the practitioner removes the energy disturbances with downward sweeping movements. In the fourth step, the practitioner serves as a channel for the transfer of universal energy to the patient. The fifth step consists of smoothing the patient's energy field and restoring a symmetrical pattern of energy flow. After the treatment, the patient rests for 10–15 minutes.

Although therapeutic touch has become a popular alternative or complementary approach in some schools of nursing in the United States and Canada, acceptance by the mainstream medical community varies. Many hospitals permit nurses and staff to perform TT on patients at no extra charge. On the other hand, however, therapeutic touch became national news in April 1998 when an elementary-school student carried out research for a science project that questioned its claims. Twenty-one TT practitioners with experience ranging from one to 27 years were blindfolded and asked to identify whether the investigator's hand was closer to their right hand or their left. Placement of the investigator's hand was determined by flipping a coin. The TT practitioners were able to identify the correct hand in only 123 (44%) of 280 trials, a figure that could result from random chance alone. Debate about the merits of TT filled the editorial pages of the *Journal of the American Medical Association* for nearly a year after the news reports, and continues to this day.

Qigong

Qigong is a form of Chinese energy therapy that is usually considered a martial art by most Westerners. It is better understood, however, as an ancient Chinese system of postures, exercises, breathing techniques and meditations. Its techniques are designed to improve and enhance the body's *qi*. According to traditional Chinese philosophy and medicine, qi is the fundamental life energy responsible for human health and vitality. Qi travels through the body along channels called meridians. There are twelve main meridians in humans. Each major body organ has qi associated with it, and each organ interacts with particular emotions on the mental level. Qigong techniques are designed to improve the balance and flow of energy throughout the meridians, and to increase the overall quantity and volume of a person's qi.

In the context of energy therapy, qigong is sometimes divided into internal and external qigong. Internal qigong refers to a person's practice of qigong exercises to maintain his or her own health and vitality. Some qigong master teachers are renowned for their skills in external qigong, in which the energy from one person is passed on to another for healing. Chinese hospitals use medical qigong along with herbs, **acupuncture** and other techniques of traditional Chinese medicine. In these hospitals, qigong healers use external qigong and also design specific internal qigong exercises for the patients' health problems.

Reiki

Reiki is a holistic alternative therapy based on Eastern concepts of energy flow and the seven chakras (energy centers) in the human body. Reiki was formulated by a Japanese teacher, Mikao Usui, around 1890, based on Vajrayana (Tibetan) Buddhism, but incorporates meditation techniques, beliefs, and symbols that are considerably older. It is distinctive among energy therapies in its emphasis on self-healing, its spiritual principles, and its accreditation of healers through a system of initiation. Reiki practitioners participate in the healing of emotional and spiritual as well as physical pain through the transmission of universal life energy, called "rei-ki" in Japanese. It is believed that ki flows throughout the universe, but that Reiki connects humans in a more direct way to the universal source. Reiki is used for the healing of animals as well as people. As of 2002, a research team at the University of Michigan is studying the effectiveness of Reiki in treating chronic pain in patients with diabetic neuropathy. Various other studies are also underway in the United States and Canada, some examining the efficacy of the therapy in coping with pain and anxiety.

Although Reiki involves human touch, it is not massage therapy. The patient lies on a table fully clothed except for shoes while the practitioner places her or his hands over the parts of the body and the chakras in sequence. The hands are held palms downward with the fingers and thumbs extended. If the person is in pain or cannot turn over, the practitioner may touch only the affected part(s). Silence or music appropriate for meditation is considered essential to the treatment. Reiki healers practice daily self-healing, in which they place their hands in traditional positions on their own bodies. They may use touch, or distant/non-touch.

Reiki healers are initiated into three levels of practice through attunements, which are ceremonies in which teachers transmit the hand positions and "sacred" symbols. Reiki I healers learn the basic hand positions and can practice direct physical, emotional or mental healing on themselves and others. Reiki II healers are taught the symbols that empower them to do distance or absentee healing. In Reiki III the healer makes a commitment to become a master teacher and do spiritual healing.

Polarity therapy

Polarity therapy, which is sometimes called polarity balancing, is a biofield therapy that resembles Reiki in its emphasis on energy flow, human touch, and the energy centers (chakras) in the human body. Polarity therapy was developed by Dr. Randolph Stone (1890-1981), an American chiropractor and naturopath. It integrates bodywork with diet, yoga-based exercise, and self-awareness techniques to release energy blockages in the patient's body, mind, or feelings. Polarity theory divides the body into three horizontal and four vertical zones (right, left, front, and back), each having a positive, negative, or neutral charge. Energy currents in the zones are correlated with five energy centers in the body corresponding to the five elements (ether, air, fire, water, and earth) of Ayurvedic medicine.

Polarity therapy can be done one-on-one or with a group of practitioners working on the patient. The therapist as well as the patient removes shoes. The patient lies fully dressed except for shoes on a massage table or bed, or on the floor. The practitioner takes the patient's history, checks reflexes and touches body parts to determine energy blocks. Polarity therapy uses three levels of touch: no touch (hands held above the body, touching only the energy fields); light touch; and a deep, massaging touch. The therapist balances energy currents in the patient's body by placing his or her "plus" hand on "negative" body parts and vice versa. Polarity therapy involves rocking the patient's body and holding the head as well as more usual massage techniques. It takes about four polarity sessions

In this Reiki treatment, the therapist is channelling energy into the head of the woman being treated. *(Roger Ressmeyer/Corbis. Reproduced by permission.)*

to treat most conditions, with each session lasting about an hour. After a course of treatment, the polarity practitioner usually suggests drinking plenty of liquids for one to two weeks together with other dietary changes as part of a general internal cleansing or detoxification program. Polarity yoga (stretching exercises) is prescribed for the patient's regular workouts at home.

Breema

Breema is a form of body movement energy therapy that combines elements of bodywork, yoga, chiropractic, and New Age philosophy. Breema began in California in 1980. Its founder is Dr. Jon Schreiber, a graduate of Palmer College of Chiropractic. The Breema Health and Wellness Center was opened in Oakland, California, in 1981. The principles of Breema are intended to free people from the conceptual body, defined as "the ideas and images of our body that we carry in our mind." The aim of Breema "is to increase vitality, not to fight sickness, and to create an atmosphere which allows the body to move toward a natural state of balance." A person receiving a Breema treatment works with an instructor or practitioner through a series of individualized exercises on a padded floor. The instructors and practitioners are certified by the Breema Center in Oakland.

Decrystallization is an important part of Breema therapy. According to Breema, decrystallization is a process in which the body is helped to release deeply held, or "crystallized," patterns of chronic discomfort, tension, or emotional pain. As the body releases its crystallizations, its "core energetic patterns" are balanced and realigned. A decrystallization program consists of one or more Breema treatments per week for a year. It includes a set of personalized self-Breema exercises.

Electromagnetic therapies

Electromagnetic therapies cover a variety of treatments that use a source of physical energy outside the body— most often magnets or electromagnetic field stimulation— to treat a range of musculoskeletal disorders. Some forms of magnetic therapy, such as bracelets, gloves, shoe inserts, and similar items containing small magnets meant to be worn near the affected body part, can be self-administered. This form of magnetic therapy

has become quite popular among professional athletes and "weekend warriors" to relieve soreness in joints and muscles from over exercise. At present, there are two hypothetical explanations of the effectiveness of magnetic therapy. One theory maintains that the magnets stimulate nerve endings in the skin surface to release endorphins, which are pain-relieving chemicals produced by the body in response to **stress** or injury. According to the second hypothesis, the magnets attract certain ions (electrically charged molecules) in the blood, which serves to increase the blood flow in that area of the body. The increased blood flow then relieves the tissue swelling and other side effects of over exercise that cause pain.

Other forms of electromagnetic therapy require special equipment and cannot be self-administered. These forms of treatment are most commonly used by naturopathic practitioners. One form, called transcranial magnetic stimulation, is used in the treatment of depression. Another form, called pulsed electromagnetic field stimulation, has been shown to be effective in the treatment of osteoarthritis.

Preparation

Most forms of energy therapy require little preparation on the patient's part except for the wearing of loose and comfortable clothes. Patients are asked to remove jewelry before a polarity balancing treatment and to remove eyeglasses and shoes prior to Reiki treatment. Qigong should not be practiced on either a full or a completely empty stomach.

Aftercare

Aftercare for therapeutic touch and Reiki usually involves a few moments of quiet rest to maximize the benefits of treatment. Aftercare for polarity therapy includes increased fluid intake for one to two weeks and other dietary adjustments that may be recommended by the practitioner.

Risks

There are no known risks associated with therapeutic touch, or polarity balancing. Using Reiki, precautions should be taken clients diagnosed with schizophrenia, **psychosis**, dissociative disorder, manic/depressive (bipolar) or borderline personality. The risk of physical injury from the exercises involved in Breema or qigong are minimal for patients who have consulted their primary physician beforehand and are working with a qualified instructor.

Mild headache has been reported as a side effect of transcranial magnetic stimulation. No side effects have been associated with self-administered magnetic therapy.

Normal results

Normal results for energy therapies include increased physical vitality, lowered blood pressure, a sense of calm or relaxation, improved sleep at night, and a strengthened immune system. Some persons report pain relief and speeded-up healing of wounds from magnetic therapy, Reiki, and qigong.

Abnormal results

Abnormal results from energy therapies include physical injury, severe headache, dizziness, depressed mood, or increased anxiety.

See also Bodywork therapies; Light therapy

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American Association of Naturopathic Physicians. 601 Valley Street, Suite 105, Seattle, WA 98109. (206) 298-0126. <www.naturopathic.org>.

American Polarity Therapy Association. 288 Bluff Street #149, Boulder, CO 80301. (303) 545-2080. <www.livelinks.com/sumeria/health/polarity.html>.

The Breema Center. 6076 Claremont Avenue. Oakland, CA 94618. (510) 428-0937. Fax (510) 428-9235. <www.breema.com>.

International Society for the Study of Subtle Energies and Energy Medicine (ISSSEEM). 356 Goldco Circle, Golden, CO 80401. (303) 278-2228. <www.vitalenergy.com/ISSSEEM>.

National Center for Complementary and Alternative Medicine (NCCAM) Clearinghouse. P.O. Box 7923, Gaithersburg, MD 20898. (888) 644-6226. TTY: (866) 464-3615. Fax: (866) 464-3616. <www.nccam.nih.gov>.

The Nurse Healers Professional Associates International (NHPAI), the Official Organization of Therapeutic Touch. 3760 S. Highland Drive, Salt Lake City, UT 84106. (801) 273-3399. <www.therapeutic-touch.org>.

Qigong Human Life Research Foundation. PO Box 5327. Cleveland, OH 44101. (216) 475-4712.

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The Kundalini Clinic. 3040 Richmond Boulevard, Oakland, CA, 94611. (510) 465-2986.

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Enuresis

Definition

Enuresis, more commonly called bed-wetting, is a disorder of elimination that involves the voluntary or involuntary release of urine into bedding, clothing, or other inappropriate places. In adults, loss of bladder control is often referred to as urinary incontinence rather than enuresis; it is frequently found in patients with late-stage Alzheimer's disease or other forms of **dementia**.

Description

Enuresis is a condition that has been described since 1500 B.C. People with enuresis wet their bed or release urine at other inappropriate times. Release of urine at night (nocturnal enuresis) is much more common than daytime, or diurnal, wetting. Enuresis commonly affects young children and is involuntary. Many cases of enuresis clear up by themselves as the child matures, although some children need behavioral or physiological treatment in order to remain dry.

There are two main types of enuresis in children. Primary enuresis occurs when a child has never established bladder control. Secondary enuresis occurs when a person has established bladder control for a period of six months, then relapses and begins wetting. To be diagnosed with enuresis, a person must be at least five years old or have reached a developmental age of five years. Below this age, problems with bladder control are considered normal.

Causes and symptoms

Symptoms

The symptoms of enuresis are straightforward—a person urinates in inappropriate places or at inappropriate times. The causes of enuresis are not so clear. A small number of children have abnormalities in the anatomical structure of their kidney or bladder that interfere with bladder control, but normally the cause is not the physical structure of the urinary system. A few children appear to have to have a lower-than-normal ability to concentrate urine, due to low levels of antidiuretic hormone (ADH). This hormone helps to regulate fluid balance in the body. Large amounts of dilute urine cause the bladder to overflow at night. For the majority of bedwetters, there is no single clear physical or psychological explanation for enuresis.

Causes in children

The fourth edition of the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision, or (*DSM-IV-TR*), does not distinguish between children who wet the bed involuntarily and those who voluntarily release urine. Increasingly, however, research findings suggest that voluntary and involuntary enuresis have different causes.

Involuntary enuresis is much more common than voluntary enuresis. Involuntary enuresis may be categorized as either primary or secondary. Primary enuresis occurs when young children lack bladder control from infancy. Most of these children have urine control problems only during sleep; they do not consciously, intentionally, or maliciously wet the bed. Research suggests that children who are nighttime-only bed wetters may have a nervous system that is slow to process the feeling of a full bladder. Consequently, these children do not wake up in time to relieve themselves. In other cases, the child's enuresis may be related to a sleep disorder.

Children with diurnal enuresis wet only during the day. There appear to be two types of daytime wetters. One group seems to have difficulty controlling the urge to urinate. The other group consciously delays urinating until they lose control. Some children have both diurnal and nocturnal enuresis.

Secondary enuresis occurs when a child has stayed dry day and night for at least six months, then returns to wetting. Secondary enuresis usually occurs at night. Many studies have been done to determine if there is a psychological component to enuresis. Researchers have found that secondary enuresis is more likely to occur after a child has experienced a stressful life event such as the birth of a sibling, divorce or death of a parent, or moving to a new house.

Several studies have investigated the association of primary enuresis and psychiatric or behavior problems. The results suggest that primary nocturnal enuresis is *not* caused by psychological disorders. Bed-wetting runs in families, however, and there is strong evidence of a genetic component to involuntary enuresis.

Unlike involuntary enuresis, voluntary enuresis is not common. It is associated with such psychiatric disorders as **oppositional defiant disorder**, and is substantially different from ordinary nighttime bed-wetting. Voluntary enuresis is always secondary.

Causes in adults

Enuresis or urinary incontinence in elderly adults may be caused by loss of independent control of body functions resulting from dementia, bladder infections, uncontrolled diabetes, side effects of medications, and weakened bladder muscles. Urinary incontinence in adults is managed by treatment of the underlying medical condition, if one is present; or by the use of adult briefs with disposable liners.

KEY TERMS

Bladder—A muscular sac in the lower abdomen that holds urine until it is discharged from the body.

Elimination—The medical term for expelling waste from the body.

Enuresis—The inability to control urination; bed-wetting.

Primary enuresis—Bed-wetting in a child who has not yet developed bladder control.

Secondary enuresis—Bed-wetting in a child who has established bladder control but has begun to wet the bed again, usually as the result of emotional stress.

Urinary incontinence—A term that is sometimes used for enuresis in adults. Urinary incontinence is often found in patients with late-stage Alzheimer's disease or other adult-onset dementias.

Urinary system—The kidney, urethra, bladder, and associated organs that process urine and eliminate it from the body.

Demographics

Enuresis is a problem of the young and is more common in boys than girls. At age five, about 7% of boys and 3% of girls have enuresis. This number declines steadily in older children; by age 18, only about 1% of adolescents experience enuresis. Studies done in several countries suggest that there is no apparent cultural influence on the incidence of enuresis in children. On the other hand, the disorder does appear to run in families; children with one parent who wet the bed as a child are five to seven times more likely to have enuresis than children whose parents did not have the disorder in childhood.

Diagnosis

Enuresis is most often diagnosed in children because the parents express concern to the child's doctor. The pediatrician or family physician will give the child a physical examination to rule out medical conditions that may be causing the problem, including structural abnormalities in the child's urinary tract. The doctor may also rule out a sleep disorder as a possible cause. In many cases the pediatrician can reassure the child's parents and give them helpful advice.

According to the American Psychiatric Association, to make a **diagnosis** of enuresis, a child must have reached the chronological or developmental age of five. Inappropriate urination must occur at least twice a week for three months; or the frequency of inappropriate urination must cause significant distress and interfere with the child's school and/or social life. Finally, the behavior cannot be caused exclusively by a medical condition or as a side effect of medication.

Treatments

Treatment for enuresis is not always necessary. About 15% of children who have enuresis outgrow it each year after age six. When treatment is desired, a physician will rule out obvious physical causes of enuresis through a physical examination and medical history. Several different treatment options are then available.

Behavior modification

Behavior modification is often the treatment of choice for enuresis. It is inexpensive and has a success rate of about 75%. The child's bedding includes a special pad with a sensor that rings a bell when the pad becomes wet. The bell wakes the child, who then gets up and goes to the bathroom to finish emptying his bladder. Over time, the child becomes conditioned to waking up when the bladder feels full.

Once this response is learned, some children continue to wake themselves help from without the alarm, while others are able to sleep all night and remain dry. A less expensive behavioral technique involves setting an alarm clock to wake the child every night after a few hours of sleep, until the child learns to wake up spontaneously. In trials, this method was as effective as the pad-and-alarm system. A newer technique involves an ultrasound monitor worn on the child's pajamas. The monitor can sense bladder size, and sets off an alarm once the bladder reaches a predetermined level of fullness. This technique avoids having to change wet bed pads.

Other behavior modifications that can be used alone or with the pad-and-alarm system include:

- restricting liquids starting several hours before bedtime
- waking the child up in the night to use the bathroom
- teaching urinary retention techniques
- giving the child positive **reinforcement** for dry nights and being sympathetic and understanding about wet nights

Treatment with medications

There are two main drugs for treating enuresis. **Imipramine**, a tricyclic antidepressant, has been used since the early 1960s. It is not clear why this antidepressant is effective in treating enuresis when other antidepressants are not. Desmopressin acetate (DDAVP) has been widely used to treat enuresis since the 1990s. It is available as a nasal spray or tablet. Both imipramine and DDAVP are very effective in preventing bed-wetting, but have high relapse rates if medication is stopped.

Alternative therapies

Some success in treating bed-wetting has been reported using hypnosis. When hypnosis works, the results are seen within four to six sessions. **Acupuncture** and massage have also been used to treat enuresis, with inconclusive results.

Psychotherapy

Primary enuresis does not require **psychotherapy**. Secondary enuresis, however, is often successfully treated with therapy. The goal of the treatment is to resolve the underlying stressful event that has caused a relapse into bed-wetting. Unlike children with involuntary enuresis, children who intentionally urinate in inappropriate places often have other serious psychiatric disorders. Enuresis is usually a symptom of another disorder. Therapy to treat the underlying disorder is essential to resolving the enuresis.

Prognosis

Enuresis is a disorder that most children outgrow. For those who do receive treatment, the overall success rate of behavioral therapy is 75%. The short-term success rate with drug treatments is even higher than with behavioral therapy. Drugs do not, however, eliminate the enuresis. Many children who take drugs to control their bed-wetting relapse when the drugs are stopped.

Prevention

Although enuresis cannot be prevented, one side effect of the disorder is the shame and social embarrassment it causes. Children who wet may avoid sleepovers, camp, and other activities where their bed-wetting will become obvious. Loss of these opportunities can cause a loss of self-esteem, social isolation, and adjustment problems. A kind, low-key approach to enuresis helps to prevent these problems.

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Tish Davidson, A.M.

Erectile dysfunction

Definition

Erectile dysfunction (ED) may be defined as the consistent inability to achieve or maintain an erection sufficient to permit satisfactory sexual intercourse. The word "consistent" is included in the definition because most men experience transient episodes of ED that are temporary and usually associated with **fatigue**, anger, depression or other stressful emotions. The use of the formerly used term "impotence" has been virtually abandoned because of its inherent **stigma** of weakness and lack of power.

Erectile dysfunction can occur as part of several mental disorders recognized by the mental health professional's manual, the *Diagnostic and Statistical Manual of Mental Disorders,* often shortened to the *DSM.* ED is the main symptom in the disorder the manual calls "male erectile disorder." ED can also be a symptom of other disorders, such as sexual dysfunction due to a general medical condition or substance-induced sexual dysfunction. In this entry, however, ED is examined and discussed as its own medical entity, and not within the strict guidelines of the *DSM*.

Description

First, it may be useful to understand the mechanisms of normal penile erection. Penile erection occurs essentially when the penis becomes engorged with blood. The anatomical compartments (two corpora cavernosa and one corpus spongiosum) are capable of being distended with seven times their normal amount of blood. When this occurs in association with relaxation of the penile muscles, erection results.

The sequence of events resulting in penile erection is complex. It is usually initiated by sexual arousal stimuli arising in the **brain** as a result of visual, auditory or olfactory sensations or erotic thoughts. Tactile (touch) sensations of the penis acting through the spinal cord play a similar role. Sexual arousal results in the release of a chemical (nitric oxide) from specialized cells. Nitric oxide causes the formation of a substance (cyclic glutamine monophosphate or cGMP) that is responsible for dilating the blood vessels of the penis and relaxing its muscles, thus allowing for an increase in blood flow and resultant penile erection. Compression of the dilated blood vessels against the firm outer lining of the penis prevents the blood from escaping and perpetuates the erection. A specialized substance (phosphodiesterase 5 or PDE-5), causes the breakdown of cGMP and, with the help of nerves from the sympathetic nervous system, allows the penis to return to its flaccid relaxed state.

Any defect in this complex cascade of events can result in erectile dysfunction.

Different men experience varying patterns of ED. Men with ED may report the inability to experience any erection from the beginning of a sexual experience, while others experience an erection that is not maintained at penetration. Other men may lose the erection during sexual intercourse, and others can only experience erection upon awakening or during self-masturbation.

KEY TERMS

Diabetes mellitus—A chronic disease affecting the metabolism of carbohydrates that is caused by insufficient production of insulin in the body.

Hypertension—High blood pressure, often brought on by smoking, obesity, or other causes; one of the major causes of strokes.

Hypogonadism—Abnormally decreased gonad function with retardation of sexual development.

Priapism—Persistent abnormal erection of the penis, usually without sexual desire, and accompanied by pain and tenderness.

Prostate gland—The gland at the base of a male's urethra that produces a component of semen.

Impact of ED

It is well-recognized that adults of all ages view sex as an important quality-of-life issue, and that the imposition of ED usually results in a reduced quality of life. In spite of this and for a number of reasons— most of them unfounded— the victims often suffer in silence. Included among the reasons for their silence are the following conceptions:

- ignorance of the availability of safe and effective therapy for ED
- inadequate information provided by the physician concerning timing of medication, need for preliminary sexual arousal, etc.
- undue concern about the irreversibility of marital discord and lack of partner support
- concerns about administration of invasive therapies, adverse effects of therapy, discomfort, inconvenience and cost of therapy
- high rates of discontinuation of therapy due to inadequacy of therapeutic response and associated adverse effects.

Causes and symptoms

Causes

A precise determination of the cause of any individual case of ED is often difficult and may be impossible because ED is often due to multiple factors. This is a consequence of the complicated nature of the human sexual response and the complex physiology of penile erection and relaxation. Normal erectile function requires the coordination of vascular, neurologic, hormonal and psychological factors and any condition that interferes with one or more of these processes may result in ED.

Attitudes concerning age and psychological factors, commonly associated with ED in the past, have changed in the last two decades. Although the prevalence of ED increases with advancing age, ED is no longer regarded as an inevitable consequence of aging. Whereas most cases of ED were once considered primarily psychological and/or psychiatric in origin, it is now well-recognized that organic, non-psychological causes of ED play a much more significant role in the development of ED. Most researchers agree that pure psychological (emotional) mechanisms are causative in 15% to 20% of cases with organic causes responsible for at least 80% of ED cases. In a number of cases, the situation is "mixed," with significant secondary psychological and social components such as guilt, depression, anxiety, tension or marital discord being present in addition to one or more underlying organic components.

Causes of ED may be grouped into those factors that arise within the individual (endogenous) and those factors arising from sources outside the body (exogenous). Endogenous factors include endocrine imbalances, cardiovascular and other medical conditions, and emotional causes. Included among exogenous factors are medications, surgery, trauma and irradiation, smoking, and alcohol and substance abuse. Many of these causes are discussed in more detail in the following list of causes:

- Diabetes mellitus. This is the single most common cause of ED by virtue of its combined nerve and blood vessel damage. At least 40% of male diabetics have ED.
- Circulation abnormalities. Vascular (circulation-related) causes include diseases of the aorta or the arteries supplying the pelvis and penis. Hardening of the arteries (arteriosclerosis) is the most common vascular cause, but damage to the arteries may result from trauma, surgery, or irradiation. Surgery involving the prostate gland may involve both the arteries and nerves in that region.
- Neurological causes, including diseases of the brain (such as **Alzheimer's disease**) and spinal cord (multiple sclerosis, for example).
- Hormonal or endocrine causes. These are uncommon causes for ED, however. ED may occur in males with deficient testicular function and low circulating levels of the male sex hormone, testosterone. These cases are referred to as hypogonadism and may be due to congenital abnormalities or testicular disease such as that accompanying mumps.

- Penile diseases: Organic causes of ED may be related to diseases of the penis. Many factors influence penile circulation. For instance, Peyronie's disease, a condition characterized by fibrous tissue and a downward bowing of the penis, limits the expandability of the penile tissues, thus preventing venous compression and allowing blood to leave the penis. Similarly, arteriosclerotic plaque, injury to blood vessels' inner lining due to trauma, surgery, or irradiation, or even aortic occlusion (blockage in a main artery leading out of the heart) can be the cause of compromised penile blood flow and prevent penile erection.
- Medications: A number of classes of medications can cause ED. Not all agents within each drug class produce the same effects. For example, some antidepressants are associated with ED, whereas an antidepressant called **trazodone** hydrochloride (Desyrel) has been used in institutional studies for the treatment of ED because of its tendency to produce priapism. Some medication classes that can cause ED include (but are not limited to): medications that reduce high blood pressure, medications taken for central nervous system diseases like Parkinson's disease (methyldopa), antidepressants, sedatives or tranquilizers like **barbiturates**, anti-anxiety medications like **diazepam** (Valium), common, non-prescribed drugs such as tobacco and alcohol, and drugs of abuse including heroin.
- Psychological factors that can precipitate ED include **stress**, fatigue, depression, guilt, low self-esteem and negative feelings for or by a sexual partner. Depressive symptoms and/or difficulty coping with anger may be particularly influential, and ED may be related to a "submissive personality."
- Lifestyle: **Obesity**, physical inactivity, cigarette smoking, and excessive intake of alcohol are risk factors for the development of ED. These suggest that changes in lifestyle may constitute an important aspect of both the therapy and prevention of ED.

The identification of risk factors for ED has an important impact not only on the treatment, but on the prevention of ED as well. For example, if a doctor is treating a patient for high blood pressure who is also at risk for ED, the doctor may make an informed decision to prescribe an effective medication that is not associated with ED instead of one that is.

ED AS A MARKER FOR OTHER DISEASES. The frequent association between ED and a number of important vascular conditions such as hypertension and coronary artery heart disease has raised the possibility that ED may serve as an important marker for the detection of these vascular disorders. Additionally, an increased incidence of depression has been noted in men with ED that is believed to be distinct from the reactive type of depression that might occur because of ED. This has led to the recognition of a possible syndrome linking depression and ED. Thus, the presence of depression should be investigated in men presenting with ED.

Symptoms

The main symptom is the inability to attain or maintain adequate erection to complete sexual activity.

As a result of this symptom, affected men may also experience depression and distress, and this symptom can cause interpersonal (including marital) issues.

Demographics

Studies indicate that in the United States at least 30 million American men suffer from some degree of erectile dysfunction (ED). Of these, 10 to 20 million have a severe degree of ED resulting in the complete inability to attain or maintain a penile erection. The number of ED victims in the U.S. is projected to increase by nearly 10 million by the year 2025. With the advancement of men's median age in western industrial countries and the general population growth in developing nations, the worldwide incidence is projected to increase to greater than 320 million by 2025. ED accounts for more than 500,000 annual visits to health care professionals.

As with other chronic disorders and the conditions that are commonly associated with ED (diabetes, hypertension, cardiovascular disease), the prevalence of ED increases with advancing age, with an estimated prevalence of 39% in men aged 40 and 67% in those aged 70. These figures may actually underestimate the true dimensions of the problem since ED is notoriously underreported, undiagnosed and under-treated because of the perceived stigma associated with the **diagnosis** of ED. It is reported that 70% of ED remains undiagnosed and in a survey of general medical practice less than 12% of men with ED reported having received treatment for it.

Diagnosis

Interview

An essential first step in the diagnosis of ED is the taking of a thorough sexual, medical, and psychosocial (both psychological and social) history. The sexual history should include information such as the frequency of sexual intercourse, its duration, the quality and degree of penile erection, the presence or absence of nocturnal erections, and the success or failure of penetration. Any sexual dysfunction on the part of the partner, such as painful intercourse (**dyspareunia**) or vaginal dryness,

should be ascertained. The use of one of several available self-directed patient questionnaires may be a useful adjunct to the sexual history. The sexual history helps in distinguishing ED from other abnormalities in sexual function such as ejaculatory and orgasmic disturbances and loss of sexual desire.

The general medical history may disclose one or more distinct causes of ED including the presence of associated conditions, the use of medications that can cause the disorder, and/or a history of substance abuse.

A psychosocial history, preferably with the participation of the patient's sexual partner, should include current sexual practices, the presence or absence of stress and performance anxiety, and any special circumstances under which ED occurs.

Physical examination

For a patient with ED, the physical examination should not differ substantially from that performed routinely by a primary care physician. The doctor looks for evidence of hypogonadism or congenital conditions in which there is defective testicular function. The examination of the genitourinary, circulatory and neurologic systems might be especially emphasized. The patient's genitalia are carefully examined for testicular size and consistency and penile deformities. A rectal examination is needed to evaluate the size and consistency of the prostate gland and for the performance of certain muscular reflexes. Vital signs such as blood pressure and pulse would be recorded. Because the presence of ED may serve as a marker for high blood cholesterol, hypertension, coronary artery heart disease, and depression, the physician may also request blood work and/or may perform other assessments to check for these conditions.

Other diagnostic methods that may be performed

Laboratory tests may be performed to evaluate levels of hormones including testosterone and prolactin.

Nocturnal studies present a true picture of erectile dysfunction due to organic causes. The most complete evaluation of nocturnal erectile function is obtained in a sleep laboratory, where patients are monitored for rapid eye movement (REM) sleep.

Duplex Doppler ultrasonography has been used extensively in the evaluation of erectile function. It provides information about both arterial and venous blood flow.

Pharmacological testing involves intracavernosal injection of a small amount of an active agent (such as 10 micrograms of alprostadil [prostaglandin E1]) that would produce a normal or priapic erection in a patient with normal erectile function but a poor response in a patient with erectile dysfunction.

There are several patient self-administered questionnaires available to assist in the evaluation of sexual function in men with erectile dysfunction. The best known and most widely used is the International Index of Erectile Function (IIEF). The IIEF addresses the five relevant domains of male sexual function: erectile function, orgasmic function, sexual desire, intercourse satisfaction, and overall satisfaction.

Treatment

The first step in the treatment of ED includes the elimination or alteration of modifiable risk factors or causes, such as lifestyle or psychosocial factors including smoking, obesity, substance and alcohol abuse, and the alteration of prescription and over-the-counter medications if necessary.

Recommended treatment options for ED include the following medications:

- Oral erectogenic medications
- PDE-5 inhibitors. Sildenafil (Viagra) is an example. It works by blocking PDE-5 thereby allowing cGMP to have a longer effect, increasing penile blood flow and producing erection.
- Yohimbine
- Apomorphine
- Alpha adrenergic blockers

Vacuum constriction device therapy, which involves a mechanical device to increase penile blood flow and erection may also be recommended. Psychosexual therapy is also recommended so that any psychological causes for ED can be detected and therapy can be instituted. Individual **psychotherapy** or **couples therapy** may be helpful. These various treatment methods can be used alone or in combination.

If those therapies are unsuccessful, the following treatment options may be recommended:

- Intracavernous therapy (ICIT). This therapy involves injection of the penile structures with substances that promote blood flow and produce erection.
- Intraurethral therapy. Medications are inserted into the urethra and act to increase blood flow and muscle relaxation, allowing for erection.
- Penile prostheses. These are various devices inserted surgically into the penis to produce the erect state.
- Surgery. In rare cases, surgery may be used to correct a defect that interferes with penile erection.

Regardless of the therapy chosen, follow-up at regular intervals and good communication between the patient and the doctor is essential. Patients need to keep their doctors informed about adverse reactions, and patients need to be informed about drug interactions. The doctor may adjust the dosage of medication, or may substitute or add a therapeutic agent into the treatment, as necessary.

The patient and his sexual partner can work with their treatment team so that they are both well-informed about various treatment options and can maximize treatment results.

Prognosis

The combination of the increased understanding of ED, an improved approach to the problem and the development of newer and more effective therapies has resulted in a marked improvement in the prognosis of ED. It is estimated that at least 65% of all cases of ED currently have a satisfactory therapeutic outcome. However, several factors affect individual prognostic forecasts. Risk factors that cannot be changed and that have a negative effect on individual prognoses include: increasing age, the presence of comorbid (co-occurring) conditions such as diabetes, and pelvic surgery in which the nerves were not spared. In contrast, potentially modifiable risk factors such as physical inactivity, smoking, excessive alcoholic intake, certain medications, and obesity improve prognosis when treated effectively.

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Ralph Myerson, M.D.

Eskalith *see* **Lithium carbonate**

Estazolam

Definition

Estazolam is a sedative-hypnotic drug belonging to the class of drugs known as benzodiazepines. It is sold in the United States under the names ProSom and Sedarest.

Purpose

Estazolam is used as a short-term treatment for **insomnia**. Given at bedtime, estazolam can help patients who have trouble falling asleep, staying asleep, or who have unwanted early morning awakening.

Description

Estazolam belongs to a group of drugs called benzodiazepines. Benzodiazepines are sedative-hypnotic drugs that help to relieve nervousness, tension, and other anxiety symptoms by slowing the central nervous system. To do this, they block the effects of a specific chemical involved in the transmission of nerve impulses in the **brain**, decreasing the excitement level of the nerve cells.

Estazolam, like other benzodiazepines, can be habit-forming and can cause tolerance. Tolerance occurs when a given dosage has less and less effect when the drug is taken over a long time. Therefore, estazolam is recommended only for short-term use.

Estazolam is available in 1- and 2-mg tablets, for oral use.

Recommended dosage

Adults are usually prescribed a single 1–2 mg dose of estazolam to be taken at bedtime. The elderly (over

KEY TERMS

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Delusion—A false belief that is resistant to reason or contrary to actual fact.

Depressant—Something that slows down functioning.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Hallucinations—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Sleep apnea—Short periods where a person stops breathing during sleep. Breathing re-starts spontaneously, however, this condition can lead a lack of oxygen in the body.

age 60) or people with serious health problems require much smaller doses, and are usually started at 0.5 mg at bedtime.

Precautions

Care must be taken when prescribing this medication to anyone with decreased liver or kidney functioning, the elderly, those with a history of substance abuse, depression, respiratory depression (such as asthma, chronic obstructive pulmonary disease, chronic bronchitis, or other chronic respiratory diseases), narrow-angle glaucoma or known sleep apnea. People with these health conditions should discuss the risks and benefits of using estazolam with their doctor before starting treatment.

Pregnant women should not use estazolam, because it causes damage to the developing fetus. Because estazolam shows up in breast milk, women who are breastfeeding a baby should not take this drug.

Because estazolam is a nervous system and respiratory depressant, it should not be taken with other such depressants, such as alcohol or other sedatives, sleeping pills, or tranquilizers. Furthermore, patients should not drive, operate dangerous machinery, or engage in hazardous activities until the drug's effects have worn off.

Suddenly discontinuing estazolam after several weeks of use may cause uncomfortable symptoms of withdrawal. Patients should discuss with their doctor how to discontinue estazolam gradually use to avoid such symptoms.

Side effects

The most common side effects of estazolam include sleepiness, slowness of movement, dizziness, and difficulty with coordination.

Less common side effects include anxiety, confusion, depression, memory loss for events occurring after the drug is taken, increased heart rate, and pounding or irregular heartbeat.

Rare side effects include confused thinking, disorientation, **delusions**, irritability, agitation, **hallucinations**, **seizures**, bizarre and/or aggressive behavior, a drop in blood pressure, weak muscles, skin rash or itching, sores in mouth or throat, fever and chills, difficulty sleeping, odd body and/or eye movements, unusual bruising or easy bleeding, severe **fatigue** or weakness, and yellow eyes or skin (jaundice).

Interactions

Cimetidine (Tagamet), **disulfiram** (Antabuse), and erythromycin (an antibiotic) may increase estazolm's sedative effects.

Rifampin may decrease the effects of estazolam.

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Rosalyn Carson-DeWitt, M.D.

Etiology of mental illness *see* **Origin of mental illnesses**

Evening primrose oil

Definition

Evening primrose oil is a dietary supplement derived from the seeds of the evening primrose plant, *Oenothera biennis*. Its Latin name is derived from the Greek word for wine, reflecting the folk belief that the plant could

relieve the symptoms of a hangover. Other names for the plant are tree primrose and sundrop. Native Americans used the leaves and bark of evening primrose as a sedative and astringent; it was given for stomach and liver complaints as well as disorders of the female reproductive system. More recently, the discovery of antioxidant and other properties of the seed oil has focused attention on its usefulness in treating a range of diseases and disorders, including as an anti-inflammatory, and for premenstrual syndrome (PMS), rheumatoid arthritis, diabetes, osteoporosis, ulcerative colitis, menopausal problems, and heart disease.

Purpose

Evening primrose oil is given by contemporary naturopaths and other alternative practitioners to relieve the discomfort of symptoms associated with PMS, eczema, sunburn, fibrocystic breast disease, arthritis, and diabetes. It is also given to lower the risk of preeclampsia and eclampsia in pregnancy and osteoporosis in older women.

Description

Evening primrose oil is obtained from the seeds of the plant by pressing. The oil can be taken directly as a liquid or in the form of capsules.

Evening primrose oil is considered a useful dietary supplement because it is a good source of essential fatty acids (EFAs), Omega 6 predominantly. EFAs are called essential fatty acids because the human body cannot produce them; they must be obtained from the diet. EFAs maintain the function of cell membranes, regulate pain and inflammation, prevent blood clots, regulate blood pressure and cholesterol levels, and help to produce hormone-like substances known as prostaglandins. Prostaglandins function as inflammation mediators in the short-term regulation of glands and other body organs. It is thought that evening primrose oil relieves the symptoms of PMS by preferentially stimulating anti-inflammatory prostaglandins.

Under normal conditions, the body uses an EFA called linoleic acid to produce a compound called gamma linoleic acid, or GLA. Evening primrose oil contains both linoleic acid (74%) and GLA (9%), making it the most familiar and popular source of GLA. The other compounds contained in evening primrose oil are oleic acid (11%) and palmitic acid (6%).

KEY TERMS

Antioxidant—Substance that protects the body from damaging reactive oxygen molecules in the body. These reactive oxygen molecules can come from inside the body or from environmental pollution and are thought to play a role in the aging process and the development of degenerative disease.

Astringent—A substance or compound that causes contraction or constriction of soft tissue.

Eczema—An inflammation of the skin characterized by itching and oozing of a clear fluid.

Essential fatty acids (EFAs)—a group of polyunsaturated fats that are essential to life and growth but cannot be produced by the body.

Fibrocystic breast disease—A benign disorder of breast tissue characterized by fibrous saclike growths (cysts) that cause pain and tenderness.

Preeclampsia—A complication of pregnancy characterized by high blood pressure, fluid retention, and protein in the urine. If the patient develops convulsions, the condition is called eclampsia.

Prostaglandins—A group of unsaturated fatty acids involved in the contraction of smooth muscle, control of inflammation, and many other body processes.

Topical—A type of medication or preparation intended for use on the skin or external suface of the body. Evening primrose oil is used in topical preparations to soothe sunburn and relieve the itching of eczema.

Recommended dosage

Evening primrose oil can be obtained in health food stores in either liquid or capsule form. Consumers are advised to look for that which is organic and cold-pressed (not oxidized by heating), and to store it in the refrigerator. Standard dosage varies according to the condition being treated. The dosage for breast pain from fibrocystic disease is 3 g per day. For sunburn, patients may take up to 8 capsules daily until the symptoms subside. Dosages for eczema and rheumatoid arthritis depend on the concentration of GLA in the preparation of evening primrose oil, and should be decided in consultation with a physician, or naturopathic practitioner.

Evening primrose oil can also be used as a topical preparation to treat sunburn and eczema. One recipe for a homemade topical preparation calls for mixing one part of diced plant with four parts of heated petroleum jelly. The mixture is stored in a tightly closed container and refrigerated, as well.

All parts of the evening primrose plant are safe to eat. The roots can be boiled and eaten like parsnips. The seeds were roasted and used as a coffee substitute when food rationing was in effect during World War II.

Precautions

Evening primrose oil should not be given to patients with epilepsy, and only after a consultation with a physician should it be given to children.

Side effects

Evening primrose oil has not been reported as having toxic or severe side effects. Some patients, however, have reported nausea, headache, and softening of the stools.

Reports of side effects from using evening primrose oil in topical preparations for sunburn and other skin problems are the same as with any EFA supplement. Bruising due to damage of the blood platelet function is possible.

Interactions

Experts in pharmacology advise against using evening primrose oil with phenytoin (Dilantin) and other anticonvulsant medications, as the oil may lower the threshold for **seizures**. No other significant drug interactions have been reported.

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Rebecca J. Frey, Ph.D.

Executive function

Definition

The term executive function describes a set of cognitive abilities that control and regulate other abilities and behaviors. Executive functions are necessary for goal-directed behavior. They include the ability to initiate and stop actions, to monitor and change behavior as needed, and to plan future behavior when faced with novel tasks and situations. Executive functions allow us to anticipate outcomes and adapt to changing situations. The ability to form concepts and think abstractly are often considered components of executive function.

Description

As the name implies, executive functions are high-level abilities that influence more basic abilities like attention, memory and motor skills. For this reason, they can be difficult to assess directly. Many of the tests used to measure other abilities, particularly those that look at more complex aspects of these abilities, can be used to evaluate executive functions. For example, a person with executive function deficits may perform well on tests of basic attention, such as those that simply ask the individual to look at a computer screen and respond when a particular shape appears, but have trouble with tasks that require divided or alternating attention, such as giving a different response depending on the stimulus presented. Verbal fluency tests that ask people to say a number of words in a certain period of time can also reveal prob-

lems with executive function. One commonly used test asks individuals to name as many animals or as many words beginning with a particular letter as they can in one minute. A person with executive function deficits may find the animal naming task simple, but struggle to name words beginning with a particular letter, since this task requires people to organize concepts in a novel way. Executive functions also influence memory abilities by allowing people to employ strategies that can help them remember information. Other tests are designed to assess cognitive function more directly. Such tests may present a fairly simple task but without instructions on how to complete it. Executive functions allow most people to figure out the task demanded through trial and error and change strategies as needed.

Executive functions are important for successful adaptation and performance in real-life situations. They allow people to initiate and complete tasks and to persevere in the face of challenges. Because the environment can be unpredictable, executive functions are vital to human ability to recognize the significance of unexpected situations and to make alternative plans quickly when unusual events arise and interfere with normal routines. In this way, executive function contributes to success in work and school and allows people to manage the stresses of daily life. Executive functions also enable people to inhibit inappropriate behaviors. People with poor executive functions often have problems interacting with other people since they may say or do things that are bizarre or offensive to others. Most people experience impulses to do or say things that could get them in trouble, such as making a sexually explicit comment to a stranger, commenting negatively on someone's appearance, or insulting an authority figure like a boss or police officer; but most people have no trouble suppressing these urges. When executive functions are impaired, however, these urges may not be suppressed. Executive functions are thus an important component of the ability to fit in socially.

Executive function deficits are associated with a number of psychiatric and developmental disorders, including **obsessive-compulsive disorder**, Tourette's syndrome, depression, **schizophrenia**, **attention-deficit/hyperactivity disorder**, and **autism**. Executive function deficits also appear to play a role in antisocial behavior. Chronic heavy users of drugs and alcohol show impairments on tests of executive function. Some of these deficits appear to result from heavy substance use, but there is also evidence suggesting that problems with executive functions may contribute to the development of substance use disorders.

Because executive functions govern so many lower-level abilities, there is some controversy about their physiological basis. Nevertheless, most people who study these abilities agree that the frontal lobes of the **brain** play a major role in executive function. The frontal lobes are the large portions of the brain cortex that lie near the front of the brain. The cortex is the site in the brain where lower level processes like sensation and perception are processed and integrated into thoughts, memories and abilities, and actions are planned and initiated. People with frontal lobe injuries have difficulty with the higher

KEY TERMS

Autism—A developmental disability that appears early in life, in which normal brain development is disrupted and social and communication skills are retarded, sometimes severely.

Cognitive—Pertaining to the mental processes of memory, perception, judgment, and reasoning.

Cortex—Region in the brain where sensation and perception are processed and integrated into thoughts, memories, and abilities; also where actions are planned and initiated.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Executive—Pertaining to supervision, planning and carrying out duties or actions.

Frontal lobes—A region of the brain that influences higher mental functions often associated with intelligence, such as the ability to foresee the consequences of actions, planning, comprehension, and mood.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tourette syndrome—Neurological disorder characterized by multiple involuntary movements and uncontrollable vocalizations called tics that come and go over years, usually beginning in childhood and becoming chronic. Sometimes the tics include inappropriate language.

level processing that underlies executive functions. Because of its complexity, the frontal cortex develops more slowly than other parts of the brain, and not surprisingly, many executive functions do not fully develop until adolescence. Some executive functions also appear to decline in old age, and some executive function deficits may be useful in early detection of mild **dementia**.

See also Autism; Dementia; Schizophrenia; Tic disorders

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International Neuropsychological Society, 700 Ackerman Road, Suite 550, Columbus, OH 43202. <http://www.acs.ohio-state.edu/ins/>.

National Academy of Neuropsychology. 2121 South Oneida Street, Suite 550, Denver, CO 80224-2594. <http://nanonline.org/>.

Danielle Barry,M.S.

Exelon *see* **Rivastigmine**

Exhibitionism

Definition

Exhibitionism is a mental disorder characterized by a compulsion to display one's genitals to an unsuspecting stranger. The ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM-IV-TR,* classifies exhibitionism under the heading of the "paraphilias," a subcategory of sexual and gender identity disorders. The **paraphilias** are a group of mental disorders marked by **obsession** with unusual sexual practices or with sexual activity involving nonconsenting or inappropriate partners (such as children or animals). The term *paraphilia* is derived from two Greek words meaning "outside of" and "friendship-love."

In the United States and Canada, the slang term "flasher" is often used for exhibitionists.

Description

Exhibitionism is described in the *DSM-IV-TR* as the exposure of one's genitals to a stranger, usually with no intention of further sexual activity with the other person. For this reason, the term exhibitionism is sometimes grouped together with expression, "voyeurism," ("peeping," or watching an unsuspecting person or people, usually strangers, undressing or engaging in sexual activity) as a "hands-off" paraphilia. This contrasts with the "hands-on disorders" which involve physical contact with other persons.

In some cases, the exhibitionist masturbates while exposing himself (or while fantasizing that he is exposing himself) to the other person. Some exhibitionists are aware of a conscious desire to shock or upset their target; while others fantasize that the target will become sexually aroused by their display.

Causes and symptoms

Causes

Several theories have been proposed regarding the origins of exhibitionism. As of 2002, however, none are considered conclusive They include:

- Biological theories. These generally hold that testosterone, the hormone that influences the sexual drive in both men and women, increases the susceptibility of

KEY TERMS

Aversion therapy—An approach to treatment in which an unpleasant or painful stimulus is linked to an undesirable behavior in order to condition the patient to dislike or avoid the behavior.

Castration—Desexing a person or animal by surgical removal of the testes (in males) or ovaries (in females). Castration is sometimes offered as a treatment option to violent rapists and pedophiles who are repeat offenders.

Comorbidity—Association or presence of two or more mental disorders in the same patient. A disorder that is said to have a high degree of comorbidity is likely to occur in patients diagnosed with other disorders that may share or reinforce some of its symptoms.

Compliance—In medicine or psychiatry, cooperation with a treatment plan or schedule of medications.

Denial—A psychological defense mechanism that reduces anxiety by excluding recognition of an addiction or similar problem from the conscious mind.

Double-blind placebo-controlled study—A study in which patients are divided into two groups—those who will recive a medication, and those who will receive a placebo (a pill that looks like the medication but has no active ingredients). Neither the patients nor their physicians know which pill any specific patient is receiving.

Klinefelter's syndrome—A genetic disorder in males characterized by the presence of an extra X chromosome in addition to the normal XY. Most men with Klinefelter's syndrome have learning problems, are sterile, and have a shortened life expectancy.

Paraphilias—A group of mental disorders that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Placebo—An inactive substance or preparation used as a control in experiments with human subjects to test the effectiveness of a medication.

Recidivism—A tendency to return to a previously treated activity, or repeated relapse into criminal or deviant behavior.

Sequela (plural, sequelae)—An abnormal condition resulting from a previous disease or disorder.

Serotonin—A chemical produced by the brain that functions as a neurotransmitter. Low serotonin levels are associated with the paraphilias as well as with mood disorders. Medications known as selective serotonin reuptake inhibitors (SSRIs) can be used to treat exhibitionism and other paraphilias.

Voyeurism—A paraphilia that involves watching unsuspecting people, usually strangers, undress or engage in sexual activity.

males to develop deviant sexual behaviors. Some medications used to treat exhibitionists are given to lower the patients' testosterone levels.

- Learning theories. Several studies have shown that emotional **abuse** in childhood and family dysfunction are both significant risk factors in the development of exhibitionism.
- Psychoanalytical theories. These are based on the assumption that male gender identity requires the male child's separation from his mother psychologically so that he does not identify with her as a member of the same sex, the way a girl does. It is thought that exhibitionists regard their mothers as rejecting them on the basis of their different genitals. Therefore, they grow up with the desire to force women to accept them by making women look at their genitals.
- Head trauma. There are a small number of documented cases of men becoming exhibitionists following traumatic **brain** injury (TBI) without previous histories of alcohol abuse or sexual offenses.
- A childhood history of attention-deficit/hyperactivity disorder (ADHD). The reason for the connection is not yet known, but researchers at Harvard have discovered that patients with multiple paraphilias have a much greater likelihood of having had ADHD as children than men with only one paraphilia.

In general, psychiatrists disagree whether exhibitionism should be considered a disorder of impulse control or whether it falls within the spectrum of obsessive-compulsive disorders (OCDs). Further research into the anatomical structure and neurochemistry of the brain may help to settle this question.

As of 2002, there are no genes that have been associated with an increased risk of exhibitionism or other paraphilias. Such chromosomal abnormalities as Klinefelter's syndrome (where males have an extra X chromosome and are usually sterile) were at one time thought to be a risk factor for the development of paraphilias, but research has not yet proved a connection.

Symptoms

One expert in the field of treating paraphilias has suggested classifying the symptoms of exhibitionism according to level of severity, based on criteria from the *DSM-III-R* (1987):

- Mild. The person has recurrent fantasies of exposing himself, but has rarely or never acted on them.
- Moderate.The person has occasionally exposed himself (three targets or fewer) and has difficulty controlling urges to do so.
- Severe. The person has exposed himself to more than three people and has serious problems with control.
- A fourth level of severity, catastrophic, would not be found in exhibitionists without other paraphilias. This level denotes the presence of sadistic fantasies which, if acted upon, would result in severe injury or death to the victim.

Because exhibitionism is a hands-off paraphilia, it rarely rises above the level of moderate severity in the absence of other paraphilias.

Demographics

The incidence of exhibitionism in the general population is difficult to estimate because persons with this disorder do not usually seek counseling by their own free will. Exhibitionism is one of the three most common sexual offenses in police records (the other two are **voyeurism** and **pedophilia**). It is rarely diagnosed in general mental health clinics, but most professionals believe that it is probably underdiagnosed and underreported.

In terms of the technical definition of exhibitionism, almost all reported cases involve males. A number of mental health professionals, however, have noted that gender bias may be built into the standard definition. Some women engage in a form of exhibitionism by undressing in front of windows as if they are encouraging someone to watch them. In addition, wearing the low-cut gowns favored by some models and actresses have been described as socially sanctioned exhibitionism. One textbook description of exhibitionism says "women exhibit everything but the genitals; men, nothing but."

Although the stereotype of an exhibitionist is a "dirty old man in a raincoat," most males arrested for exhibitionism are in their late teens or early twenties. The disorder appears to have its onset before age 18. Like most paraphilias, exhibitionism is rarely found in men over 50 years of age.

In the U.S., most exhibitionists are Caucasian males. About half of exhibitionists are married.

Diagnosis

Diagnosis of exhibitionism is complicated by several factors. For example, most persons with the disorder come to therapy because of court orders. Some are motivated by fear of discovery by employers or family members, and a minority of exhibitionists enter therapy because their wife or girlfriend is distressed by the disorder. Emotional attitudes toward the disorder vary; some men maintain that the only problem they have with exhibitionism is society's disapproval of it; others, however, feel intensely guilty and anxious.

A second complication of diagnosing exhibitionism is the high rate of comorbidity among the paraphilias as a group and between the paraphilias as a group and other mental disorders. In other words, a patient in treatment for exhibitionism is highly likely to engage in other forms of deviant sexual behavior and to suffer from depression (an anxiety or substance-abuse disorder). In addition, many patients with paraphilias do not cooperate with physicians, who may have considerable difficulty making an accurate diagnosis of other disorders that may also exist.

A diagnosis of exhibitionism follows a somewhat different pattern from the standard procedures for diagnosing most mental disorders. A thorough workup in a clinic for specialized treatment of sexual disorders includes the following components:

- A psychiatric evaluation and mental status examination to diagnose concurrent psychiatric and medical conditions, and to rule out **schizophrenia**, **post-traumatic stress disorder** (PTSD), **mental retardation**, and depression.
- A neurologic examination to rule out head trauma, **seizures**, or other abnormalities of brain structure and function, followed by a **computed tomography** (CT) scan or **magnetic resonance imaging** (MRI), if needed.
- Blood and urine tests for substance abuse and sexually transmitted diseases, including an HIV screen.
- Assessment of sexual behaviors. This includes creation of a sex hormone profile and responses to questionnaires. The questionnaires are intended to measure cog-

nitive distortions regarding rape and other forms of coercion, pedophilia, aggression, and impulsivity.

Treatments

Exhibitionism is usually treated with a combination of **psychotherapy**, medications, and adjunctive treatments.

Psychotherapy

Several different types of psychotherapy have been found helpful in treating exhibitionism:

- **Cognitive-behavioral therapy** (CBT). This approach is generally regarded as the most effective form of psychotherapy for exhibitionism. Patients are encouraged to recognize the irrational justifications that they offer for their behavior, and to alter other distorted thinking patterns.
- Orgasmic reconditioning. In this technique, the patient is conditioned to replace fantasies of exposing himself with fantasies of more acceptable sexual behavior while masturbating.
- **Group therapy.** This form of therapy is used to get patients past the **denial** frequently associated with paraphilias, and as a form of relapse prevention.
- Twelve-step groups for sexual addicts. Exhibitionists who feel guilty and anxious about their behavior are often helped by the social support and emphasis on healthy spirituality found in these groups, as well as by the cognitive restructuring that is built into the twelve steps.
- **Couples therapy** or **family therapy**. This approach is particularly helpful for patients who are married and whose marriages and family ties have been strained by their disorder.

Medications

There are several different classes of drugs used to treat the patient with exhibitionism and the other paraphilias. However, one difficulty in evaluating the comparative efficacy of different medications should be noted: ethical limitation. Double-blind placebo-controlled studies of medication treatment of sexually deviant men raises the ethical question of the possibility of relapse in the subjects who receive the placebo. Withholding a potentially effective drug in circumstances that might lead to physical or psychological injury to a third party is difficult to justify.

As of 2002, medications are the only form of treatment for patients with exhibitionism that have the capability to suppress deviant behaviors. The categories of drugs used to treat exhibitionism are as follows:

- Selective serotonin reuptake inhibitors (SSRIs). The SSRIs show promise in treating the paraphilias, as well as depression and other mood disorders. It has been found that decreased levels of serotonin in the brain result in an increased sex drive. The SSRIs are appropriate for patients with mild- or moderate-level paraphilias; these patients include the majority of exhibitionists.
- Female hormones. Estrogens have been used to treat sexual offenders since the 1940s. Medroxyprogesterone acetate, or MPA, is the most widely used hormonal medication in the U.S. for the treatment of people with exhibitionism. Medroxyprogesterone acetate works by stimulating the liver to produce a chemical that speeds up the clearance of testosterone from the bloodstream. It is effective as long as patients are take their MPA as prescribed by their physicians. Unfortunately, MPA can cause several troublesome side effects in some patients. These include nausea, vomiting, weight gain, and headache.
- Luteinizing hormone-releasing hormone (LHRH) agonists. These drugs are sometimes described to be the equivalent of pharmacologic castration. They work by reducing the release of gonadotropin hormones. The LHRH agonists include such drugs as triptorelin (Trelstar), leuprolide acetate, and goserelin acetate.
- Antiandrogens. These drugs block the uptake and metabolism of testosterone and reduce the blood levels of testosterone. The antiandrogens include cyproterone acetate (CPA) and flutamide. Cyproterone acetate has been used in Germany to treat exhibitionists since the early 1970s, and most long-term studies of the CPA have been done by German psychiatrists. The drug appears to have minimal side effects in long-term use and significantly reduces recidivism (relapse and repetition of the deviant behavior).

Surgery

Surgical castration, which involves removal of the testes, is effective in significantly reducing levels of testosterone in blood plasma. This form of treatment for paraphilias, however, is generally reserved for more serious offenders than exhibitionists (violent rapists and pedophiles with a history of repeated offenses, for example).

Other treatment methods

Another method of treating patients with exhibitionism disorder, used more frequently in the 1970s and 1980s than today, is electroshock aversion. While a mild electric shock was administered, the patient was shown pictures, projected onto a screen, of men exposing them-

selves. In 2002, **aversion therapy** involves asking the patient to fantasize a sequence of events leading up to his exhibitionism. Then, a very unpleasant scene is inserted at a crucial point in the sequence. The patient might, for example, be asked to imagine a police officer approaching as he exposes himself, or to think of his target fighting back or laughing at him.

Another treatment method that is often offered to people with exhibition disorder is **social skills training**. It is thought that some men develop paraphilias partially because they do not know how to form healthy relationships, whether sexual or nonsexual, with other people. Although social skills training is not considered a substitute for medications or psychotherapy, it appears to be a useful adjunctive treatment for exhibitionism disorder.

Legal considerations

People with exhibitionism disorder are at risk for lifetime employment problems if they acquire a police record. An attorney who specializes in employment law has pointed out that the Americans with Disabilities Act (ADA), enacted by Congress in 1990 to protect workers against discrimination on grounds of mental impairment or physical disability, does not protect persons with paraphilias. People with exhibitionism disorder were specifically excluded by Congress from the provisions of the ADA, along with voyeurs and persons with other sexual behavior disorders.

Prognosis

The prognosis for people with exhibition disorder depends on a number of factors, including the age of onset, the reasons for the patient's referral to psychiatric care, degree of his cooperation with the therapist, and comorbidity with other paraphilias or other mental disorders. For some patients, exhibitionism is a temporary disorder related to sexual experimentation during their adolescence. For others, however, it is a lifelong problem with potentially serious legal, interpersonal, financial, educational, and occupational consequences. People with exhibition disorder have the highest recidivism rate of all the paraphilias; between 20% and 50% of men arrested for exhibitionism are re-arrested within two years.

Prevention

One important preventive strategy includes the funding of programs for the treatment of paraphilias in adolescents. According to one expert in the field, males in this age group have not been studied and are undertreated, yet it is known that paraphilias are usually established before age 18. Recognition of paraphilias in adolescents and treatment for those at risk would lower the risk of recidivism. A second important preventive approach is early recognition and appropriate treatment of people who have committed child abuse.

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ORGANIZATIONS

Augustine Fellowship, Sex and Love Addicts Anonymous. PO Box 119, New Town Branch, Boston, MA 02258. (617) 332-1845.

National Association on Sexual Addiction Problems (NASAP). 22937 Arlington Avenue, Suite 201, Torrance, CA 90501. (213) 546-3103.

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Exposure treatment

Definition

Exposure treatment is a technique that is widely used in **cognitive-behavioral therapy** (CBT). Exposure treatment is used for a variety of anxiety disorders, and it has also recently been extended to the treatment of substance-related disorders. Generally speaking, exposure treatment involves presenting a patient with anxiety-producing material for a long enough time to decrease the intensity of their emotional reaction. As a result, the feared situation or thing no longer makes the patient anxious. Exposure treatment can be carried out in real situations, which is called in vivo exposure; or it can be done through imagination, which is called imaginal exposure. The category of imaginal exposure includes **systematic desensitization**, which asks the patient to imagine certain aspects of the feared object or situation combined with relaxation. Graded or graduated exposure refers to exposing the patient to the feared situation in a gradual manner. Flooding refers to exposing the patient to the anxiety-provoking or feared situation all at once and kept in it until the anxiety and fear subside. There are several variations in the delivery of exposure treatment: patient-directed exposure instructions or self-exposure; therapist-assisted exposure; group exposure; and exposure with response prevention.

Purpose

The basic purpose of exposure treatment is to decrease a person's anxious and fearful reactions (emotions, thoughts, or physical sensations) through repeated exposures to anxiety-producing material. This reduction of the patient's anxiety response is known as habituation. A related purpose of exposure treatment is to eliminate the anxious or fearful response altogether so that the patient can face the feared situation repeatedly without

KEY TERMS

Cognitive restructuring—An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

Cue—Any behavior or event in a person's environment that serves to stimulate a particular response. For example, the smell of liquor may be a cue for some people to pour themselves a drink.

Desensitization—The reduction or elimination of an overly intense reaction to a cue by controlled repeated exposures to the cue.

Extinction—The elimination or removal of a person's reaction to a cue as a result of exposure treatment.

Flooding—A type of exposure treatment in which the patient is exposed to an anxiety-provoking or feared situation all at once and kept in it until the anxiety and fear subside.

Habituation—The reduction of a person's emotional or behavioral reaction to a cue by repeated or prolonged exposure.

Hierarchy—In exposure therapy, a list of feared items or situations, ranked from least fearsome to most fearsome.

In vivo—A Latin phrase that means "in life." In modeling and exposure therapies, it refers to practicing new behaviors in a real setting, as distinct from using imagery or imagined settings.

Interoceptive—Referring to stimuli or sensations that arise inside the body. In interoceptive exposure treatment, the patient is asked to exercise or perform other actions that produce feared internal physical sensations.

Modality—The medical term for a method of treatment.

Subjective units of distress (SUDS) scale—A scale used by patients during exposure treatment to rate their levels of fear and anxiety with numbers from zero to 100.

Virtual reality—A realistic simulation of an environment, produced by a computer system using interactive hardware and software.

experiencing anxiety or fear. This elimination of the anxiety response is known as extinction.

Precautions

Exposure treatment is generally a safe treatment method; however, some patients may find that the level of anxiety that occurs during treatment sessions is higher than they can handle. Some studies of exposure treatment have reported a high dropout rate, perhaps because the method itself produces anxiety. In addition, exposure treatment is not effective for all patients; after treatment, some continue to experience anxiety symptoms.

Description

Exposure treatment usually begins with making a list or hierarchy of situations that make the patient anxious or fearful. The situations are ranked on a scale of zero (representing the situation producing the least anxiety) to ten (representing the situation of highest anxiety). In addition, patients are usually asked to rate their level of anxiety in each situation on a scale from zero (no anxiety or discomfort) to 100 (extreme anxiety and discomfort). This scale is called the subjective units of distress scale, or SUDS. Patients may be asked to provide SUDS ratings at regular intervals during exposure treatment, for example every five minutes.

Methods of delivering exposure treatment

PATIENT-DIRECTED EXPOSURE. Patient-directed exposure is the simplest variation of exposure treatment. After the patient makes his or her hierarchy list with the therapist, he or she is instructed to move through the situations on the hierarchy at his or her own rate. The patient starts with the lowest anxiety situation on the list, and keeps a journal of his or her experiences. Patient-directed exposure is done on a daily basis until the patient's fears and anxiety have decreased. For example, if a patient is afraid of leaving the house, the first item on the hierarchy might be to stand outside the front door for a certain period of time. After the patient is able to perform this action without feeling anxious, he or she would move to the next item on the hierarchy, which might be walking to the end of the driveway. Treatment would proceed in this way until the patient has completed all the items on the hierarchy. During therapy sessions, the therapist reviews the patient's journal; gives the patient positive feedback for any progress that he or she has made; and discusses any obstacles that the patient encountered during exposures to the feared situation.

THERAPIST-ASSISTED EXPOSURE. In this form of exposure treatment, the therapist goes with the patient to the feared location or situation and provides on-the-spot coaching to help the patient manage his or her anxiety. The therapist may challenge the patient to experience the maximum amount of anxiety. In prolonged in vivo exposure, the therapist and patient stay in the situation as long as it takes for the anxiety to decrease. For example, they might remain in a crowded shopping mall for four or more hours. The therapist also explores the patient's thoughts during this exposure so that any irrational ways of thinking can be confronted.

GROUP EXPOSURE. In group exposure, self-exposure and practice are combined with group education and discussion of experiences during exposure to feared situations. These sessions may last as long as three hours and include 30 minutes of education, time for individual exposure practice, and 45 minutes of discussion. Group sessions may be scheduled on a daily basis for 10–14 days.

Exposure treatment for specific anxiety disorders

AGORAPHOBIA. Many research studies have shown that graded exposure treatment is effective for **agoraphobia**. Long-term studies have shown that improvement can be maintained for as long as seven years. Exposure treatment for agoraphobia is best done in vivo, in the actual feared situation, for example entering a packed subway car. Exposure treatment for agoraphobia is likely to be more effective when the patient's spouse or friend is involved, perhaps because of the support a companion can offer the patient during practice sessions.

PANIC DISORDER. Exposure treatment is the central component of cognitive-behavioral treatment for **panic disorder**. Treatment for this disorder involves identifying specific fears within the patient's experience of panic, such as fears of being sick, fears of losing control, and fears of embarrassment. Once these fears are identified, the patient is instructed to expose himself or herself to situations in which the fearful thoughts arise (walking away from a safe person or place, for example). The rationale behind this instruction is that enduring the anxiety associated with the situation will accustom the patient to the situation itself, so that over time the anxiety will diminish or disappear. In this way, the patient discovers that the feared consequences do not happen in real life.

In some patients, physical symptoms of panic lead to fears about the experience of panic itself. Fears related to the physical symptoms associated with panic can be targeted for treatment by inducing the bodily sensations that mimic those experienced during a **panic attack**. This technique is called interoceptive exposure. The patient is asked to induce the feared sensations in a number of ways. For example, the patient may spin in a revolving chair to induce dizziness or run up the stairs to induce increased heart rate and shortness of breath. The patient

is then instructed to notice what the symptoms feel like, and allow them to remain without doing anything to control them. With repeated exposure, the patient learns that the bodily sensations do not signal harm or danger, and therefore need not be feared. The patient is taught such strategies as muscle relaxation and slow breathing to control anxiety before, during, and after the exposure.

Interoceptive exposure treatment for panic usually begins with practice sessions in the therapist's office. The patient may be instructed to practice at home and then practice in a less "safe" environment, such as the patient's work setting or a nearby park. The next step is the addition of the physical activities that naturally produce the feared symptoms. Situational or in vivo exposure would then be introduced for patients with agoraphobia combined with panic disorder. The patient would be instructed to go back into a situation that he or she has been avoiding, such as an elevator or busy railroad terminal. If the patient develops symptoms of anxiety, he or she is instructed to use the techniques for controlling anxiety that were previously learned.

The effectiveness of exposure treatment for decreasing panic attacks and avoidance has been well demonstrated. In research studies, 50%–90% of patients experience relief from symptoms.

SPECIFIC PHOBIA AND SOCIAL PHOBIA. Graded exposure is used most often to treat **specific phobia** or simple phobia. In graded exposure, the patient approaches the feared object or situation by degrees. For example, someone afraid of swimming in the ocean might begin with looking at photographs of the ocean, then watch movies of people swimming, then go to the beach and walk along the water's edge, and then work up to a full swim in the ocean. Graded exposure can be done through patient-directed instruction or therapist-assisted exposure. Research studies indicate that most patients respond quickly to graded exposure treatment, and that the benefits of treatment are well maintained.

Treatment for **social phobia** usually combines exposure treatment with cognitive restructuring. This combination seems to help prevent a recurrence of symptoms. In general, studies of exposure treatment for social phobia have shown that it leads to a reduction of symptoms. Since cognitive restructuring is usually combined with exposure, it is unclear which component is responsible for the patients' improvement, but there is some indication that exposure alone may be sufficient.

Exposure treatment can be more difficult to arrange for treating social phobia, however, because the patient has less control over social situations, which are unpredictable by their nature and can unexpectedly become more intense and anxiety-provoking. Furthermore, social exchanges usually last only a short time; therefore, they may not provide the length of exposure that the patient needs.

OBSESSIVE-COMPULSIVE DISORDER. The most common non-medication treatment for **obsessive-compulsive disorder** (OCD) is exposure to the feared or anxiety-producing situation plus response prevention (preventing the patient from performing a compulsive behavior, such as hand washing after exposure to something thought to be contaminated). This form of treatment also uses a hierarchy, and begins with the easiest situation and gradually moves to more difficult situations. Research has shown that exposure to contamination situations leads to a decrease in fears of contamination, but does not lead to changes in the compulsive behavior. In a similar fashion, the response prevention component leads to a decrease in compulsive behavior, but does not affect the patient's fears of contamination. Since each form of treatment affects different OCD symptoms, a combination of exposure and response prevention is more effective than either modality by itself. Exposure combined with response prevention also appears to be effective for treating OCD in children and adolescents.

Prolonged continuous exposure is better than short, interrupted periods of exposure in treating OCD. On average, exposure treatment of OCD requires 90-minute sessions, although the frequency of sessions varies. Some studies have shown good results with 15 daily treatments spread over a period of three weeks. This intensive treatment format may be best suited for cases that are more severe and complex, as in patients suffering from depression as well as OCD. Patients who are less severely affected and are highly motivated may benefit from sessions once or twice a week. Treatment may include both therapist-assisted exposure and self-exposure as homework between sessions. Imaginal exposure may be useful for addressing fears that are hard to include in vivo exposure, such as fears of a loved one's death. Patients usually prefer gradual exposure to the most distressing situation in their hierarchy; however, gradual exposure does not appear to be more effective than flooding or immediate exposure to the situation.

POST-TRAUMATIC STRESS DISORDER. Exposure treatment has been used successfully in the therapy of **post-traumatic stress disorder** (PTSD) resulting from such traumatic experiences as combat, sexual assault, and motor vehicle accidents. Research studies have reported encouraging results for exposure treatment in reducing PTSD or PTSD symptoms in children, adolescents, and adults. Such intrusive symptoms of PTSD as nightmares and flashbacks may be reduced by having the patient relive the emotional aspects of the trauma in a safe therapeutic environment. It may take 10–15 exposure sessions to decrease the negative physical sensations associated with PTSD. These sessions

may range from one to two hours in length and may occur once or twice a week. Relaxation techniques are usually included before and after exposure. The exposure may be therapist-assisted or patient-directed.

A recent study showed that imaginal exposure and cognitive treatment are equally effective in reducing symptoms associated with chronic or severe PTSD, but that neither brought about complete improvement. In addition, more patients treated with exposure worsened over the course of treatment than patients treated with cognitive approaches. This finding may have been related to the fact that the patients receiving exposure treatment had less frequent sessions with long periods of time between sessions. Some patients diagnosed with PTSD, however, do not seem to benefit from exposure therapy. They may have difficulty tolerating exposure, or have difficulty imagining, visualizing, or describing their traumatic experiences. The use of cognitive therapy to help the patient focus on thoughts may be a useful adjunctive treatment, or serve as an alternative to exposure treatment.

Many persons who have undergone sexual assault or rape meet *DSM-IV-TR* criteria for PTSD. They may re-experience the traumatic event, avoid items or places associated with the trauma, and have increased levels of physical arousal. Exposure treatment in these cases involves using either imaginal or in vivo exposure to reduce anxiety and any tendencies to avoid aspects of the situation that produce anxiety (also known as avoidance behavior). Verbal description of the event (imaginal exposure) is critical for recovery, although it usually feels painful and threatening to patients. It is important that the patient's verbal description of the traumatic event, along with the expression of thoughts and feelings related to it, occur as early in the treatment process as possible. It is in the patient's "best long-term interest to experience more discomfort temporarily in order to suffer less in the long run."

Prolonged exposure is the most effective non-medical treatment for reducing traumatic memories related to PTSD. It combines flooding with systematic desensitization. The goal is to expose patients using both imaginal and in vivo exposure techniques in order to reduce avoidance behavior and decrease fears. Prolonged exposure may occur over nine to 12 ninety-minute sessions. During the imaginal exposure phase of treatment, the patient is asked to describe the details of the traumatic experience repeatedly, in the present tense. The patient uses the SUDS scale to monitor levels of fear and anxiety. The in vivo component occurs outside the therapist's office; it involves the client exposing himself or herself to cues in the environment that he or she has been avoiding— for example, the place where the motor vehicle accident or rape occurred. The patient is instructed to stay in the fear-producing situation for at least 45 minutes, or until their anxiety levels have gone down significantly on the SUDS rating scale. Often patients will use a coach or someone who will stay with them at the beginning of in vivo practice. The coach's role gradually decreases over time as the patient experiences less anxiety.

Recent innovations in exposure treatment

VIRTUAL REALITY EXPOSURE TREATMENT. Virtual reality is a technique that allows a person to participate actively in a computer-generated (or virtual) scenario or environment. The participant has the sense of being present in the virtual environment. Virtual reality uses a device mounted on the participant's head that shows computer graphics and visual displays in real time, and tracks the person's body movements. Some forms of virtual reality also allow participants to hold a second device in their hands that enables them to interact more fully with the virtual environment, such as opening a car door.

Virtual reality has been proposed as a new way of conducting exposure therapy because it can provide a sense of being present in a feared situation. Virtual reality exposure may be useful for treating such phobias as fear of heights, flying, or driving, as well as for treating PTSD. This method appears to have several advantages over standard exposure therapy. First, virtual reality may offer patients a greater sense of control because they can instantly turn the device on and off or change its level of intensity. Second, virtual reality would protect patients from harm or social embarrassment during their practice sessions. Third, it could be implemented regardless of the patient's ability to imagine or to remain with prolonged imaginal exposure. These proposed advantages of virtual reality over standard exposure therapy have yet to be tested, however.

Some studies have been conducted using virtual reality in the treatment of patients with fear of heights and fear of flying, and in a sample of Vietnam veterans diagnosed with PTSD. These studies of virtual reality exposure therapy have limitations in terms of study design and small sample size, but their positive results suggest that virtual reality exposure therapy deserves further investigation.

CUE EXPOSURE TREATMENT FOR ALCOHOL DEPENDENCE. Cue exposure is a relatively new approach to treating substance-related disorders. It is designed to recreate real-life situations in a safe therapeutic environment that expose patients repeatedly to alcohol-related cues, such as the sight or smell of alcohol. It is thought that this repeated exposure to cues, plus prevention of the usual response (drinking alcohol) will reduce and possibly eliminate urges experienced in reaction to the cues.

Persons diagnosed with alcohol dependence face a number of alcohol-related cues in their environment, including moods associated with previous drinking pat-

terns; people, places, times, and objects associated with the pleasurable effects of alcohol; and the sight or smell of alcoholic beverages. Exposure to these cues increases the patient's risk of relapse, because the cues can interfere with a person's use of coping skills to resist the urge to drink. The purpose of cue exposure is to teach patients coping skills for responding to these urges. It is thought that a person who practices coping skills in the presence of cues will find the coping skills strengthened, along with the conviction that he or she can respond effectively when confronted by similar cues in real-life situations.

There are various approaches to cue exposure. The choice of cues is usually based on treatment philosophy and goals, which may require abstinence from alcohol or permit moderate drinking. In abstinence-only programs, patients may be exposed to actual alcohol cues and/or imagined high-risk situations. This imaginal exposure is useful for dealing with cues and circumstances that cannot be reproduced in treatment settings, such as fights. Patients learn and practice urge-specific coping skills. While a patient may learn to cope successfully with one cue (such as the smell of alcohol), the urge to drink may reappear in response to another cue, such as seeing a friend with whom they used to go to bars. The patient would then learn how to manage this particular cue. This program may take six to eight individual or group sessions and may occur on an inpatient or outpatient basis. Often patients remain in the treatment setting for several hours after the exposure to ensure that any lasting urges are safely managed with the therapist's help.

More specifically, cue exposure focuses on the aspect of alcohol consumption that produces the strongest urge. The patient would report each change in their level of urgency, using a scale of zero to ten that resembles the SUDS scale. The urge to drink usually peaks after one to five minutes. When the desire for a drink arises, the patient is instructed to focus on the cue to see what happens to their desire. In most cases the urge subsides within 15 minutes, which is often different from what the patient expected. In later sessions, the patient is instructed when the urge peaks to imagine using the coping skills that he or she was recently taught. The patient may also be instructed to imagine being in high-risk situations and using the coping skills. Some examples of these coping skills include telling oneself that the urge will go away; picturing the negative consequences of drinking alcohol; and thinking of the positive consequences of staying sober.

Although there has been little research on cue exposure, available studies show positive outcomes in terms of decreasing the patients' consumption of alcohol. There have been, however, few outcome studies comparing cue exposure treatment to other treatment approaches. It may be hard to separate the benefits due to exposure from the benefits due to coping skills training. In any event, cue exposure treatment is a promising approach that deserves further study to determine if either component alone is sufficient or if a combination of the two is more effective.

Normal results

Progress in exposure therapy is often slow in the beginning, and occasional setbacks are to be expected. As the patient gains experience with various anxiety-producing situations, his or her rate of progress may increase. While flooding can produce positive results more quickly than graded exposure, it is rarely used because of the high level of discomfort associated with it.

See also Agoraphobia; Alcohol and related disorders; Anxiety and anxiety disorders; Anxiety-reduction techniques; Cognitive-behavioral therapy; Obsessive-compulsive disorder; Panic attack; Panic disorder; Systematic desensitization

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Joneis Thomas, Ph.D.

Expressive language disorder

Definition

Expressive language disorder occurs when an individual has problems expressing him or herself using spoken language.

Description

Expressive language disorder is generally a childhood disorder. There are two types of expressive language disorder: the developmental type and the acquired type. Developmental expressive language disorder does not have a known cause and generally appears at the time a child is learning to talk. Acquired expressive language disorder is caused by damage to the **brain**. It occurs suddenly after events such as **stroke** or traumatic head injury. The acquired type can occur at any age.

Causes and symptoms

Causes

There is no known cause of developmental expressive language disorder. Research is ongoing to determine which biological or environmental factors may be the cause. Acquired expressive language disorder is caused by damage to the brain. Damage can be sustained during a stroke, or as the result of traumatic head injury, **seizures**, or other medical conditions. The way in which acquired expressive language disorder manifests itself in a specific person depends on which parts of the brain are injured and how badly they are damaged.

Symptoms

Expressive language disorder is characterized by a child having difficulty expressing him- or herself using speech. The signs and symptoms vary drastically from child to child. The child does not have problems with the pronunciation of words, as occurs in **phonological disorder**. The child does have problems putting sentences together coherently, using proper grammar, recalling the appropriate word to use, or other similar problems. A child with expressive language disorder is not able to communicate thoughts, needs, or wants at the same level or with the same complexity as his or her peers. The child often has a smaller vocabulary than his or her peers.

Children with expressive language disorder have the same ability to understand speech as their peers, and have the same level of intelligence. Therefore, a child with this disorder may understand words that he or she cannot use in sentences. The child may understand complex spoken sentences and be able to carry out intricate instructions, although he or she cannot form complex sentences.

There are many different ways in which expressive language disorder can manifest itself. Some children do not properly use pronouns, or leave out functional words such as "is" or "the." Other children cannot recall words that they want to use in the sentence and substitute general words such as "thing" or "stuff." Some children cannot organize their sentences so that the sentences are easy to understand. These children do comprehend the material

they are trying to express—they just cannot create the appropriate sentences with which to express their thoughts.

Demographics

Expressive language disorder is a relatively common childhood disorder. Language delays occur in 10–15% of children under age three, and in 3–7% of school-age children. Expressive language disorder is more common in boys that in girls. Studies suggest that developmental expressive language disorder occurs two to five times more often in boys as girls. The developmental form of the disorder is far more common than the acquired type.

Diagnosis

To diagnose expressive language disorder, children must be performing below their peers at tasks that require communication in the form of speech. This can be hard to determine because it must be shown that an individual understands the material, but cannot express that comprehension. Therefore, non-verbal tests must be used in addition to tests that require spoken answers. Hearing should also be evaluated, because children who do not hear well may have problems putting together sentences similar to children with expressive language disorder. In children who are mildly hearing impaired, the problem can often be resolved by using hearing aids to enhance the child's hearing. Also, children who speak a language other than English (or the dominant language of their society) in the home should be tested in that language if possible. The child's ability to communicate in English may be the problem, not the child's ability to communicate in general.

The ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revised (known as the *DSM-IV-TR*), states that there are four general criteria for diagnosing expressive language disorder. The first is that the child communicates using speech at a level that is less developed than expected for his or her intelligence and ability to understand spoken language. This problem with communication using speech must create difficulties for the child in everyday life or in achieving goals. The child must understand what is being said at a level that is age-appropriate, or at a developmental level consistent with the child's. Otherwise the diagnoses should be **mixed receptive-expressive language disorder**. If the child has **mental retardation**, poor hearing, or other problems, the difficulties with speech must be greater than is generally associated with the handicaps that the child has.

Treatment

There are two types of treatment used for expressive language disorder. The first involves the child working one-on-one with a speech therapist on a regular schedule and practicing speech and communication skills. The second type of treatment involves the child's parents and teachers working together to incorporate spoken language that the child needs into everyday activities and play. Both of these kinds of treatment can be effective, and are often used together.

Prognosis

The developmental form of expressive language disorder generally has a good prognosis. Most children develop normal or nearly normal language skills by high school. In some cases, minor problems with expressive language may never resolve. The acquired type of expressive language disorder has a prognosis that depends on the nature and location of the brain injury. Some people get their language skills back over days or months. For others it takes years, and some people never fully recover expressive language function.

Prevention

There is no known way to prevent developmental expressive language disorder. Because acquired language disorder is caused by damage to the brain, anything that would help to prevent brain damage may help to prevent that type of the disorder. This can include such things ranging from lowering cholesterol in order to prevent stroke to wearing a bicycle helmet to prevent traumatic brain injury.

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American Psychological Association. 750 First Street NE, Washington, DC 20002-4242. (800) 374-2721. <www.apa.org>.

American Speech-Language-Hearing Association. 10801 Rockville Pike, Rockville, MD 20852. (800) 638-8355. <http://www.asha.org>.

Tish Davidson, A.M.

F

Factitious disorder

Definition

Factitious disorder (FD) is an umbrella category that covers a group of mental disturbances in which patients intentionally act physically or mentally ill without obvious benefits. According to one estimate, the unnecessary tests and waste of other medical resources caused by FD cost the United States $40 million per year. The name factitious comes from a Latin word that means "artificial" or "contrived."

The *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV-TR*, fourth edition) distinguishes factitious disorder from **malingering**, which is defined as pretending illness when the individual has a clear motive—usually to benefit economically or to avoid legal trouble.

Factitious disorder is sometimes referred to as hospital **addiction**, pathomimia, or polysurgical addiction. Variant names for individuals with FD include hospital vagrants, hospital hoboes, peregrinating patients, problem patients, and professional patients.

Description

Cases of factitious disorder appear in the medical literature as early as Galen, a famous Roman physician of the second century A.D. The term factitious is derived from a book by an English physician named Gavin, published in 1843, entitled *On Feigned and Factitious Diseases*. The modern study of factitious disorder, however, began with a 1951 article in *Lancet* by a British **psychiatrist**, Richard Asher, who also coined the term Munchausen's syndrome to describe a chronic subtype of FD. The name comes from an eighteenth-century German baron who liked to embellish stories of his military exploits in order to impress his listeners. In 1977, it was Gellengerg who first reported a case of FD with primarily psychological symptoms. Factitious disorder was recognized as a formal diagnostic category by *DSM-III* in 1980.

DSM-IV-TR defines factitious disorder as having three major subtypes: FD with predominantly psychological signs and symptoms; FD with predominantly physical signs and symptoms; and FD with combined psychological and physical signs and symptoms. A fourth syndrome, known as Ganser syndrome, has been classified in the past as a form of factitious disorder, although *DSM-IV-TR* groups it with the dissociative disorders.

DSM-IV-TR specifies three criteria for factitious disorder:

- The patient is intentionally producing or pretending to have physical or psychological symptoms or signs of illness.
- The patient's motivation is to assume the role of a sick person.
- There are no external motives (as in malingering) that explain the behavior.

Psychological FD

Factitious disorder with predominantly psychological signs and symptoms is listed by *DSM-IV-TR* as the first subcategory of the disorder. It is characterized by the individual feigning psychological symptoms.

Some researchers have suggested adding the following criteria for this subtype of FD:

- The symptoms are inconsistent, changing markedly from day to day and from one **hospitalization** to the next.
- The changes are influenced by the environment (as when the patient feels observed by others) rather than by the treatment.
- The patient's symptoms are unusual or unbelievable.
- The patient has a large number of symptoms that belong to several different psychiatric disorders.

Physical FD

Factitious disorder wih predominantly physical signs and symptoms is the most familiar to medical per-

KEY TERMS

Conversion disorder—A type of somatoform disorder in which unconscious psychological conflicts or other factors take the form of physical symptoms that are produced unintentionally. Conversion disorder is part of the differential diagnosis of factitious disorder.

Forensic—Pertaining to courtroom procedure or evidence used in courts of law.

Ganser syndrome—A rare subtype of factitious disorder accompanied by dissociative symptoms. It is most often seen in male patients under severe stress in prison or courtroom settings.

Gridiron abdomen—An abdomen with a network of parallel scars from repeated surgical operations.

Malingering—Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

Masochism—A mental disorder in which a person obtains sexual satisfaction through pain or humiliation inflicted by the self or by another person. The term is sometimes used more generally to refer to a tendency to find pleasure in submissiveness or self-denial.

sonnel. Chronic FD of this type is often referred to as Munchausen's syndrome. The most common ways of pretending illness are: presenting a factitious history (claiming to have had a seizure that never happened); combining a factitious history with external agents that mimic the symptoms of disease (adding blood from a finger prick to a urine sample); or combining a factitious history with maneuvers that produce a genuine medical condition (taking a psychoactive drug to produce psychiatric symptoms). In most cases, these patients sign out of the hospital when they are confronted by staff with proof of their pretending, usually in the form of a laboratory report. Many individuals with Munchausen's syndrome move from hospital to hospital, seeking treatment, and thus are known commonly as "hospital hoboes."

FD with mixed symptoms

Factitious disorder in this category is characterized by a mix of psychological and physical signs and symptoms.

FD not otherwise specified

Factitious disorder not otherwise specified is a category that *DSM-IV* included to cover a bizarre subtype in which one person fabricates misleading information about another's health or induces actual symptoms of illness in the other person. First described in 1977 by an American pediatrician, this syndrome is known as Munchausen syndrome by proxy (MSBP) and almost always involves a parent (usually the mother) and child. MSBP is now understood as a form of child **abuse** involving premeditation rather than impulsive acting out. Many pediatricians in the United States believe that MSBP is underdiagnosed.

Ganser syndrome

Ganser syndrome is a rare disorder (about a 100 documented cases worldwide) that has been variously categorized as a factitious disorder or a dissociative disorder. It is named for a German psychiatrist named Sigbert Ganser, who first described it in 1898 from an examination of male prisoners who were thought to be psychotic. Ganser syndrome is used to describe dissociative symptoms and the pretending of **psychosis** that occur in forensic settings.

There are four symptoms regarded as diagnostic of Ganser syndrome:

- *Vorbeireden*: A German word that means "talking beside the point," it refers to a type of approximate answer to an examiner's questions that may appear silly but usually indicates that the patient understands the question. If examiner asks how many legs a dog has, the patient may answer, "Five."
- Clouding of consciousness: The patient is drowsy or inattentive.
- Conversion symptoms: These are physical symptoms produced by unconscious psychological issues rather than diagnosable medical causes. A common conversion symptom is temporary paralysis of an arm or leg.
- Hallucinations.

Virtual FD

Although virtual factitious disorder does not appear as a heading in any present diagnostic manual, it is a phenomenon that has appeared with increasing frequency with the spread of the Internet. The growing use of the personal computer has affected presentations of factitious disorder in two important ways. First, computers allow people with sufficient technical skills to access medical records from hospital databases and cut-and-paste changes into their own records in order to falsify their medical histories. Second, computers allow people to enter Internet chat rooms for persons with serious illnesses and pretend to be a patient with that illness in order to obtain attention and sympathy. "Munchausen by

Internet" can have devastating effects on chat groups, destroying trust when the hoax is exposed.

Causes and symptoms

Causes

The causes of factitious disorder, whether physical or psychiatric, are difficult to determine because these patients are often lost to follow-up when they sign out of the hospital. **Magnetic resonance imaging** (MRI) has detected abnormalities in the **brain** structure of some patients with chronic FD, suggesting that there may be biological or genetic factors in the disorder. PET scans of patients diagnosed with Ganser syndrome have also revealed brain abnormalities. The results of EEG (**electroencephalography**) studies of these patients are nonspecific.

Several different psychodynamic explanations have been proposed for factitious disorder. These include:

- Patients with FD are trying to re-enact unresolved childhood issues with parents.
- They have underlying problems with masochism.
- They need to be the center of attention and feel important.
- They need to receive care and nurturance.
- They are bothered by feelings of vulnerability.
- Deceiving a physician allows them to feel superior to an authority figure.

There are several known risk factors for factitious disorder, including:

- The presence of other mental or physical disorders in childhood that resulted in the patient's getting considerable medical attention.
- A history of significant past relationships with doctors, or of grudges against them.
- Present **diagnosis** of borderline, narcissistic, or antisocial personality disorder.

Symptoms

SYMPTOMS OF FACTITIOUS DISORDER IN ADULTS OR ADOLESCENTS. Reasons for suspecting factitious disorder include:

- The individual's history is vague and inconsistent; or the individual has a long medical record with many admissions at different hospitals in different cities.
- The patient has an unusual knowledge of medical terminology or describes the illness as if they are reciting a textbook description of it.
- The patient is employed in a medical or hospital-related occupation.
- *Pseudologia fantastica*, a Latin phrase for "uncontrollable lying," is a condition in which the individual provides fantastic descriptions of events that never took place.
- The patient visits emergency rooms at times such as holidays or late Friday afternoons when experienced staff are not usually present and obtaining old medical records is difficult.
- The patient has few visitors even though he or she claims to be an important person.
- The patient is unusually accepting of surgery or uncomfortable diagnostic procedures.
- The patient's behavior is controlling, attention-seeking, hostile, or disruptive.
- The symptoms are present only when the patient thinks he or she is being watched.
- The patient is abusing substances, particularly prescription pain-killers or tranquilizers.
- The course of the "illness" fluctuates, or complications develop with unusual speed.
- The patient has multiple surgical scars, a so-called "gridiron abdomen," or evidence of self-inflicted wounds or injuries.

SYMPTOMS OF MUNCHAUSEN SYNDROME BY PROXY. Factors that suggest MSBP include:

- The victim is a young child; the average age of MSBP victims is 40 months.
- There is a history of long hospitalizations and frequent emergency room visits.
- Siblings have histories of MSBP, failure to thrive, or death in early childhood from an unexplained illness.
- The mother is employed in a health care profession.
- The mother has been diagnosed with depression or histrionic or borderline personality disorder.
- There is significant dysfunction in the family.

Demographics

The demographics of factitious disorder vary considerably across the different subtypes. Most individuals with the predominantly psychological subtype of FD are males with a history of hospitalizations beginning in late adolescence; few of these people, however, are older than 45. For non-chronic factitious disorder with predominantly physical symptoms, women outnumber men by a 3:1 ratio. Most of these women are between 20 and 40

years of age. Individuals with Munchausen syndrome are mostly middle-aged males who are unmarried and estranged from their families. Mothers involved in MSBP are usually married, educated, middle-class women in their early 20s.

Little is known about the rates of various subcategories of factitious disorder in different racial or ethnic groups.

The prevalence of factitious disorder worldwide is not known. In the United States, some experts think that FD is underdiagnosed because hospital personnel often fail to spot the deceptions that are symptomatic of the disorder. It is also not clear which subtypes of factitious disorders are most common. Most observers in developed countries agree, however, that the prevalence of factitious physical symptoms is much higher than the prevalence of factitious psychological symptoms. A large teaching hospital in Toronto reported that 10 of 1,288 patients referred to a consultation service had FD (0.8%). The National Institute for Allergy and Infectious Disease reported that 9.3% of patients referred for fevers of unknown origin had factitious disorder. A clinic in Australia found that 1.5% of infants brought in for serious illness by parents were cases of Munchausen syndrome by proxy.

Diagnosis

Diagnosis of factitious disorder is usually based on a combination of laboratory findings and the gradual exclusion of other possible diagnoses. In the case of MSBP, the abuse is often discovered through covert video surveillance.

The most important differential diagnoses, when factitious disorder is suspected, are malingering, **conversion disorder**, or another genuine psychiatric disorder.

Treatments

Medications

Medications have not proved helpful in treating factitious disorder by itself, although they may be prescribed for symptoms of anxiety or depression if the individual also meets criteria for an anxiety or mood disorder.

Psychotherapy

As of 2002, knowledge of the comparative effectiveness of different psychotherapeutic approaches is limited by the fact that few people diagnosed with FD remain in long-term treatment. In many cases, however, the factitious disorder improves or resolves if the individual receives appropriate therapy for a co-morbid psychiatric disorder. Ganser syndrome usually resolves completely with supportive **psychotherapy**.

One approach that has proven helpful in confronting patients with an examiner's suspicions is a supportive manner that focuses on the individual's emotional distress as the source of the illness rather than on the anger or righteous indignation of hospital staff. Although most individuals with FD refuse psychiatric treatment when it is offered, those who accept it appear to benefit most from supportive rather than insight-oriented therapy.

Family therapy is often beneficial in helping family members understand the individual's behavior and their need for attention.

Legal considerations

In dealing with cases of Munchausen syndrome by proxy, physicians and hospitals should seek appropriate legal advice. Although covert video surveillance of parents suspected of MSBP is highly effective (between 56% and 92%) in exposing the fraud, it may also be considered grounds for a lawsuit by the parents on grounds of entrapment. Hospitals can usually satisfy legal concerns by posting signs stating that they use hidden video monitoring.

All 50 states presently require hospitals and physicians to notify law enforcement authorities when MSBP is suspected, and to take steps to protect the child. Protection usually includes removing the child from the home, but it should also include an evaluation of the child's sibling(s) and long-term monitoring of the family. Criminal prosecution of one or both parents may also be necessary.

Prognosis

The prognosis of factitious disorder varies by subcategory. Males diagnosed with the psychological subtype of FD are generally considered to have the worst prognosis. Self-mutilation and **suicide** attempts are common in these individuals. The prognosis for Munchausen's syndrome is also poor; the statistics for recurrent episodes and successful suicides range between 30% and 70%. These individuals do not usually respond to psychotherapy. The prognosis for non-chronic FD in women is variable; some of these patients accept treatment and do quite well. This subcategory of FD, however, often resolves itself after the patient turns 40. MSBP involves considerable risks for the child; 9–10% of these cases end in the child's death.

Ganser syndrome is the one subtype of factitious disorder with a good prognosis. Almost all patients recover

within days of the diagnosis, especially if the **stress** that precipitated the syndrome is resolved.

Prevention

As of 2002, factitious disorder is not sufficiently well understood to allow for effective preventive strategies—apart from protection of child patients and their siblings in cases of MSBP.

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Rebecca J. Frey, Ph.D.

False belief of pregnancy *see* **Pseudocyesis**

Family education

Definition

Family education is the ongoing process of educating family members about a serious mental illness in order to improve their coping skills and their ability to help a relative affected by the illness.

Purpose

When someone is diagnosed with a chronic illness, such as diabetes or heart disease, efforts are typically made by his/her doctor not only to educate the individual directly affected by the illness, but to educate and involve his/her family in treatment and care. Historically, this has not been the case with severe mental illnesses such as **schizophrenia**, major depression, **bipolar disorder**, or **schizoaffective disorder**.

More often than not, mental health professionals did not educate families about what to expect or how to care for their loved one. In fact, for much of the twentieth century it was believed that mental illness was caused by overly strict or overly permissive parenting styles, and families were unfairly blamed for causing these disorders. Mothers were labeled "schizophrenogenic" and even well-meaning clinicians tried to keep them and other fam-

ily members at a distance. Bateson's "double-bind" theory suggested that contradictory messages and communications by parents were the root cause of the problem. Because of these ideas and the **stigma** associated with mental illness, families felt isolated and alone, with few resources to assist them. After **diagnosis**, the only recourse for most families was to go to public libraries to read and learn as much as they could on their own.

Over the last 20 years, advances in genetics, neuroscience, and imaging techniques have provided new evidence that severe mental illnesses are neurobiological in origin. With this scientific knowledge has come greater awareness and understanding that these are "no-fault" **brain** illnesses, and that neither families nor patients should be blamed. Rather, they both should receive the necessary information and support to help them better cope with these complex disorders.

Description

In the United States and elsewhere, the large majority of individuals with severe mental illness live with their families and depend on them for housing, financial assistance, advocacy, and support. For this reason families require knowledge and skills to actively help their relative avoid relapse, benefit from treatment, and achieve recovery. Specifically, family caregivers require information about: the illness and its symptoms, how to better communicate with their family member and professionals, pros and cons of different treatment options, medications and their therapeutic uses and their adverse side effects, signs of relapse, availability of community services and supports, how to access benefits and entitlements, and how to handle crises or bizarre and troubling behaviors. Because living with an individual with a serious mental disorder can be very stressful, family education must also focus on teaching families about the importance of taking care of themselves.

The National Alliance for the Mentally Ill (NAMI) is an umbrella organization of more than 1,100 local support and advocacy groups in 50 states. The organization comprises families and individuals affected by serious mental illness who come together for family education, mutual support, and advocacy. Through conferences, **support groups**, and newsletters, family members have opportunities to educate one another and exchange experiences. The organization has also made great inroads in educating mental health professionals about the importance of educating family members and involving them in plans for the patient's treatment and rehabilitation. On a more formal level, NAMI has sponsored "Family to Family" a 12-week education course that has been taught to 50,000 family members in more than 42 states. Taught by family volunteers, this is the first peer program in family education in the United States.

Family education is slowly becoming an integral part of treatment, as the recommendation for this has been incorporated into practice guidelines for professionals. Families are also making use of a new generation of books about mental illness—some written by professionals, and others written by, and for, family members. Families are also increasingly using the internet to learn more about mental disorders. The 2002 film, "A Beautiful Mind" was the story about Nobel-prize-winning mathematician John Nash, who suffered from schizophrenia. The film won an Oscar for best picture of the year. This reflects a changing attitude toward mental illness, which in the past was only discussed behind closed doors.

Family education for parents of children and adolescents

Because major mental illnesses tend to occur in adolescence or early adulthood, most family interventions focus on parents of adult children. However, any parent of a younger child with an emotional or behavioral disturbance can testify to the extraordinary challenges involved in coordinating care for their family member. For this reason, more public and private agencies are beginning to provide training, information, education, and financial assistance to family members of children and adolescents with emotional disturbances. The results of research about family education interventions for parents of children with serious emotional disturbances are just beginning to emerge. Some research suggests that family participation improves service delivery and patient outcomes for this group. In a randomized controlled trial of the training of 200 parents who did or did not receive training, while there were no significant effects on child mental health status, those family members who were trained showed significant knowledge enhancement and increased effectiveness.

Results

Research conducted over the past decade has provided evidence that family education and support leads to improved patient outcome. For example, **family psychoeducation** provided by mental health professionals, has such a compelling research base that it is considered a practice based on the findings of real-life studies of family education and support.

Another type of therapy discussed in the scientific literature has been used in China and India. The "family consultation" model uses individualized, private consultations between the family and a trained consultant to assist the family on an as-needed basis.

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Irene S. Levine, Ph.D.

Family psychoeducation

Definition

Family psychoeducation is a method based on clinical findings for training families to work together with mental health professionals as part of an overall clinical treatment plan for their family members. Family psychoeducation has been shown to improve patient outcomes for persons with **schizophrenia** and other major mental illnesses.

Purpose

The goal of family psychoeducation is to prevent patients with severe mental illnesses from relapsing, and to promote their re-entry into their home communities, with particular regard for their social and occupational functioning. To achieve that goal, family psychoeducation programs seek to provide families with the information they need about mental illness and the coping skills that will help them deal with their loved one's psychiatric disorder.

An associated goal of these programs is support for the patients' families. Families experience many burdens (financial, social, and psychological) in serving as long-term caregivers for their loved ones. Although the primary focus of family psychoeducation groups is improved patient outcomes, an essential intermediate goal is to promote the well-being of the family.

Description

There are several different models of family psychoeducation. Although they include many common elements, these different models include: single- and multiple-family groups; mixed groups that include family members and consumers (patients); groups of varying duration ranging from nine months to more than five years; and groups that focus on patients and families at different phases in the illness. Family psychoeducation programs have been studied extensively and refined by a number of researchers, including Drs. Ian Falloon, Gerald Hogarty, William McFarlane, and Lisa Dixon.

The evidence suggests that multi-family groups, which bring together several patients and their families, lead to better outcomes than single-family psychoeducation groups. The origins of multiple-family **group therapy** go back as far as 1960, when these groups were first assembled to solve ward-management problems in a psychiatric hospital. Lasting a minimum of nine months, the programs provided their participants with information about mental illness, its symptoms and treatment; med-

KEY TERMS

Burden—First described by Treudley in 1946, this term generally refers to the consequences for the family of close contact with a person who is severely mentally ill.

Meta-analysis—The statistical analysis of a large collection of analyses from individual studies for the purpose of integrating the findings.

ication and its side effects; how to communicate with a person with mental illness; and techniques for **crisis intervention** and mutual problem-solving.

Dixon recently outlined the characteristics of successful family psychoeducation programs. They include:

- The programs consider schizophrenia an illness like any other.
- They are led by mental health professionals.
- They are part of a total treatment plan that includes medication.
- Families are treated as partners rather than patients.
- The programs focus primarily on patient outcomes, secondarily on family outcomes.
- The programs differ from traditional **family therapy** in that they do not treat families as part of the problem; they see them as part of the solution.

It is also important that **family education** programs take into account the phase of the patient's illness; the life cycle of both the patient and the family; and the family's cultural context.

Results

A large body of evidence supports the use of family psychoeducation as a "best practice" for young adults with schizophrenia and their families. Because of this compelling evidence, researchers at the University of Maryland, as part of the Schizophrenia Patient Outcomes Research Team (PORT), identified family psychoeducation as an evidence-based practice that should be offered to all families. This and other research studies have shown reduced rates of relapse and lower rates of **hospitalization** among consumers and families involved in these programs. Other outcomes included increased rates of patient participation in **vocational rehabilitation** programs and employment; decreased costs of care; and improved well-being of family members.

A meta-analysis of 16 individual studies found that family interventions of fewer than 10 sessions have no effect on the reduction of family burden. There are also several controlled studies that support the effectiveness of single-and multiple-family interventions for **bipolar disorder**, major depression, **obsessive-compulsive disorder**, **anorexia nervosa**, and **borderline personality disorder**. Studies of family psychoeducation have been conducted outside the United States in China, Norway, and the Netherlands; and with a Hispanic population in Los Angeles, California.

Unfortunately, putting family psychoeducation into effect in clinical settings has not kept pace with research. The PORT study found that only 31% of patients studied reported that their families received information about their illness. One recent strategy to expand these programs includes integrating family psychoeducation into assertive community treatment (ACT) programs.

See also Case management

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Irene S. Levine, Ph.D.

Family therapists *see* **Marital and family therapists**

Family therapy

Definition

Family therapy is a form of **psychotherapy** that involves all the members of a nuclear or extended family. It may be conducted by a pair of therapists—often a man and a woman—to treat gender-related issues or serve as role models for family members. Although some types of family therapy are based on behavioral or psychodynamic principles, the most widespread form is based on family systems theory, an approach that regards the entire family as the unit of treatment, and emphasizes such factors as relationships and communication patterns rather than traits or symptoms in individual members.

Purpose

The purpose of family therapy is to identify and treat family problems that cause dysfunction. Therapy focuses on improvement in specific areas of functioning for each member, including communication and problem-solving skills.

Family therapy is often recommended when:

- A family member has schizophrenia or suffers from another severe **psychosis**; the goal in these cases is to help other family members understand the disorder and adjust to the psychological changes that may be occurring in the patient.
- Problems cross generational boundaries, such as when parents share a home with grandparents, or children are being raised by grandparents.
- Families deviate from social norms (unmarried parents, gay couples rearing children, etc.). These families may or may not have internal problems, but could be troubled by societal attitudes.
- Members come from mixed racial, cultural, or religious backgrounds.
- One member is being scapegoated, or their treatment in individual therapy is being undermined.
- The identified patient's problems seem inextricably tied to problems with other family members.
- A blended (i.e. step-) family is having adjustment difficulties.

Precautions

Before family therapy begins, family members are required to undergo a comprehensive clinical evaluation (interview) that includes questions of a personal and sensitive nature. Honest communication between the family members and the therapist is essential; people who are not willing to to discuss and change behaviors may not benefit from therapy.

Families that may not be considered suitable candidates for family therapy include those in which:

- One or both parents is psychotic or has been diagnosed with antisocial or paranoid personality disorder.
- Cultural or religious values are opposed to, or suspicious of, psychotherapy.
- Some family members cannot participate in treatment sessions because of illness or other physical limitations.
- Individuals have very rigid personality structures and might be at risk for an emotional or psychological crisis.
- Members cannot or will not be able to meet regularly for treatment.
- The family is unstable or on the verge of break-up.

Intensive family therapy may be difficult for psychotic family members.

Description

Family therapy is a relatively recent development in psychotherapy. It began shortly after World War II, when doctors who were treating schizophrenic patients noticed that the patients' families communicated in disturbed ways. The doctors also found that patients' symptoms rose or fell according to the level of tension between their parents. These observations led to considering a family as an organism (or system) with its own internal rules, patterns of functioning, and tendency to resist change. When the therapists began to treat the families as whole units instead of focusing solely on the hospitalized member, they found that in many cases the schizophrenic family member improved. (This does not mean that **schizophrenia** is caused by family problems, although they may aggravate its symptoms.) This approach was then applied to families

with problems other than schizophrenia. Family therapy is becoming an increasingly common form of treatment as changes in American society are reflected in family structures; it is also helpful when a child or other family member develops a serious physical illness.

Family therapy tends to be short term, usually several months in length, aimed at resolving specific problems such as eating disorders, difficulties with school, or adjustments to bereavement or geographical relocation. It is not normally used for long-term or intensive restructuring of severely dysfunctional families.

In therapy sessions, all members of the family and both therapists (if there is more than one) are present. The therapists try to analyze communication and interaction between all members of the family; they do not side with specific members, although they may make occasional comments to help members become more conscious of patterns previously taken for granted. Therapists who work as a team also model new behaviors through their interactions with each other.

Family therapy is based on systems theory, which sees the family as a living organism that is more than the sum of its individual members and evaluates family members in terms of their position or role within the system. Problems are treated by changing the way the system works rather than trying to "fix" a specific member.

Family systems theory is based on several major concepts:

The identified patient

The identified patient (IP) is the family member with the symptom that has brought the family into treatment. The concept of the IP is used to keep the family from scapegoating the IP or using him or her as a way of avoiding problems in the rest of the system.

Homeostasis

This concept presumes that the family system seeks to maintain its customary organization and functioning over time. It tends to resist change. The family therapist can use homeostasis to explain why a certain family symptom has surfaced at a given time, why a specific member has become the IP, and what is likely to happen when the family begins to change.

The extended family field

The extended family field is the nuclear family plus the network of grandparents and other members of the extended family. This concept is used to explain the intergenerational transmission of attitudes, problems, behaviors, and other issues.

Differentiation

Differentiation refers to each family member's ability to maintain his or her own sense of self while remaining emotionally connected to the family; this is the mark of a healthy family.

Triangular relationships

Family systems theory maintains that emotional difficulties in families are usually triangular—whenever any two persons have problems with each other, they will "triangle in" a third member to stabilize their own relationship. These triangles usually interlock in a way that maintains homeostasis. Common family triangles include a child and its parents; two children and one parent; a parent, a child, and a grandparent; three siblings; or, husband, wife, and an in-law.

Preparation

Families are often referred to a specialist by a pediatrician or other primary care provider. (Some estimates suggest that as many as 50% of pediatric office visits concern developmental problems in children that are affecting their families.) Physicians may use symptom checklists or psychological screeners to assess a family's need for therapy.

Family therapists can be psychiatrists, clinical psychologists, or other professionals certified by a specialty board in marriage and family therapy. They will usually evaluate a family for treatment by scheduling a series of interviews with members of the immediate family, including young children, as well as significant or symptomatic members of the extended family. This allows the therapist(s) to learn how each family member sees the problem and provides a first impression of the family's functioning. Therapists typically evaluate the level and types of emotions expressed, patterns of dominance and submission, roles played by family members, communication styles, and the existence of emotional triangles. They also note whether these patterns are rigid or relatively flexible.

Preparation also usually includes creating a genogram, a diagram that depicts significant persons and events in the family's history. They include annotations about the medical history and major personality traits of each member and help uncover intergenerational patterns of behavior, marriage choices, family alliances and conflicts, the existence of family secrets, and other information that sheds light on the family's present situation.

Risks

There are no major risks involved in receiving family therapy, especially if family members seek the therapy with honesty, openness, and a willingness to change. Changes that result from the therapy may be seen by some as "risks"—the possible unsettling of rigid personality defenses in individuals, or the unsettling of couple relationships that had been fragile before the beginning of therapy, for example.

Normal results

The goal of therapy is the identification and resolution of the problem that is causing the family's unhealthy interactions. Results vary, but in good circumstances they include greater insight, increased differentiation of individual family members, improved communication within the family, and loosening of previously automatic behavior patterns.

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Rebecca J. Frey, Ph.D.

Fatigue

Introduction

Fatigue may be defined as a subjective state in which one feels tired or exhausted, and in which the capacity for normal work or activity is reduced. There is, however, no commonly accepted definition of fatigue when it is considered in the context of health and illness. This lack of definition results from the fact that a person's experience of fatigue depends on a variety of factors. These factors include culture; personality; the physical environment (light, noise, vibration); availability of social support through networks of family members and friends; the nature of a particular fatiguing disease or disorder; and the type and duration of work or exercise. For example, the experience of fatigue associated with disease will be different for someone who is clinically depressed, is socially isolated, and is out of shape, as compared to another person who is not depressed, has many friends, and is aerobically fit.

Fatigue is sometimes characterized as normal or abnormal. For example, the feeling of tiredness or even exhaustion after exercising is a normal response and is relieved by resting; many people report that the experience of ordinary tiredness after exercise is pleasant. Moreover, this type of fatigue is called *acute* since the onset is sudden and the desired activity level returns after resting. On the other hand, there is a kind of fatigue that is not perceived as ordinary; that may develop insidiously over time; is unpleasant or seriously distressing; and is not resolved by rest. This kind of fatigue is abnormal and is called *chronic*.

Some researchers regard fatigue as a defense mechanism that promotes the effective regulation of energy expenditures. According to this theory, when people feel tired they take steps to avoid further **stress** (physical or emotional) by resting or by avoiding the stressor. They are then conserving energy. Since chronic fatigue is not normal, however, it is an important symptom of some mental disorders; of a variety of physical diseases with known etiologies (causes); and of medical conditions that have no biological markers although they have recognizable syndromes (patterns of symptoms and signs).

Fatigue is sometimes described as being primary or secondary. Primary fatigue is a symptom of a disease or mental disorder, and may be part of a cluster of such symptoms as pain, fever, or nausea. As the disease or disorder progresses, however, the fatigue may be intensified by the patient's worsening condition, by the other disease symptoms, or by the surgical or medical treatment given to the patient. This subsequent fatigue is called secondary.

KEY TERMS

Biological marker—An indicator or characteristic trait of a disease that facilitates differential diagnosis (the process of distinguishing one disorder from other, similar disorders).

Deconditioning—Loss of physical strength or stamina resulting from bed rest or lack of exercise.

Electrolytes—Substances or elements that dissociate into electrically charged particles (ions) when dissolved in the blood. The electrolytes in human blood include potassium, magnesium, and chloride.

Metabolism—The group of biochemical processes within the body that release energy in support of life.

Stress—A physical and psychological response that results from being exposed to a demand or pressure.

Syndrome—A group of symptoms that together characterize a disease or disorder.

Risk factors

Fatigue is a common experience. It is one of the top ten symptoms that people mention when they visit the doctor. Some people, however, are at higher risk for developing fatigue. For example, the risk for women is about 1.5 times the risk for men, and the risk for people who don't exercise is twice that of active people. Some researchers question whether women really are at higher risk, since they are more likely than men to go to the doctor with health problems; also, men are less likely to admit they feel fatigued. Other risk factors include **obesity**; smoking; use of alcohol; high stress levels; depression; anxiety; and low blood pressure. Having low blood pressure is usually considered desirable in the United States, but is regarded as a treatable condition in other countries. Low blood pressure or postural hypotension (sudden lowering of blood pressure caused by standing up) may cause fatigue, dizziness, or fainting.

Major sources of chronic fatigue

Disease

There are many diseases and disorders in which fatigue is a major symptom— for example, cancer, cardiovascular disease, emphysema, multiple sclerosis, rheumatic arthritis, systemic lupus erythematosus, HIV/AIDS, infectious mononucleosis, chronic fatigue syndrome, and fibromyalgia. The reasons for the fatigue, however, vary according to the organ system or body function affected by the disease. Physical reasons for fatigue include:

- Circulatory and respiratory impairment. When the patient's breathing and blood circulation are impaired, or when the patient has anemia (low levels of red blood cells), body tissues do not receive as much oxygen and energy. Hence, the patient experiences a general sense of fatigue. Fatigue is also an important warning sign of heart trouble because it precedes 30%–55% of myocardial infarctions (heart attacks) and sudden cardiac deaths.
- Infection. Microorganisms that disturb body metabolism and produce toxic wastes cause disease and lead to fatigue. Fatigue is an early primary symptom of chronic, nonlocalized infections found in such diseases as acquired immune deficiency syndrome (AIDS), Lyme disease, and tuberculosis.
- Nutritional disorders or imbalances. Malnutrition is a disorder that promotes disease. It is caused by insufficient intake of important nutrients, vitamins, and minerals; by problems with absorption of food through the digestive system; or by inadequate calorie consumption. Protein-energy malnutrition (PEM) occurs when people do not consume enough protein or calories; this condition leads to wasting of muscles and commonly occurs in developing countries. In particular, young children who are starving are at risk of PEM, as are people recovering from major illness. In general, malnutrition damages the body's immune function and thereby encourages disease and fatigue. Taking in too many calories for the body's needs, on the other hand, results in obesity, which is a predictor of many diseases related to fatigue.
- Dehydration. Dehydration results from water and sodium imbalances in body tissues. The loss of total body water and sodium may be caused by diarrhea, vomiting, bed rest, exposure to heat, or exercise. Dehydration contributes to muscle weakness and mental confusion; it is a common and overlooked source of fatigue. Once fatigued, people are less likely to consume enough fluids and nutrients, which makes the fatigue and confusion worse.
- Deconditioning. This term refers to generalized organ system deterioration resulting from bed rest and lack of exercise. In the 1950s and 1970s, the National Aeronautics and Space Administration (NASA) studied the effects of bed rest on healthy athletes. The researchers found that deconditioning set in quite rapidly (within 24 hours) and led to depression and weakness. Even mild exercise can counteract deconditioning, however, and has become an important means of

minimizing depression and fatigue resulting from disease and hospitalization.

- Pain. When pain is severe enough, it may disrupt sleep and lead to the development of such **sleep disorders** as **insomnia** or **hypersomnia**. Insomnia is the term for having difficulty falling and/or staying asleep. Hypersomnia refers to excessive sleeping. In general, disrupted sleep is not restorative; people wake up feeling tired, and as a result their pain is worsened and they may become depressed. Furthermore, pain may interfere with movement or lead to too much bed rest, which results in deconditioning. Sometimes pain leads to social isolation because the person cannot cope with the physical effort involved in maintaining social relationships, or because family members are unsympathetic or resentful of the ill or injured person's reduced capacity for work or participation in family life. All of these factors worsen pain, contributing to further sleep disruption, fatigue, and depression.
- Stress. When someone experiences ongoing pain and stress, organ systems and functional processes eventually break down. These include cardiovascular, digestive, and respiratory systems, as well as the efficient elimination of body wastes. According to the American Psychiatric Association, various chronic diseases are related to stress, including regional enteritis (intestinal inflammation); ulcerative colitis (a disease of the colon); gastric ulcers; rheumatoid arthritis; cardiac angina, and dysmenorrhea (painful menstruation). These diseases deplete the body's levels of serotonin (a neurotransmitter important in the regulation of sleep and wakefulness, as well as depression), and endorphins (opiate-like substances that moderate pain). Depletion of these body chemicals leads to insomnia and chronic fatigue.
- Sleep disorders. There are a variety of sleep disorders that cause fatigue, including insomnia, hypersomnia, sleep apnea, and restless legs syndrome. For example, hypersomnia may be the result of **brain** abnormalities caused by viral infections. Researchers studying the aftermath of infectious mononucleosis proposed that exposure to viral infections might change brain function with the effect of minimizing restorative sleep; hence, some people developed hypersomnia. Another common disorder is sleep apnea, in which the patient's breathing stops for at least ten seconds, usually more than 20 times per hour. Snoring is common. People may experience choking and then wake up gasping for air; they may develop daytime hypersomnia (daytime sleepiness) to compensate. Sleep apnea is associated with aging, weight gain, and depression. It is also a risk factor for **stroke** and myocardial infarctions. Restless legs syndrome is a condition in which very uncomfortable sensations in the patient's legs cause them to move and wake up from sleep, or keep them from falling asleep. All of these disorders reduce the quality of a person's sleep and are associated with fatigue.

Fibromyalgia and chronic fatigue syndrome

Fibromyalgia (also known as myofascial syndrome or fibrositis) is characterized by pain and achiness in muscles, tendons, and ligaments. There are 18 locations on the body where patients typically feel sore. These locations include areas on the lower back and along the spine, neck, and thighs. A diagnostic criterion for fibromyalgia (FM) is that at least 11 of the 18 sites are painful. In addition to pain, people with FM may experience sleep disorders, fatigue, anxiety, and irritable bowel syndrome. Some researchers maintain, however, that when fatigue is severe, chronic, and persistent, FM is indistinguishable from chronic fatigue syndrome (CFS). The care that patients receive for FM or CFS depends in large measure on whether they were referred to a rheumatologist (a doctor who specializes in treating diseases of the joints and muscles), neurologist, or **psychiatrist**.

Some doctors do not accept CFS (also known as myalgic encephalomyelitis in Great Britain) as a legitimate medical problem. This refusal is stigmatizing and distressing to the person who must cope with disabling pain and fatigue. It is not uncommon for people with CFS to see a number of different physicians before finding one who is willing to diagnose CFS. Nevertheless, major health agencies, such as the Centers for Disease Control (CDC) in the United States, have studied the syndrome. As a result, a revised CDC case definition for CFS was published in 1994 that lists major and minor criteria for **diagnosis**. The major criteria of CFS include the presence of chronic and persistent fatigue for at least six months; fatigue that does not improve with rest; and fatigue that causes significant interference with the patient's daily activities. Minor criteria include such flu-like symptoms as fever; sore throat; swollen lymph nodes; myalgia (muscle pain); difficulty with a level of physical exercise that the patient had performed easily before the illness; sleep disturbances; and headaches. Additionally, people often have difficulty concentrating and remembering information; they experience extreme frustration and depression as a result of the limitations imposed by CFS. The prognosis for recovery from CFS is poor, although the symptoms are manageable.

Psychological disorders

While fatigue may be caused by many organic diseases and medical conditions, it is a chief complaint for several mental disorders, including **generalized anxiety**

disorder and clinical depression. Moreover, mental disorders may coexist with physical disease. When there is considerable symptom overlap, the differential diagnosis of fatigue is especially difficult.

GENERALIZED ANXIETY DISORDER. People are diagnosed as having generalized anxiety disorder (GAD) if they suffer from overwhelming worry or apprehension that persists, usually daily, for at least six months; and if they also experience some of the following symptoms: unusual tiredness, restlessness and irritability, problems with concentration, muscle tension, and disrupted sleep. Such stressful life events as divorce, unemployment, illness, or being the victim of a violent crime are associated with GAD, as is a history of psychiatric problems. Some evidence suggests that women who have been exposed to danger are at risk of developing GAD; women who suffer loss are at risk of developing depression, and women who experience danger and loss are at risk of developing a mix of both GAD and depression.

While the symptoms of CFS and GAD overlap, the disorders have different primary complaints. Patients with CFS complain primarily of tiredness, whereas people with GAD describe being excessively worried. In general, some researchers believe that anxiety contributes to fatigue by disrupting rest and restorative sleep.

DEPRESSION. In the fourth edition of the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV*), the presence of depressed mood or sadness, or loss of pleasure in life, is an important diagnostic criterion for depression. Daily fatigue, lack of energy, insomnia and hypersomnia are indicators of a depressed mood. The symptoms of depression overlap with those of CFS; for example, some researchers report that 89% of people with depression are fatigued, as compared to 86%–100% of people with CFS. The experience of fatigue, however, seems to be more disabling with CFS than with depression. Another difference between CFS and depression concerns the onset of the disorder. Most patients with CFS experience a sudden or acute onset, whereas depression may develop over a period of weeks or months. Also, while both types of patients experience sleep disorders, CFS patients tend to have difficulty falling asleep, whereas depressed patients tend to wake early in the morning.

Some researchers believe that there is a link between depression, fatigue, and exposure to too much REM sleep. There are five distinct phases in human sleep. The first two are characterized by light sleep; the second two by a deep restorative sleep called slow-wave sleep; and the last by rapid eye movement or REM sleep. Most dreams occur during REM sleep. Throughout the night, the intervals of REM sleep increase and usually peak around 8:30 A.M. A sleep deprivation treatment for depression involves reducing the patient's amount of REM sleep by waking him or her around 6:00 A.M. Researchers think that some fatigue associated with disease may be a form of mild depression and that reducing the amount of REM sleep will reduce fatigue by moderating depression.

Managing fatigue

The management of fatigue depends in large measure on its causes and the person's experience of it. For example, if fatigue is acute and normal, the person will recover from feeling tired after exertion by resting. In cases of fatigue associated with influenza or other infectious illnesses, the person will feel energy return as they recover from the illness. When fatigue is chronic and abnormal, however, the doctor will tailor a treatment program to the patient's needs. There are a variety of approaches that include:

- Aerobic exercise. Physical activity increases fitness and counteracts depression.
- Hydration (adding water). Water improves muscle turgor or tension and helps to carry electrolytes.
- Improving sleep patterns. The patient's sleep may be more restful when its timing and duration are controlled.
- Pharmacotherapy (treatment with medications). The patient may be given various medications to treat physical diseases or mental disorders; to control pain; or to manage sleeping patterns.
- Psychotherapy. There are several different treatment approaches that help patients manage stress; understand the motives that govern their behavior; or change maladaptive ideas and negative thinking patterns.
- Physical therapy. This form of treatment helps patients improve or manage functional impairments or disabilities.

In addition to seeking professional help, people can understand and manage fatigue by joining appropriate **self-help groups**; reading informative books; seeking information from clearinghouses on the Internet; and visiting web sites maintained by national organizations for various diseases.

See also Brain; Breathing-related sleep disorder; Caffeine and related sleep disorders; Circadian rhythm sleep disorder; Pain disorder; Self-help groups; Somatization and somatoform disorders

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National Chronic Fatigue Syndrome and Fibromyalgia Association. P.O. Box 18426, Kansas City, MO 64133. (816) 313-2000. <http://www.4woman.gov/nwhic/references/mdreferrals/ncfsfa.htm>.

Tanja Bekhuis, Ph.D.

Feeding disorder of infancy or early childhood

Definition

Feeding disorder of infancy or early childhood is characterized by the failure of an infant or child under six years of age to eat enough food to gain weight and grow normally over a period of one month or more. The disorder can also be characterized by the loss of a significant amount of weight over one month. Feeding disorder is similar to failure to thrive, except that no medical or physiological condition can explain the low food intake or lack of growth.

Description

Infants and children with a feeding disorder fail to grow adequately, or even lose weight with no underlying medical explanation. They do not eat enough energy or nutrients to support growth and may be irritable or apathetic. Factors that contribute to development of a feeding disorder include lack of nurturing, failure to read the child's hunger and satiety cues accurately, poverty, or parental mental illness. Successful treatment involves dietary, behavioral, social, and psychological **intervention** by a multidisciplinary team of health professionals.

Causes and symptoms

Causes

Feeding disorder of infancy or early childhood can occur with inappropriate parent-child interactions, such as failure to read the child's hunger cues or forcing food when the child is not hungry. Lack of nurturing and/or parental aggression, anger, or **apathy** can make eating a negative experience for the child, increasing the risk of feeding disorders.

Feeding disorders are more common in infants and children who are born prematurely, had a low birth weight, or who are developmentally delayed. Many medical (or physiological) causes can contribute to eating difficulties, eating aversions, or failure to thrive, including:

- diseases of the central nervous system
- metabolic diseases
- sensory defects
- anatomical abnormalities, such as cleft palate
- muscular disorders, such as cerebral palsy
- heart disease
- gastrointestinal diseases, such as Crohn's disease

To meet criteria for a true feeding disorder of infancy or childhood, these medical conditions must be ruled out.

Symptoms

Because the child or infant with a feeding disorder is not consuming enough energy, vitamins, or minerals to support normal growth, symptoms resemble those seen in malnourished or starving children. The infant or child may be irritable, difficult to console, apathetic, withdrawn, and unresponsive.

Delays in development, as well as growth, can occur. In general, the younger the child, the greater the risk of developmental delays associated with the feeding disorder.

Laboratory abnormalities may also be associated with the disorder. Blood tests may reveal a low level of protein or hemoglobin in the blood. Hemoglobin is an iron-containing substance in blood that carries oxygen to body cells.

This mother is consoling her new baby. Infants who are not nurtured, or whose caregiver becomes angry or are apathetic at feedings are more likely to develop feeding disorders. *(Laura Dwight/CORBIS. Reproduced by permission.)*

Demographics

Although minor feeding problems are common in infancy and childhood, true feeding disorder of infancy or early childhood is estimated to occur in 1% to 3% of infants and children. Children separated from their families or living in conditions of poverty or **stress** are at greater risk. Mental illness in a parent, or child **abuse** or **neglect**, may also increase the risk of the child developing a feeding disorder.

Diagnosis

Between 25% and 35% of normal children experience minor feeding problems. In infants born prematurely, 40% to 70% experience some type of feeding problem. For a child to be diagnosed with feeding disorder of infancy or early childhood, the disorder must be severe enough to affect growth for a significant period of time. Generally, growth failure is considered to be below the fifth percentile of weight and height.

Feeding disorder of infancy or early childhood is diagnosed if all four of the following criteria are present:

- Failure to eat adequately over one month or more, with resultant weight loss or failure to gain weight.
- Inadequate eating and lack of growth not explained by any general medical or physiological condition, such as gastrointestinal problems, nervous system abnormalities, or anatomical deformations.
- The feeding disorder cannot be better explained by lack of food or by another mental disorder, such as rumination disorder.
- The inadequate eating and weight loss or failure to gain weight occurs before the age of six years. If feeding behavior or weight gain improves when another person feeds and cares for the child, the existence of a true feeding disorder, rather than some underlying medical condition, is more likely.

Treatments

Successful treatment of feeding disorders requires a multidisciplinary team approach to assess the child's needs and to provide recommendations and education to improve feeding skills, behavior, and nutrient intake. The multidisciplinary team for treatment of feeding disorders in childhood usually includes physicians specializing in problems of the gastrointestinal tract or of the ear, nose, and throat; a dietitian, a **psychologist**, a speech pathologist, and an occupational therapist. Support from **social workers** and physicians in related areas of medicine is also helpful.

An initial evaluation should focus on feeding history, including detailed information on type and timing of food intake, feeding position, meal duration, energy and nutrient intake, and behavioral and parental factors that influence the feeding experience. Actual observation of a feeding session can give valuable insight into the cause of the feeding disorder and appropriate treatments. A medical examination should also be conducted to rule out any potential medical problems or physical causes of the feeding disorder.

After a thorough history is taken and assessment completed, dietary and behavioral therapy is started. The goal of diet therapy is to gradually increase energy and nutrient intake as tolerated by the child to allow for catch up growth. Depending on the diet history, energy and nutrient content of the diet may be kept lower initially to avoid vomiting and diarrhea. As the infant or child is able to tolerate more food, energy and nutrient intake is gradually increased over a period of one to two weeks, or more. Eventually, the diet should provide about 50% more than normal nutritional needs of infants or children of similar age and size.

Behavioral therapy can help the parent and child overcome conditioned feeding problems and food aversions. Parents must be educated to recognize their child's hunger and satiety cues accurately and to promote a pleasant, positive feeding environment. Changing the texture of foods, the pace and timing of feedings, the position of the body, and even feeding utensils can help the child overcome aversions to eating. If poverty, abuse, or parental mental illness contribute to the feeding disorder, these issues must also be addressed.

Prognosis

If left untreated, infants and children with feeding disorders can have permanent physical, mental, and behavioral damage. However, most children with feeding disorders show significant improvements after treatment, particularly if the child and parent receive intensive nutritional, psychological, and social intervention.

Prevention

Providing balanced, age-appropriate foods at regular intervals—for example, three meals and two or three snacks daily for toddlers—can help to establish healthy eating patterns. If a child is allowed to fill up on soft drinks, juice, chips, or other snacks prior to meals, appetite for other, more nutritious foods will decrease.

Positive infant and childhood feeding experiences require the child to communicate hunger and satiety effectively and the parent or caregiver to interpret these signals accurately. This set of events requires a nurturing environment and an attentive, caring adult. Efforts should be made to establish feeding as a positive, pleasant experience. Further, forcing a child to eat or punishing a child for not eating should be avoided.

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Female orgasmic disorder

Definition

Female orgasmic disorder (FOD) is the persistent or recurrent inability of a woman to have an orgasm (climax or sexual release) after adequate sexual arousal and sexual stimulation. According to the handbook used by mental health professionals to diagnose mental disorders, the ***Diagnostic and Statistical Manual of Mental Disorders***, 4th Edition, Text Revision (also known as the *DSM-IV-TR)*, this lack of response can be primary (a woman has never had an orgasm) or secondary (acquired after trauma), and can be either general or situation-specific. There are both physiological and psychological causes for a woman's inability to have an orgasm. To receive the **diagnosis** of FOD, the inability to have an orgasm must not be caused only by physiological problems or be a symptom of another major mental health problem. FOD may be diagnosed when the disorder is caused by a combination of physiological and psychological difficulties. To be considered FOD, the condition must cause personal distress or problems in a relationship. In earlier versions of the *Diagnostic and Statistical Manual of Mental Disorders,* FOD was called "inhibited sexual orgasm."

Description

FOD is the persistent or recurrent inability of a woman to achieve orgasm. This lack of response affects the quality of the woman's sexual experiences. To understand FOD, it is first necessary to understand the physiological changes that normally take place in a woman's body during sexual arousal and orgasm.

Normally, when a woman is sexually excited, the blood vessels in the pelvic area expand, allowing more blood to flow to the genitals. This is followed by the seepage of fluid out of blood vessels and into the vagina to provide lubrication before and during intercourse. These events are called the "lubrication-swelling response."

Body tension and blood flow to the pelvic area continue to build as a woman receives more sexual stimulation; this occurs either by direct pressure on the clitoris or as pressure on the walls of the vagina and cervix. This tension builds as blood flow increases. When tension is released, pleasurable rhythmic contractions of the uterus and vagina occur; this release is called an "orgasm." The contractions carry blood away from the genital area and back into general circulation.

It is normal for orgasms to vary in intensity, length, and number of contractions from woman to woman, as

KEY TERMS

Cervix—The neck or narrow lower end of a woman's uterus.

Clitoris—The most sensitive area of the external genitals. Stimulation of the clitoris causes most women to reach orgasm.

Uterus—The hollow muscular sac in which a fetus develops; sometimes called the womb.

Vagina—The part of the female reproductive system that opens to the exterior of the body and into which the penis is inserted during sexual intercourse.

well as in a single individual from experience to experience. Unlike men, woman can have multiple orgasms in a short period of time. Mature women, who may be more sexually experienced than younger women, may find it easier to have orgasms than adolescents or the sexually inexperienced.

In FOD, sexual arousal and lubrication occur. Body tension builds, but the woman is unable or has extreme difficulty reaching climax and releasing the tension. This inability can lead to frustration and unfulfilling sexual experiences for both partners. FOD often occurs in conjunction with other **sexual dysfunctions**. Also, lack of orgasm can cause anger, frustration, and other problems in the relationship.

Causes and symptoms

With FOD, a woman either does not have an orgasm or has extreme difficulty regularly reaching climax. It is normal for women to lack this response occasionally, or to have an orgasm only with specific types of stimulation. The occasional failure to be reach orgasm or dependence on a particular type of stimulation is not the same as FOD.

The causes of FOD can be both physical and psychological. FOD is most often a primary or lifelong disorder, meaning that a woman has never achieved orgasm under any type of stimulation, including self-stimulation (masturbation), direct stimulation of the clitoris by a partner, or vaginal intercourse. Some women experience secondary, or acquired FOD. These women have had orgasms, but lose the ability after illness, emotional trauma, or as a side effect of surgery or medication. Acquired FOD is often temporary.

FOD can be generalized or situation-specific. In generalized FOD, the failure to have an orgasm occurs with different partners and in many different settings. In situational FOD, inability to reach climax occurs only with specific partners or under particular circumstances. FOD may be due either to psychological factors or a combination of physiological and psychological factors, but not due to physiological factors alone.

Physiological causes of FOD include:

- damage to the blood vessels of the pelvic region
- spinal cord lesions or damage to the nerves in the pelvic area
- side effects of medications (antipsychotics, antidepressants, narcotics) or illicit substance abuse
- removal of the clitoris (also called female genital mutilation, a cultural practice in parts of Africa, the Middle East, and Asia)

Psychological causes of FOD include:

- past sexual abuse, rape, incest, or other traumatic sexual experience
- emotional abuse
- fear of becoming pregnant
- fear of rejection by partner
- fear of loss of control during orgasm
- self-image problems
- relationship problems with partner
- life stresses, such as financial worries, job loss, or divorce
- guilt about sex or sexual pleasure
- religious or cultural beliefs about sex
- other mental health disorders such as major depression

FOD is more likely to have a psychological, rather than a physical cause. Inadequate time spent in foreplay, inadequate arousal, lack of appropriate sexual stimulation, poor sexual communication with a partner, and failure to continue with stimulation for an adequate length of time may cause failure to climax, but are not considered causes of FOD.

Demographics

Inability to have an orgasm, discontent with the quality of orgasms, and the ability to have orgasms only with one type of stimulation are common sexual complaints among women. Some studies have found that about half of all women experience some orgasmic difficulties, but not of all these difficulties are considered FOD. About 50% of women experience orgasm through direct clitoral stimulation but not during intercourse, thus not meeting the criteria for a diagnosis of FOD. About 10% of women

never experience an orgasm, regardless of the situation or stimulation. These women are more likely to be unmarried, young, and sexually inexperienced.

Diagnosis

FOD is diagnosed through a medical and psychological history and history of the conditions under which orgasm fails to occur. It is especially helpful for the clinician or sex therapist to understand how long the problem has persisted, and whether it is general or situational. FOD is sometimes found in conjunction with **sexual aversion disorder** and **female sexual arousal disorder**, making the diagnosis complex. To be diagnosed with FOD, the lack of orgasmic response must occur regularly over an extended period of time; based on the clinician's judgment, it must be less than would be reasonable based on age, sexual experience, and the adequacy of sexual stimulation. The lack of orgasm must cause emotional distress or relationship difficulties for the woman and be caused either only by psychological factors alone or by a combination of psychological and physical factors. According to the American Psychiatric Association (APA), a diagnosis of FOD is not appropriate if failure to climax is due only to physiological factors. FOD is also not diagnosed if it is a symptom of another major psychological disorder, such as depression.

Treatments

When failure to reach orgasm is caused by a physical problem, the root problem is treated. In other cases, a combination of education, counseling, **psychotherapy**, and sex therapy are used—often along with directed exercises to increase stimulation and decrease inhibitions—either for the individual or for the couple. As of 2002, clinical trials are under way to investigate the effect of sildenafil (Viagra) on women's sexual response. Sildenafil has already been proved effective in helping men to attain and maintain an erection.

Sex therapists have special training to help individuals and couples focus on overcoming specific sexual dysfunctions. In **couples therapy**, they often assign "homework" that focuses on relaxation techniques, sexual exploration, improving sexual communication, decreasing inhibitions, and increasing direct clitoral stimulation. Individually, a woman might be encouraged to masturbate either through self-stimulation or with a vibrator. In addition, Kegel exercises, which improve the strength and tone of the muscles in the genital area, may be recommended.

Traditional psychotherapy, or **talk therapy** alone or in conjunction with sex therapy, can be effective in resolving psychological causes of FOD, especially when those causes are rooted in past sexual or emotional exploitation or cultural taboos. Psychotherapy is also helpful in resolving relationship tensions that develop as a result of frustration from FOD.

Prognosis

Many women with FOD can be helped to achieve orgasm through a combination of psychotherapy and guided sexual exercises. However, this does not mean that they will be able to achieve orgasm all the time or in every situation, or that they will always be satisfied with the strength and quality of their climax. Couples often need to work through relationship issues that have either caused or resulted from FOD before they see improvement. This process takes time and requires a joint commitment to problem solving.

Prevention

There are no sure ways to prevent FOD. However, reducing life factors that cause **stress** can be effective. Seeking counseling or psychotherapy for past trauma, or when problems begin to appear in a relationship, can help minimize sexual dysfunction problems.

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American Association of Sex Educators, Counselors, and Therapists (AASECT). P. O. Box 238, Mount Vernon, IA 53214-0238. (319) 895-8407. <www.aasect.org>.

Sexual Information and Education Council of the United States (SIECUS). West 42nd Street, Suite 350, New York, NY 10036-7802. <www.siecus.org>.

Tish Davidson, A.M.

Female sexual arousal disorder

Definition

Female sexual arousal disorder (FSAD) refers to the persistent or recurrent inability of a woman to achieve or maintain an adequate lubrication-swelling response during sexual activity. This lack of physical response may be either lifelong or acquired, and either generalized or situation-specific. FSAD has both physiological and psychological causes. The results of FSAD are often sexual avoidance, painful intercourse, and sexual tension in relationships.

Description

FSAD results from the body's inability to undergo specific physiological changes, called the lubrication-swelling response, in response to sexual desire and stimulation. This lack of response then affects the woman's desire for and satisfaction obtained from intercourse. To understand FSAD, it is helpful to have an outline of the physiological changes that normally take place in a woman's body during sexual arousal.

William Masters and Virginia Johnson were the first researchers to examine extensively the physical components of human sexual arousal. They recorded four stages of sexual response: excitement, plateau, climax (or orgasm), and resolution. Since then, other models have been suggested that include the emotional aspects of arousal. One model suggests three stages: desire, arousal, and orgasm. FSAD affects the excitement or arousal stage of sexual activity.

Normally, when a woman is aroused and sexually excited, the first physiological change that she experiences is expansion of the blood vessels in the pelvic region, allowing more blood to flow to her lower abdomen and genitals. Some women notice this as a feeling of fullness in the pelvis and either consciously or involuntarily contract the muscles in the genital area.

The increased blood flow also causes a phenomenon called transudation, which refers to the seepage of fluid through the walls of the blood vessels. In this case, the fluid seeps into the vagina to provide lubrication before and during intercourse. Often this moisture is noticeable to the woman and her partner. Lubrication of the vagina can happen very rapidly, within a minute.

The increase in blood flow produces other changes in the tissues of the female genitals. The upper part of the vagina, the uterus, the cervix, and the clitoris all expand. At the same time, the lower third of the vagina and the outer labia swell, so that the opening to the vagina becomes smaller. The inner labia also swell, and push apart the opening to the vagina. These changes taken together make up the lubrication-swelling response and are designed to facilitate the entry of the penis into the vagina.

A woman with FSAD either does not have these physical responses or does not maintain them through completion of sexual activity. The lack of arousal and lubrication may result in painful intercourse (**dyspareunia**), emotional distress, or relationship problems.

Causes and symptoms

The symptoms of FSAD include lack of or insufficient transudation. A woman diagnosed with FSAD does not produce enough fluid to lubricate the vagina. As a result, intercourse is often painful and unsatisfactory. The woman may then avoid sexual activity and intimacy, creating relationship difficulties.

The causes of FSAD are quite complex. For some women, FSAD is a lifelong disorder; they have never experienced a normal lubrication-swelling response. For other women FSAD develops after illness or emotional trauma, through physiological changes, or as a side effect of surgery, radiation therapy for cancer, or medication. FSAD can be generalized, occurring with different partners and in many different settings, or it can be situation-specific, occurring only with certain partners or under particular circumstances. In addition, FSAD may be due either to psychological factors or to a combination of physiological and psychological factors.

Physiological causes of FSAD include:

- damage to the blood vessels of the pelvic region resulting in reduced blood flow
- damage to the nerves in the pelvic area resulting in diminished arousal
- general medical conditions that damage blood vessels (coronary artery disease, high blood pressure, diabetes mellitus)
- nursing a baby (lactation)
- general medical conditions that cause changes in hormone levels (thyroid disorders, adrenal gland disorders, removal of the ovaries)
- lower levels of sex hormones due to aging (menopause)
- side effects of medications (antidepressants, antipsychotic drugs, drugs to lower blood pressure, sedatives, birth control pills, or other hormone-containing pills)

Psychological causes of FSAD include:

- chronic mild depression (dysthymia)

- emotional **stress**
- past sexual **abuse**
- emotional abuse
- bereavement
- self-image problems
- relationship problems with partner
- other mental health disorders (major depression, **post-traumatic stress disorder** or **obsessive-compulsive disorder**)

The physical and psychological factors leading to FSAD often appear together. For example, a woman who does not experience arousal because of illness or the side effects of medication may then develop self-image and relationship problems that reinforce her difficulty in reaching arousal.

Demographics

It is difficult to determine the incidence of FSAD, because many women are reluctant to seek help for this problem. FSAD may also be present concurrently with other female **sexual dysfunctions** and be difficult to distinguish from them. In addition, there is some disagreement in the medical community on the exact descriptions of different female sexual dysfunctions. A recent review of the medical literature, however, found that 22–43% of women experience some form of sexual dysfunction. One study that looked specifically at lubrication found that about 20% of women reported problems in this area. Both of these estimates include women whose dysfunction arises from physiological and psychological causes.

Diagnosis

FSAD is usually diagnosed when a woman reports her concerns to her doctor, usually a gynecologist (a doctor who specializes in women's health issues); although she may also discuss it with a family doctor or a psychotherapist. The doctor will take a complete medical and psychological history, including a list of the medications that the patient is currently taking. The doctor will then give the patient a physical examination to evaluate medical aspects of the disorder; if necessary, blood and urine samples may be taken for laboratory testing to rule out previously undiagnosed diabetes or other medical conditions. In order to be diagnosed with FSAD, the lack of lubrication-swelling response must happen persistently or intermittently over an extended period. It is normal for women to have occasional problems with arousal, and these occasional difficulties are not the same as FSAD. The lack of sexual response must cause emotional distress or relationship difficulties for the woman and be caused either only by psychological factors or by a combination of psychological and physical factors to meet the criteria for a **diagnosis** of FSAD.

According to the mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (2000), which is also called *DSM-IV-TR*, a diagnosis of FSAD is not appropriate if problems with arousal are caused only by physiological factors. These factors may include injuries to the genital area, illness, or menopause. When the causes are only physiological, a diagnosis of sexual dysfunction due to a general medical condition is appropriate. If lack of arousal is caused by the side effects of medication or substance abuse, a diagnosis of substance-induced sexual dysfunction would be made. FSAD is also not diagnosed if it

KEY TERMS

Adrenal gland—A small organ located above each kidney that produces hormones related to the sex drive.

Cervix—The neck or narrow lower end of a woman's uterus.

Clitoris—The most sensitive area of the external genitals. Stimulation of the clitoris causes most women to reach orgasm.

Labia—The outside folds of tissue that surround the clitoris and the opening of the urethra in women.

Menopause—A period of decreasing hormonal activity in women, when ovulation stops and conception is no longer possible.

Pelvis—The basin-like cavity in the human body below the abdomen, enclosed by a framework of four bones.

Penis—The external male sex organ.

Thyroid—A gland in the neck that produces the hormone thyroxin, which is responsible for regulating metabolic activity in the body. Supplemental synthetic thyroid hormone is available as pills taken daily when the thyroid fails to produce enough hormone.

Uterus—The hollow muscular sac in which a fetus develops; sometimes called the womb.

Vagina—The part of the female reproductive system that opens to the exterior of the body and into which the penis is inserted during sexual intercourse.

is a symptom of another major psychological disorder. If a woman receives inadequate sexual stimulation from a partner, that also is not considered a cause of FSAD.

Treatments

Treatment varies depending on the cause of FSAD. When there are physical causes, the root problem or disease is treated. Many women who have difficulties with lubrication due to naturally decreasing hormone levels associated with aging are helped by hormone replacement therapy (HRT). There are also nonprescription preparations available in pharmacies for supplementing the woman's natural lubricant. Many women find these preparations quite satisfactory, particularly if they have only occasional problems with arousal.

The United States Food and Drug Administration has approved one medical device for treating FSAD. The Eros-Clinical Therapy Device (Eros-CTD) is a small vacuum pump that fits over the clitoral area. The pump produces a gentle sucking action that stimulates blood flow in the area. In clinical trials the device proved safe and effective in increasing blood flow, sensation, and vaginal lubrication. In May 2000, it was approved for the treatment of FSAD. As of 2002, clinical trials are underway to investigate whether sildenafil (Viagra) can increase blood flow to the genital area in women as it does in men.

Psychotherapy, or **talk therapy**, is most commonly used to treat the psychosocial aspects of FSAD. Sex therapy focuses primarily on the sexual dysfunction. Sex therapists have special training to help individuals and couples overcome their sexual difficulties. Traditional psychotherapy focuses on problems in relationships, seeking to clarify problems, identify emotions, improve communication, and promote problem-solving strategies. Therapy can involve either the woman alone or the woman and her partner (**couples therapy**). Many couples experiencing sexual dysfunction develop relationship problems related to sexual expectations, and benefit from traditional psychotherapy even when difficulties with sexual arousal are resolved.

Prognosis

Because FSAD has multiple causes, individual response to treatment varies widely. Difficulties with lubrication related to the menopause generally have a good prognosis. Stress-related difficulties with arousal typically resolve when the stressor is no longer present. Couples often need to work through relationship issues that have either caused or resulted from sexual dysfunction before they see an improvement in sexual arousal. This process takes time and a joint commitment to problem-solving.

Prevention

There are no sure ways to prevent FSAD. Eating a healthy, well-balanced diet, getting enough rest, having regular gynecological check-ups, and seeking counseling or psychotherapy when problems begin to appear in a relationship can help minimize sexual arousal problems.

See also Female orgasmic disorder; Sexual aversion disorder

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ORGANIZATIONS

American Association of Sex Educators, Counselors, and Therapists (AASECT). P. O. Box 238, Mount Vernon, IA 53214-0238. (319) 895-8407. <www.aasect.org>.

Sexual Information and Education Council of the United States (SIECUS). West 42nd Street, Suite 350, New York, NY 10036-7802, <www.siecus.org>.

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Fetishism

Definition

Fetishism is a form of paraphilia, a disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving non-human objects, the suffering or humiliation of oneself or one's partner (not merely simulated), or children or other non-

consenting persons. The essential feature of fetishism is recurrent intense sexual urges and sexually arousing fantasies involving specific objects. While any object may become a fetish, the distinguishing feature is its connection with sex or sexual gratification. A **diagnosis** of fetishism is made only if an individual has acted on these urges, is markedly distressed by them, or if the fetish object is required for gratification.

For some people with a paraphilia such as fetishism, paraphilic fantasies or stimuli may be necessary for erotic arousal and are always included in sexual activity, or the presence of the fetish object may occur only episodically. For example, the fetish object may only be necessary for arousal during periods of **stress**, and at other times, the person is able to function sexually without the fetish or stimuli related to the fetish.

Description

As stated, a fetish is a form of paraphilia, and in fetishism, the affected person has created a strong association between an object and sexual pleasure or gratification. A fetish is not simply a pleasant memory—it is a dominant component of most sexual situations. Most fetishes are objects or body parts. Common fetishes involve items of clothing, stuffed animals, or other non-sexual objects. Body fetishes may involve breasts, legs, buttocks, or genitals.

A person with a fetish often spends significant amounts of time thinking about the object of the fetish. Further, the object is intimately related to sexual pleasure or gratification. In the extreme, the presence of the fetish object is required for sexual release and gratification.

Causes and symptoms

Causes

The cause of the association between an object and sexual arousal may be adolescent curiosity or a random association between the object and feelings of sexual pleasure. A random association may be innocent or unappreciated for its sexual content when it initially occurs. For example, a male may enjoy the texture or tactile sensation of female undergarments or stockings. At first, the pleasurable sensation occurs randomly, and then, in time and with experience, the behavior of using female undergarments or stockings as part of sexual activity is reinforced, and the association between the garments and the sexual arousal is made. A person with a fetish may not be able to pinpoint exactly when his or her fetish began. A fetish may be related to activities associated with sexual **abuse**.

KEY TERMS

Paraphilia—A disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Symptoms

Early symptoms for a fetish involve touching the object of desire. The amount of time spent thinking about the fetish object may increase. Over time, the importance of the fetish object expands. In the extreme, it becomes a requirement for achieving sexual pleasure and gratification.

Demographics

How many people have a fetish and the extent to which the fetish influences their lives and sexual activities are not accurately known. In some rare instances, people with fetishes may enter the legal system as a result of their fetishes, and those cases may be counted or tracked.

Paraphilias such as fetishism are uncommon among females, but some cases have been reported. Females may attach erotic thoughts to specific objects such as items of clothing or pets, but these are uncommon elements in sexual activity. Virtually no information is available on family patterns.

Diagnosis

A diagnosis of a paraphilia involving a fetish is most commonly made by taking a detailed history or by direct observation. The diagnosis is made only if a person has actually obtained sexual gratification by using the fetish object, or has been markedly distressed by the inability to use such an object if contact with the fetish object is needed for sexual success. Occasionally discussing admiration for a particular object or finding an object to be arousing does not indicate a diagnosis of fetishism.

Treatments

In the earliest stages of behavior therapy, fetishes were narrowly viewed as attractions to inappropriate objects. Aversive stimuli such as shocks were administered to persons undergoing therapy. This approach was not successful. People with fetishes have also been

behaviorally treated by orgasmic reorientation, which attempts to help them develop sexual responses to culturally appropriate stimuli that have been otherwise neutral. This therapy has had only limited success.

Most persons who have a fetish never seek treatment from professionals. Most are capable of achieving sexual gratification in culturally appropriate situations. As of 2002, American society seems to have developed more tolerance for persons with fetishes than in the past, thus further reducing the already minimal demand for professional treatment.

Prognosis

The prognosis for eliminating a fetish is poor because most people with a fetish have no desire to change or eliminate it. Most cases in which treatment has been demanded as a condition of continuing a marriage have not been successful. Most fetishes are relatively harmless in that most do not involve other persons or endanger the person with the fetish. Persons with a fetish rarely involve non-consenting partners.

The personal prognosis for a person with a fetish is good if the fetish and related activities do not impact others or place the person with the fetish in physical danger.

Prevention

Most experts agree that providing gender-appropriate guidance in culturally appropriate situations will prevent the formation of a fetish. The origin of some fetishes may be random associations between a particular object or situation and sexual gratification. There is no way to predict such as association.

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Figure drawings

Definition

Figure drawings are projective diagnostic techniques in which an individual is instructed to draw a person, an object, or a situation so that cognitive, interpersonal, or psychological functioning can be assessed.

Purpose

A projective test is one in which a test taker responds to or provides ambiguous, abstract, or unstructured stimuli, often in the form of pictures or drawings. While other projective tests, such as the **Rorschach Technique** and **Thematic Apperception Test**, ask the test taker to interpret existing pictures, figure drawing tests require the test taker to create the pictures themselves. In most cases, figure drawing tests are given to children. This is because it is a simple, manageable task that children can relate to and enjoy.

Some figure drawing tests are primarily measures of cognitive abilities or cognitive development. In these tests, there is a consideration of how well a child draws and the content of a child's drawing. In some tests, the child's self-image is considered through the use of the drawings. In other figure drawing tests, interpersonal relationships are assessed by having the child draw a family or some other situation in which more than one person is present. Some tests are used for the evaluation of child **abuse**. Other tests involve personality interpretation through drawings of objects, such as a tree or a house, as well as people. Finally, some figure drawing tests are used as part of the diagnostic procedure for specific types of psychological or neuropsychological

impairment, such as central nervous system dysfunction or **mental retardation**.

Precautions

Despite the flexibility in administration and interpretation of figure drawings, these tests require skilled and trained administrators familiar with both the theory behind the tests and the structure of the tests themselves. Interpretations should be made with caution and the limitations of projective tests should be considered. It is generally a good idea to use projective tests as part of an overall test battery. There is little professional support for the use of figure drawing, so the examples that follow should be interpreted with caution.

Description

The Draw-A-Man Test, developed by Goodenough in 1926 was the first formal figure drawing test. It was used to estimate a child's cognitive and intellectual abilities reflected in the drawing's quality. The test was later revised by Harris in 1963 as the Goodenough Harris Drawing Test (GHDT), which included a detailed scoring system and allowed for drawings of men, women, and the self. The scoring system primarily reflected the way in which the child is maturing cognitively. The GHTD is appropriate for children between the ages of three and 17, although it has been found to be most useful for children between three and 10.

The Draw-A-Person test (DAP) was developed by Machover in 1948 and used figure drawings in a more projective way, focusing on how the drawings reflected the anxieties, impulses, self-esteem, and personality of the test taker. In this test, children are first asked to draw a picture of a person. Then, they are asked to draw a picture of a person of the sex opposite of the first drawing. Sometimes, children are also asked to draw a picture of the self and/or family members. Then, they are asked a series of questions about themselves and the drawings. These questions can be about the mood, the ambitions, and the good and bad qualities of the people in the drawings. The pictures and the questions on the DAP are meant to elicit information about the child's anxieties, impulses, and overall personality. The DAP is the most frequently used figure drawing test today. A scoring system appropriate for adults was developed in 1993 by Mitchel, Trent, and McArthur.

In 1992, Naglieri and his colleagues created a more specific scoring system for figure drawing tests called the Draw-A-Person: Screening Procedure of Emotional Disturbance (DAP:SPED), based on a large standardization sample. This scoring method includes 55 items rated by the test administrator and based on the child's drawings and responses to questions. The DAP:SPED is appropriate for children aged six to 17. It is often used as a screening method for children who may be having difficulties with regard to social adjustment and require further evaluation.

KEY TERMS

Projective test—A psychological test in which the test taker responds to or provides ambiguous, abstract, or unstructured stimuli, often in the form of pictures or drawings.

Reliability—The ability of a test to yield consistent, repeatable results.

Standardization—The administration of a test to a sample group of people for the purpose of establishing scoring norms. The DAP:SPED structured scoring system was standardized using a sample of over 2,300 children and adolescents.

Validity—The ability of a test to measure accurately what it claims to measure.

The **House-Tree-Person** (HTP) **test**, created by Buck in 1948, provides a measure of a self-perception and attitudes by requiring the test taker to draw a house, a tree, and a person. The picture of the house is supposed to conjure the child's feelings toward his or her family. The picture of the tree is supposed to elicit feelings of strength or weakness. The picture of the person, as with other figure drawing tests, elicits information regarding the child's self-concept. The HTP, though mostly given to children and adolescents, is appropriate for anyone over the age of three.

The Kinetic Family Drawing technique (KFD), developed in 1970 by Burns and Kaufman, requires the test taker to draw a picture of his or her entire family. Children are asked to draw a picture of their family, including themselves, "doing something." This picture is meant to elicit the child's attitudes toward his or her family and the overall family dynamics. The KFD is sometimes interpreted as part of an evaluation of child abuse.

The Kinetic School Drawing technique (KSD), developed in 1974 by Prout and Phillips, requires the child to draw a picture of himself or herself, a teacher, and one or more classmates. This picture is meant to elicit the child's attitudes toward people at school and his or her functioning in the school environment.

Results

As with all projective measures, scoring on figure drawing tests is more subjective. Specific scoring systems, such as the DAP:SPED can be used to provide more objective information. Most figure drawing tests have some sort of objective scoring system; however, the instructions given to the child, the questions asked by the test administrator, and the administrator's interpretations of the drawings are flexible and this makes it difficult to compare results between children, even on the same measure. Also, many clinicians choose not to rely on the scoring systems and rely entirely on their own intuitive judgments regarding their interpretation of picture content.

Figure drawings are often interpreted with regard to appropriate cognitive development. Naglieri's DAP:SPED scoring system includes a consideration of what features in a drawing are appropriate for children of various ages. For example, five-year old children are expected to make fairly basic drawings of people, consisting of a head, eyes, nose, mouth, body, arms, and legs. An 11-year-old, on the other hand is expected to have more details in the picture, such as a more defined neck, clothes, and arms in a particular direction.

Sometimes, figure drawings are assessed with regard to self-image. Children often project themselves in the drawings. For example, females with body image concerns may reflect these concerns in their drawings. Victims of sexual abuse may stress sexual characteristics in their drawings.

Psychological, neuropsychological, or emotional dysfunction can also be considered in figure drawing interpretation. This type of interpretation is often done with figure drawings made by adults. For example, a person who omits or distorts body parts may suffer from emotional impairment. Excessive detail with regard to the sexual nature of the drawing may indicate sexual maladjustment.

Family dynamics are also interpreted through figure drawings. For example, in the Kinetic Family Drawing test, a picture where family members are in separate rooms may indicate isolation or a lack of interaction between family members.

Figure drawings are also interpreted with regard to child abuse. In 1994, Von Hutton developed a scoring system for both the HTP and DAP focusing on indicators of child abuse that may be present in drawings. The drawing of the family in the KFD test may also provide indicators of abuse.

There has been much debate over the overall reliability and validity of figure drawing tests (and projective tests in general). For example, when structured scoring systems are used, the DAP has been found to be a reliable measure, especially for cognitive development in children. However, with regard to specific personality characteristics, self-image issues, or personality dysfunctions, there has been relatively little support for the use of figure drawings.

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Ali Fahmy, Ph.D.

Flooding *see* **Exposure treatment**

Fluoxetine

Definition

Fluoxetine is an antidepressant of the type known as selective serotonin reuptake inhibitors (SSRI). It is sold in the United States under the brand names Prozac and Sarafem.

Purpose

Fluoxetine is used to treat depression, premenstrual syndrome, bulimia, and **obsessive-compulsive disorder**.

Description

Serotonin is a **neurotransmitter**—a **brain** chemical that carries nerve impulses from one nerve cell to another. Researchers think that depression and certain other mental disorders may be caused, in part, because there is not enough serotonin being released and transmitted in the brain. Like the other SSRI antidepressants, **fluvoxamine** (Luvox), **sertraline** (Zoloft), and **paroxetine** (Paxil), fluoxetine increases the level of brain serotonin (also known as 5-HT). Increased serotonin levels in the brain may be beneficial in patients with obsessive-compulsive dirder, alcoholism, certain types of headaches, **post-traumatic stress disorder** (PTSD), pre-menstrual tension and mood swings, and **panic disorder**.

Fluoxetine was the first of the class of antidepressants called selective serotonin reuptake inhibitors (SSRIs) to be approved for use in the United States. In 2000, fluoxetine was approved by the FDA for use in treating premenstrual dysphoric disorder.

The benefits of fluoxetine develop slowly over a period of several weeks. Patients should be aware of this and continue to take the drug as directed, even if they feel no immediate improvement.

Fluoxetine (marketed as Prozac) is available in 10-, 20-, and 40-mg capsules, 10-mg tablets, and in a liquid solution with 20 mg of active drug per 5 ml. Prozac Weekly capsules are a time-release formula containing 90 mg of active drug. Sarafem is available in 10- and 20-mg capsules.

Recommended dosage

Fluoxetine therapy in adults is started as a single 20-mg dose, initially taken in the morning. Depending on the patient's response after four to six weeks of therapy, this dose can be increased up to a total of 80 mg per day. Doses over 20 mg per day can be given as equally divided morning and afternoon doses.

Precautions

Patients taking fluoxetine should be monitored closely for **insomnia**, anxiety, mania, significant weight loss, **seizures**, and thoughts of **suicide**.

Caution should also be exercised when prescribing fluoxetine to patients with impaired liver or kidney function, the elderly (over age 60) children, individuals with known manic-depressive disorder or a history of seizures, people with diabetes, and individuals expressing ideas of committing suicide.

Individuals should not take MAO inhibitors during fluoxetine therapy, for two weeks prior to beginning fluoxetine therapy, and for five weeks after stopping fluoxetine therapy.

Care should be taken to weigh the risks and benefit of this drug in women who are, or wish to become, pregnant, as well as in breast-feeding mothers.

People with diabetes should monitor their blood or urine sugar more carefully, since fluoxetine can affect blood sugar.

Until an individual understands the effects that fluoxetine may have, he or she should avoid driving, operating dangerous machinery, or participating in hazardous activities. Alcohol should not be used while taking fluoxetine.

KEY TERMS

Bulimia—An eating disorder characterized by binges in which large amounts of food are consumed, followed by forced vomiting.

Obsessive-compulsive disorder—Disorder in which the affected individual has an obsession (such as a fear of contamination, or thoughts he or she doesn't like to have and can't control) and feels compelled to perform a certain act to neutralize the obsession (such as repeated handwashing).

Pre-menstrual syndrome—A severe change in mood that occurs in women immediately prior to, and during, their menstrual period.

Side effects

More common side effects include decrease sexual drive, restlessness, difficulty sitting still, skin rash, hives, and itching.

Less common side effects include fever and/or chills, and pain in joints or muscles.

Rare side effects include pain or enlargement of breasts and/or abnormal milk production in women, seizures, fast heart rate, irregular heartbeats, red or purple spots on the skin, low blood sugar and its symptoms (anxiety, chills, cold sweats, confusion, difficulty concentrating, drowsiness, excess hunger, rapid heart rate, headache, shakiness or unsteadiness, severe **fatigue**), low blood sodium and its symptoms (including confusion, seizures, drowsiness, dry mouth, severe thirst, decreased energy), serotonin syndrome (usually at least three of the following: diarrhea, fever, sweatiness, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking), excitability, agitation, irritability, pressured talking, difficulty breathing, and odd body or facial movements.

Interactions

Fluoxetine interacts with a long list of other medications. People starting this drug should review the other medications they are taking with their physician and pharmacist for possible interactions. Patients should always inform all of their health care providers, including dentists, that they are taking fluoxetine.

When taken with fluoxetine, blood levels of the following drugs may increase: benzodiazepines, **beta blockers**, **carbamazepine**, dextromethorphan, **haloperidol**,

atorvastatin, lovastatin, simvastatin, phenytoin, and tricyclic antidepressants.

The following drugs may increase the risk of serotonin syndrome: dexfenfluramine, fenfluramine, and tryptophan.

When **buspirone** is taken with fluoxetine, the therapeutic effect of buspirone may be impaired.

Low blood sodium may occur when fluoxetine is taken along with diuretics.

Increased risk of mania and high blood pressure occurs when selegiline is taken along with fluoxetine.

Severe, fatal reactions have occurred when fluoxetine is given along with MAO inhibitors.

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Fluphenazine

Definition

Fluphenazine is a phenothiazine antipsychotic sold under the brand names Permitil and Prolixin in the United States. It is also available under its generic name.

Purpose

Fluphenazine is a drug used to treat psychotic disorders, agitation, and **dementia**.

Description

Fluphenazine is one of many drugs in the group called the phenothiazines. Phenothiazines work by inhibiting the actions of the **brain** chemicals, dopamine and norepinephrine, which are overproduced in individuals with **psychosis**.

Fluphenazine is available in 1-mg, 2.5-mg, 5-mg, and 10-mg tablets, a liquid concentrate containing 5 mg per 1 mL, a rapid-onset injectable form containing 2.5 mg per 1 mL, and a long-acting injectable form containing 25 mg per 1 mL.

Recommended dosage

In children over age 16 and in adults, fluphenazine is usually given in oral dosages ranging from 0.5–10 mg daily. The total dosage is usually divided and taken two to four times throughout the day. The dosage is typically reduced at a gradual pace over time to a range between 1 mg and 5 mg. Older adults usually receive lower doses that begin in the range of 1 mg–2.5 mg per day. In children under age 16, the usual range is 0.25–3.5 mg per day divided into several doses. Maximum dosage is normally 10 mg per day for this age group.

This drug is also available by injection. In adults, injections into the muscle range from 1.25–10 mg per day divided into several doses. A long-acting injectable form can also be administered to patients who have been stabilized on the drug. The dose for the long-acting preparation ranges from 12.5–25 mg given every one to four weeks in adults. The dosage for children is lower in all cases.

Precautions

People with a history of depression, lung problems, heart disease, glaucoma, **seizures**, and kidney disease should take fluphenazine only after careful evaluation by their physician. In addition, those undergoing alcohol withdrawal and those who have received **electroconvulsive therapy** should take this drug with great caution and close physician supervision after discussing the risks and benefits with their doctor. Those over age 60 and children under age 12 should take fluphenazine only after a thorough assessment from their physician. Pregnant women should use fluphenazine with great caution.

Fluphenazine may cause drowsiness. People who take this drug should not drive, operate heavy machinery, or perform other hazardous tasks requiring mental alertness until they see how the drug affects them. People taking fluphenazine should avoid significant exposure to sunlight, as the drug may cause people to sunburn more easily. This drug can sometimes change the color of urine to a pinkish or reddish-brown color. Fluphenazine use can make people more susceptible to heat and increase the risk of heatstroke. People taking fluphenazine should get up slowly after being in a reclining position because of potential dizziness.

Side effects

Relatively common side effects that accompany fluphenazine include drowsiness, dizziness, rash, dry mouth, **insomnia**, **fatigue**, muscular weakness, anorexia, blurred vision, some loss of muscular control, and amenorrhea (lack of menstruation) in women.

Dystonia (difficulty walking or moving) may occur with fluphenazine use. This condition may subside in 24 to 48 hours even when the person continues taking the drug and usually disappears when fluphenazine is discontinued.

Fluphenazine use may lead to the development of symptoms that resemble Parkinson's disease. These symptoms may include a tight or mask-like expression on the face, drooling, tremors, pill-rolling motions in the hands, cogwheel rigidity (abnormal rigidity in muscles characterized by jerky movements when the muscle is passively stretched), and a shuffling gait. Taking anti-Parkinson drugs **benztropine** mesylate or **trihexyphenidyl** hydrochloride along with the fluphenazine usually controls these symptoms.

Fluphenazine has the potential to produce a serious side effect called **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements that may appear late in therapy and may not disappear even after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage of fluphenazine. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period.

An occasionally reported side effect of fluphenazine is neuroleptic malignant syndrome. This is a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia (fast heartbeat), and arrhythmias (irregular heartbeat). People who think they may be experiencing any side effects from this or any other medication should talk to their physician promptly.

KEY TERMS

Agitation—Excessive restlessness or emotional disturbance that is often associated with anxiety or psychosis.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Interactions

Barbiturates lower the level of fluphenazine in the blood. The blood pressure drugs known as beta-blockers tend to decrease the level of fluphenazine in the blood. Bromocriptine, a drug used for Parkinson's disease, also lowers the level of fluphenazine in the blood. Conversely, antimalarial drugs can increase the level of fluphenazine in the blood.

The combination of fluphenazine with the drugs known as cyclic antidepressants lowers the concentrations of both drugs in the blood. Fluphenazine inhibits the blood pressure-lowering effects of the drug called guanadrel. Levodopa, a drug given to patients with Parkinson disease, is less effective when combined with fluphenazine. The combination of fluphenazine with meperidine can cause very low blood pressure and significant depression of the central nervous system. The use of the muscle relaxant, orphenadrine, can lower the effective levels of fluphenazine in the blood.

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Flurazepam

Definition

Flurazepam is a benzodiazepine hypnotic (sleeping medication) that is given by mouth. It is sold in the United States under the brand name of Dalmane, but is also manufactured and sold by several companies under its generic name.

Purpose

Flurazepam is used for the short-term treatment of **insomnia**, which is a sleep disorder characterized by difficulty in falling or staying asleep.

Description

Flurazepam is a benzodiazepine, which means that it belongs to a class of drugs whose primary action is to reduce the patient'sanxiety, relax the skeletal muscles, and bring on sleep. Flurazepam is chemically and pharmacologically related to such other benzodiazepine hypnotics as **temazepam** (Restoril), **triazolam** (Halcion), **quazepam** (Doral), and **estazolam**. All the benzodiazepines work by enhancing the effects of a naturally occurring chemical in the body called gamma-aminobutyric acid (GABA). GABA is a neurotransmitter, or chemical that helps to conduct nerve impulses across the tiny gaps between nerve cells. GABA acts to lower the level of activity in the central nervous system; it is involved in muscle relaxation, sedation, and sleep, and plays a role in preventing seizure activity.

Flurazepam decreases the time it takes the patient to fall asleep, thus reducing the number of nighttime awakenings and increasing the length of total sleep time. The difference between a benzodiazepine like flurazepam that is used to help patients fall asleep and those that are used as tranquilizers is the way that each type acts in the **brain**. The sleep-inducing benzodiazepines are faster in getting to the part of the brain that controls sleep. They also reach higher levels of concentration there than the benzodiazepines that are used as tranquilizers.

Flurazepam is available in 15- and 30-mg capsules.

Recommended dosage

The usual dose of flurazepam is 15–30 mg taken by mouth at bedtime. Older or physically weakened patients are usually given the lower dose. Women who are pregnant or nursing a baby, and children younger than 15 should not be given flurazepam. In addition, the drug should not be used for longer than four weeks.

KEY TERMS

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Central nervous system depressant—Any drug that lowers the level of stimulation or excitement in the central nervous system.

Central nervous system stimulant—Any drug that raises the level of activity in the central nervous system.

Gamma-aminobutyric acid (GABA)—A neurotransmitter that helps to lower or reduce the level of excitement in the nerves, leading to muscle relaxation; calmness; sleep; and prevention of seizures.

Hypnotic—A type of medication that induces sleep.

Metabolism—The group of biochemical processes within the body that release energy in support of life.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Rebound effect—A physical reaction to stopping a medication characterized by the reappearance of the symptom that the medication was given to suppress. For example, people who stop taking flurazepam may experience rebound insomnia.

Precautions

Some of the flurazepam is metabolized (broken down) in the body to form another compound called desalkylflurazepam, which can also cause drowsiness the next day because it remains in the body for hours. This "hangover" effect is most common in people who are taking flurazepam on a daily basis. People who are taking flurazepam may not be able to operate machinery safely or drive a car the next day.

Patients who take flurazepam for several days or weeks may experience a reaction called rebound insomnia when they stop taking it. When a person takes a medication for sleep on a regular basis, the body adjusts to the presence of the drug. It tries to counteract the effects of the medication. As a result, when the person stops taking the sleeping medication, the body will take a few nights to return to its normal condition. During this period of readjustment, the person may experience a few sleepless hours each night.

The sleepiness that flurazepam brings about may be intensified if the patient drinks alcoholic beverages or takes other medications that contain central nervous system depressants. Common types of medications that may cause problems when combined with flurazepam include tranquilizers and antihistamines.

Elderly patients who are taking flurazepam should be monitored for signs of dizziness or loss of coordination. They are at increased risk of falling if they wake up and get out of bed during the night to get a drink of water or use the bathroom.

Side effects

Some people experience dizziness, daytime drowsiness, and loss of coordination while they are taking flurazepam. Elderly patients may lose their balance and fall. Less common side effects include blurred vision, nausea and vomiting, diarrhea or constipation, nightmares, and a feeling of depression.

Interactions

The effects of flurazepam are increased by other central nervous system depressants. These types of chemicals include alcohol, sedatives, and antihistamines (allergy medications). In addition, flurazepam may interact with anti-seizure medications.

See also Sedatives and related disorders; Sleep disorders

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Fluvoxamine

Definition

Fluvoxamine is an antidepressant of the type known as selective serotonin reuptake inhibitors (SSRI). It is marketed in the United States under the brand name Luvox.

Purpose

Fluvoxamine is used to treat depression. It is also the first SSRI to be approved by the United States Food and Drug Administration (FDA) for use in **obsessive-compulsive disorder** in children, adolescents, and adults.

Description

Serotonin is a **brain** chemical that carries nerve impulses from one nerve cell to another. Researchers think that depression and certain other mental disorders may be caused, in part, because there is not enough serotonin being released and transmitted in the brain. Like the other SSRI antidepressants, **fluoxetine** (Prozac), **sertraline** (Zoloft), and **paroxetine** (Paxil), fluvoxamine increases the level of brain serotonin (also known as 5-HT). Increased serotonin levels in the brain may be beneficial in patients with obsessive-compulsive dirder, alcoholism, certain types of headaches, **post-traumatic stress disorder** (PTSD), pre-menstrual tension and mood swings, and **panic disorder**.

Fluvoxamine was approved for use in adults in 1993. In 1997, the United States Food and Drug Administration (FDA) approved this medication for the treatment of obsessive-compulsive disorder in children and adolescents.

Fluvoxamine is available in 25-, 50- and 100-mg tablets.

Recommended dosage

Fluvoxamine therapy in adults is started as a single 50-mg dose taken at bedtime. Based on the patient's response to the medication, the dosage can be increased by 50 mg every four to seven days, until maximum benefit is achieved. Maximum dosage is 300 mg per day. Dosage over 100 mg per day should be given as equally divided morning and afternoon doses.

Fluvoxamine therapy in children is started as a single 25-mg dose, initially taken at bedtime. Based on the patient's response to the medication, the dosage can be increased by 25 mg every four to seven days, until maximum benefit is achieved. Maximum dosage in children is 200 mg per day. Dosage over 100 mg per day should be given as equally divided morning and afternoon doses.

Precautions

Patients taking fluvoxamine should be monitored closely for the onset of mania, **seizures**, thoughts of **suicide**, and skin problems (including itching, hives, rashes).

People with impaired liver function, **bipolar disorder** (manic depression), a history of seizures, or individuals contemplating suicide should take fluvoxamine only under close physician supervision.

A group of serious side effects, called serotonin syndrome, has resulted from the combination of SSRI drugs such as fluvoxamine and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Serotonin syndrome usually consists of at least three of the following symptoms: diarrhea, fever, extreme perspiration, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking. Because of this, fluvoxamine should never be taken in combination with MAO inhibitors. People taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor and wait at least 14 days before starting fluvoxamine or any other antidepressant. The same holds true when discontinuing fluvoxamine and starting an MAO inhibitor.

Physicians and their patients should weigh the risks and benefits of this drug for women who are or wish to become pregnant, as well as in breast-feeding mothers.

Until an individual understands the effects that fluvoxamine may have on them, he or she should avoid driving, operating dangerous machinery, or participating in hazardous activities.

People should not use alcohol while taking fluvoxamine.

Side effects

Common side effects of fluvoxamine therapy include decreased sex drive or decreased sexual performance.

Less common side effects of fluvoxamine therapy include changes in mood, behavior, or thinking, difficulty breathing, difficulty urinating, and twitches or uncontrollable movements of the face or body.

Rare side effects include difficulty moving, blurred vision, clumsiness or problems with balance, seizures, difficulty moving the eyes, increased uncontrollable movements of the body or face, changes in the menstrual period, redness or irritation of the eyes or skin, peeling, itching or burning sensation of the skin, sore throat, fever and/or chills, easy bruising, nosebleeds, abnormal milk production in women, and symptoms of serotonin syndrome (usually at least three of the following: restlessness, overexcitement, irritability, confusion, diarrhea, fever, overactive reflexes, difficulty with coordination, uncontrollable shivering or shaking, trembling or twitching).

Interactions

Fluvoxamine interacts with a long list of other medications. Anyone starting this drug should review the other medications they are taking with their physician and pharmacist for possible interactions. Patients should always inform all their health care providers, including dentists, that they are taking fluvoxamine.

When taken together with fluvoxamine, the effect of the following drugs may be enhanced: benzodiazepines, **beta blockers**, **clozapine**, cholesterol-lowering drugs such as atorvastatin, lovastatin, and simvastatin, antiseizure drugs phenytoin and **carbamazepine**, tricyclic antidepressants, and **pimozide**.

The diet pills dexfenfluramine and fenfluramine may increase the incidence of serotonin syndrome when taken with fluvoxamine.

When **buspirone** is given with fluvoxamine, the therapeutic effect of buspirone may be decreased and the risk of seizures increased.

Increased risk of mania and high blood pressure occurs with selegiline.

Severe, fatal reactions mentioned above have occurred when fluvoxamine is given along with MAO inhibitors.

Fluvoxamine given with warfarin (a blood thinner) may increase the possibility of bleeding.

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Rosalyn Carson-DeWitt, M.D.

Folie á deux *see* **Shared psychotic disorder**

Frotteurism

Definition

Frotteurism is a disorder in which a person derives sexual pleasure or gratification from rubbing, especially the genitals, against another person, usually in a crowd. The person being rubbed is a victim. Frotteurism is a paraphilia, a disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving objects, the suffering or humiliation of oneself or one's partner (not merely simulated), or children or other nonconsenting persons.

Description

The primary focus of frotteurism is touching or rubbing one's genitals against the clothing or body of a nonconsenting person. This behavior most often occurs in situations that allow rapid escape. Frottage (the act of rubbing against the other person) is most commonly practiced in crowded places such as malls, elevators, on busy sidewalks, and on public transportation vehicles.

The most commonly practiced form of frotteurism is rubbing one's genitals against the victim's thighs or buttocks. A common alternative is to rub one's hands over the victim's genitals or breasts.

Most people who engage in frottage (sometimes called frotteurs) usually fantasize that they have an exclusive and caring relationship with their victims during the moment of contact. However, once contact is made and broken, the frotteur realizes that escape is important to avoid prosecution.

Causes and symptoms

Causes

There is no scientific consensus concerning the cause of frotteurism. Most experts attribute the behavior to an initially random or accidental touching of another's genitals that the person finds sexually exciting. Successive repetitions of the act tend to reinforce and perpetuate the behavior.

Symptoms

In order for the disorder to be clinically diagnosed, the symptoms must meet the diagnostic criteria as listed in the professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***. These symptoms include:

- Recurrent, intense, or sexually arousing fantasies, sexual urges, or behaviors that involve touching and rubbing against a nonconsenting person.
- The person has acted on these sexual urges, or the fantasies or urges cause significant distress to the individual or are disruptive to his everyday functioning.

Demographics

Males are much more likely to engage in frotteurism than are females. Females are the most common victims of frotteurism. Most acts of frottage are peformed by those between 15 to 25 years of age. After the age of 25, the acts decline.

KEY TERMS

Frottage—The act of touching or rubbing against the body or genitals of a non-consenting individual.

Medroxyprogesterone—A progestin, a female hormone.

Paraphilia—A disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Diagnosis

Most people with frotteurism never seek professional help, but people with the disorder may come into the mental health system as a result of a court order. The **diagnosis** is established in an interview between the person accused of frotteurism and the mental health professional (a **psychiatrist** or a **psychologist**, for example). In the interview, the individual acknowledges that touching others is a preferred or exclusive means of sexual gratification. Because this acknowledgment can bring criminal charges, the disorder is underdiagnosed and its prevalence is largely unknown. In some cases, other people besides the accused may be interviewed, including observers or the victim.

Treatments

For treatment to be successful, the frotteur must want to modify existing patterns of behavior. This initial step is difficult for most people with this disorder to take.

Behavior therapy is commonly used to try to treat frotteurism. The frotteur must learn to control the impulse to touch nonconsenting victims. Medroxyprogesterone, a female hormone, is sometimes prescribed to decrease sexual desire.

Frotteurism is a criminal act in many jurisdictions. It is usually classified as a misdemeanor. As a result, legal penalties are often minor. It is also not easy to prosecute frotteurs as intent to touch is difficult to prove. In their defense statements, the accused often claim that the contact was accidental.

The primary goal of frotteurism is to touch the clothing or body of a non-consenting person. This behavior most often occurs in situations that allow rapid escape, and frotteurism is most commonly practiced in crowded places such as public transportation vehicles. *(Patrik Giardino/CORBIS. Photo reproduced by permission.)*

Prognosis

The prognosis for eliminating frotteurism is poor as most frotteurs have no desire to change their behavior. Since frotteurism involves nonconsenting partners and is against the law in many jurisdictions, the possibility of embarrassment may deter some individuals.

Prevention

Most experts agree that providing guidance as to behavior that is culturally acceptable will prevent the development of a paraphilia such as frotteurism. The origin of some instances of frotteurism may be a truly accidental contact that becomes associated with sexual gratification. There is no way to predict when such an association will occur.

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American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. FAX (202) 682-6850. Web site: <http://www.psych.org/>.

American Psychological Association. 750 First Street NW, Washington, DC, 20002-4242, Telephone: (800) 374-2721 or (202) 336-5500. Web site: <http://www.apa.org/>.

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Fugue *see* **Dissociative fugue**

G

Gabapentin

Definition

Gabapentin is an anti-seizure medication. It is sold in the United States under the trade name Neurontin.

Purpose

Gabapentin is used in combination with other anti-seizure (anticonvulsant) drugs to manage partial **seizures** with or without generalization in individuals over the age of 12. Gabapentin can also be used to treat partial seizures in children between the ages of three and 12. Off-label uses (legal uses not specifically approved by the United States Food and Drug Administration [FDA]) include treatment of severe, chronic pain caused by nerve damage (such as occurs in shingles, diabetic neuropathy, multiple sclerosis, or post-herpetic neuralgia). Studies are also looking at using gabapentin to treat **bipolar disorder** (also known as manic-depressive disorder).

Description

Brain cells normally transmit nerve impulses from one cell to another by secreting chemicals known as **neurotransmitters**.

Gabapentin is chemically related to a naturally occurring neurotransmitter called GABA (gamma-amino butyric acid). The actual mechanism of action by which gabapentin acts in the brain to control seizures and treat pain is not known, although it appears to alter the action of nerve cells.

Gabapentin was approved for use in the United States in 1993. A liquid formulation was approved for use in 2000. Use in children ages three to 12 was also approved by the FDA in 2000.

Gabapentin is available in 100-, 300-, and 400-mg capsules; in 600- and 800- mg tablets; and in a liquid solution containing 250 mg per 5 ml.

Recommended dosage

People over the age of 12 should be started on 300 mg gabapentin taken three times a day. The dose can be increased up to a total of 1,800 mg per day. In some instances, doses of up to 3,600 mg per day have been tolerated.

Children should receive a dosage of 10–15 mg per kg of body weight per day, divided into three equal doses.

Chronic pain may be treated with 300–3,600 mg per day, divided into three equal doses.

When gabapentin is used for bipolar disorder, the starting dose is usually 300 mg taken at bedtime. Depending on the patient's response, the dose can be increased every four to seven days. Many people receive maximum therapeutic benefit at 600 mg per day, although some people have required up to 4,800 mg per day.

Precautions

Women who are breast-feeding and people who have decreased kidney functioning should discuss the risks and benefits of this drug with their physician. Women who are or wish to become pregnant will also require a careful assessment of the risks and benefits of gabapentin.

Patients should not suddenly discontinue gabapentin, as this can result in an increased risk of seizures. If the medication needs to be discontinued, the dosage should be reduced gradually over a week.

Until an individual understands the effects that gabapentin may have, he or she should avoid driving, operating dangerous machinery, or participating in hazardous activities. Alcohol should be avoided while taking gabapentin.

Side effects

Patients who experience the following side effects of gabapentin should check with their doctor immediately.

KEY TERMS

Diabetic neuropathy—A condition in which the nerve endings, particularly in the legs and feet, become less sensitive. Minor injuries, such as blisters or callouses, are not felt and can thus become infected and become more serious problems.

Multiple sclerosis—A disease characterized by patches of hardened tissue in the brain or spinal cord, paralysis, and/or muscle tremors.

Neuralgia—Pain that extends along the course of a nerve.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

These include more common side effects, such as unsteadiness, clumsiness, and uncontrollable back-and-forth eye movements or eye rolling. Less common side effects include depression, irritability, other mood changes or changes in thinking, and decreased memory. Rare side effects include pain in the lower back or side, difficulty urinating, fever and/or chills, cough, or hoarseness.

Children under age 12 who have the following more common side effects should also check with their doctor immediately: aggressive behavior, irritability, anxiety, difficulty concentrating and paying attention, crying, depression, mood swings, increased emotionality, hyperactivity, suspiciousness or distrust.

Multiple side effects often occur when a patient starts taking gabapentin. While these side effects usually go away on their own, if they last or are particularly troublesome, the patient should consult a doctor. More common side effects that occur when first starting to take gabapentin include blurred or double vision, muscle weakness or pain, swollen hand, feet, or legs, trembling or shaking, and increased **fatigue** or weakness. Less common side effects that occur when initiating gabapentin treatment include back pain, constipation, decreased sexual drive, diarrhea, dry mouth and eyes, frequent urination, headache, indigestion, low blood pressure, nausea, ringing in the ears, runny nose, slurred speech, difficulty thinking and sleeping, weight gain, twitching, nausea and/or vomiting, weakness.

Interactions

Antacids can decrease gabapentin levels in the blood. They should be taken at least two hours before taking gabapentin.

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Rosalyn Carson-DeWitt, M.D.

Galantamine

Definition

Galantamine belongs to a class of drugs called acetylcholinesterase inhibitors. In the United States galantamine is sold under brand name Reminyl.

Purpose

Galantamine is used to treat the symptoms of **Alzheimer's disease**. Galantamine is also being evaluated for the treatment of respiratory depression, mania, **vascular dementia** due to **stroke** or cardiac arrest that causes **brain** lesions, and reversal of side effects, such as blurred vision and mental changes caused by medications such as scopolamine.

Description

Alzheimer's disease develops when brain cells, called neurons, undergo an early, and selective death. It is believed that the premature death of these neurons may be prevented if stimulated by a brain chemical called acetylcholine. Acetylcholine is recycled by an enzyme called acetylcholinesterase. Galantamine works by inhibiting this enzyme. The inhibition of acetylcholinesterase increases the concentration of available acetylcholine.

Galantamine has only been studied, and is only used, in patients with mild-to-moderate Alzheimer's disease according to the Alzheimer's Disease Assessment Scale.

Galantamine is available in 4-mg, 8-mg, and 12-mg tablets.

Recommended dosage

The recommended initial dose of galantamine in adults is 4 mg twice daily. After a minimum of four weeks of treatment with galantamine, it may be increased to 8 mg twice daily. Further increases to 12 mg twice

daily should be initiated only after a minimum of four weeks at the previous dose.

Increased side effects associated with higher doses may prevent the increase in dose in some patients. Patients with moderate liver or kidney problems should not exceed 16 mg of galantamine daily.

Precautions

Galantamine should not be used in patients with severe liver or kidney problems. Since there are no well-controlled studies for the use of galantamine in pregnancy, galantamine should only be used if the potential benefits justify the potential risks to the fetus.

Patients who are undergoing anesthesia or bladder or gastrointestinal surgery should take galantamine only after a discussion with their physician. Patients with gastrointestinal problems should be closely monitored if it is decided that they should take galantamine. Galantamine should be used under close physician supervision in patients who have Parkinson's disease, severe asthma, or obstructive pulmonary disease. Because galantamine may slow down the heart, patients with any heart condition, and especially patients taking other medications that slow down the heart, should be evaluated before starting galantamine.

Side effects

The most common side effects reported with the use of galantamine are nausea, vomiting, diarrhea, anorexia, and abdominal pain. These occur most often at dosage-escalation periods. The average duration of nausea is five to seven days. These side effects tend to be less frequent if the patient is taking a total daily dosage of 16 mg. Eleven percent of patients receiving 24 mg daily lose weight, while 6% of patients receiving 16 mg daily experience weight loss.

Other common side effects include dizziness, headache, tremor, **fatigue**, depression, agitation, irritation, and **insomnia**. These side effects have a higher incidence and severity if higher doses are used. If side effects become severe, the dosage should be adjusted downward under physician supervision.

Interactions

There is currently little data regarding potential drug interactions with galantamine. Medications that are known to increase levels of galantamine in the body include cimetidine, erythromycin, ketoconazole, and **paroxetine**.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Acetylcholinesterase—The chemical responsible for the breakdown of acetylcholine

Parkinson's disease—A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness.

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Gambling *see* **Pathological gambling disorder**

Ganser's syndrome

Definition

Ganser's syndrome is a rare disorder in which the affected person gives approximate answers to questions that have right and wrong answers, such as "What is 5 minus 3?"

KEY TERMS

Factitious disorder—A type of mental disturbance in which patients intentionally act physically or mentally ill without obvious benefits. It is distinguished from malingering by the absence of an obvious motive, and from conversion disorder by intentional production of symptoms.

Malingering—Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

Description

Although this disorder was previously classified as a **factitious disorder**, the American Psychiatric Association has redefined Ganser's syndrome and placed it in the category called "Dissociative Disorder Not Otherwise Specified." Sometimes called "the syndrome of approximate answers," Ganser's syndrome is most often seen in male prisoners. In the past, this was so much the case that early clinicians called the syndrome "prison psychosis," despite the fact that it is not a true **psychosis**. (Psychosis is characterized by a radical change in personality and a distorted sense of reality.) The disorder has also been referred to as hysterical pseudodementia, due to the resemblance of responses to those of demented patients. However, data on the prevalence of the syndrome and on links within families have not been gathered and analyzed.

Ganser's syndrome is usually sudden in onset and, like **malingering**, seems to arise in response to an opportunity for personal gain or the avoidance of some responsibility. The patient will offer nearly correct replies when asked questions about facts of common knowledge, such as the number of days in a year, the number of months in a year, subtracting seven from 100, the product of four times five, etc. To such questions, the patient may respond by stating that there are 360 days in a year, 11 months in a year, 94 for the result of subtracting seven from 100, and that 21 is the product of four times five. These persons appear to have no difficulty in understanding questions asked, but appear to provide incorrect answers deliberately.

This syndrome is seen in conjunction with a preexisting severe personality disorder. However, unless the patient is willing to admit to the manufactured nature of the symptoms, or unless there is conclusive objective evidence contradicting the syndrome, determining whether the patient has a true disorder may be impossible. As with its sudden onset, disappearance of the symptoms can be just as fast. However, symptoms can also appear to worsen if the patient believes someone is watching. When reviewing a case of Ganser's syndrome, the clinician must consider factitious disorder and malingering as alternative diagnoses.

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Gender dysphoria *see* **Gender identity disorder**

Gender identity disorder

Definition

Gender identity disorder is a condition characterized by a persistent feeling of discomfort or inappropriateness concerning one's anatomic sex. The disorder typically begins in childhood with gender identity problems and is manifested in adolescence or adulthood by a person dressing in clothing appropriate for the desired gender, as opposed to one's birth gender. In extreme cases, persons with gender identity disorder may seek gender reassignment surgery, also known as a sex-change operation.

Description

Gender identity disorder is distressing to those who have it. It is especially difficult to cope with because it remains unresolved until gender reassignment surgery has been performed. Most people with this disorder grow up feeling rejected and out of place. **Suicide** attempts and substance abuse are common. Most adolescents and adults with the disorder eventually attempt to pass or live as members of the opposite sex.

Gender identity disorder may be as old as humanity. Cultural anthropologists and other scientists have observed a number of cross-gender behaviors in classical and Hindu mythology, Western and Asian classical history, and in many late nineteenth- and early twentieth-cen-

tury pre-literate cultures. This consistent record across cultures and time lends support to the notion that the disorder may be, at least in part, biological in origin. Not all behavioral scientists share this conclusion, however.

Gender identity and gender-appropriate behaviors are generally learned. This learning first occurs at home and later outside the home. Behavioral experimentation, particularly when a child is young, is considered normal. As they grow, children will often experiment with a variety of gender role behaviors as they learn to make the fine distinctions between masculine and feminine role expectations of the society in which they live. Some young boys occasionally exhibit behaviors that Western culture has traditionally labeled "feminine." Examples of these behaviors include wearing a dress, using cosmetics, or playing with dolls.

In a similar manner, some young girls will occasionally assume masculine roles during play. An example of this behavior includes pretending to be the father when playing house. Some girls temporarily adopt a cluster of masculine behaviors. These youngsters are often designated as tomboys. Most experts agree that such temporary or episodic adopting of behaviors opposite to one's gender is normal and usually constitute learning experiences in the acquisition of normal sex role socialization.

In pathological cases, however, children deviate from the normal model of exploring masculine and feminine behaviors. Such children develop inflexible, compulsive, persistent, and rigidly stereotyped patterns. On one extreme are boys who become excessively masculine. The opposite extreme is seen in effeminate boys who reject their masculinity and rigidly insist that they are really girls or that they want to become mothers and bear children.

Such males frequently avoid playing with other boys, dress in girls' clothing, play predominantly with girls, try out cosmetics and wigs, and display stereotypically feminine gait, arm movements, and body gestures. Although much less common, some girls may similarly reject traditionally feminine roles and mannerisms in favor of masculine characteristics. Professional **intervention** is required for both extremes of gender behavior.

This disorder is different from transvestitism or **transvestic fetishism**, in which cross-dressing occurs for sexual pleasure. Furthermore, the transvestite does not identify with the other sex.

Adults with gender identity disorder sometimes live their lives as members of the opposite sex. They often cross-dress and prefer to be seen in public as a member of the other sex. Some people with the disorder request sex-change or sex reassignment surgery.

KEY TERMS

Cross-dressing—Wearing clothing and other attire appropriate to the opposite sex.

Paraphilia—A disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Transsexual—A person whose gender identity is opposite his or her biologic sex.

Transvestite—A person who derives sexual pleasure or gratification from dressing in clothing of the opposite sex.

Persons with gender identity disorder frequently complain that they were born the wrong sex. They may describe their sexual organs as being ugly and may refrain from touching their genitalia. People with gender identity disorder may try to hide their secondary sex characteristics. For instance, males may try to shave off or pluck their body hair. Many elect to take female hormones in an effort to enlarge their breasts. Females may try to hide their breasts by binding them.

Causes and symptoms

Causes

There is no clearly understood or universally agreed-upon cause for gender identity disorder. However, most experts agree that there may be a strong biological basis for the disorder.

The sex of a human baby is determined by chromosomes. Males have a Y chromosome, in addition to a X chromosome, while females have two X chromosomes. The Y chromosome contains a gene known as the testes determining factor. This gene causes cells in an embryo to differentiate and develop male genitals. Embryos without the testes determining factor continue to develop undifferentiated as females.

The newly formed male testes release significant quantities of male hormones during the third month of pregnancy, further enhancing male differentiation. This sudden surge of hormones occurs again in males sometime between the second and twelfth week after birth. It is important to note that there is no corresponding feminizing hormonal surge sequence observed in females at this age.

These facts provide the biological basis for gender identity disorder. Male hormonal surges must occur not only in sufficient amounts, but also during a short window of time to cause masculinization of the developing infant. If there is insufficient androgen, the hormone primarily responsible for masculinization, or the surge comes too early or too late, the developing infant may be incompletely masculinized.

Disruptions of hormonal surges may come from a variety of sources. A partial list includes a disorder in the mother's endocrine system, common maternal **stress**, or maternal medications or some other toxic substances yet to be identified.

Recent post-mortem studies conducted on male-to-female transsexuals, non-transsexual men, and non-transsexual women show a significant difference in the volume of a portion of the hypothalamus that is essential for sexual behavior. While further investigations are needed, these initial studies seem to confirm that one's sense of gender resides in the **brain** and that it may be chemically determined.

In addition to biological factors, environmental conditions, such as socialization, seem to contribute to gender identity disorder. Social learning theory, for example, proposes that a combination of observational learning and different levels and forms of **reinforcement** by parents, family, and friends determine a child's sense of gender, which, in turn, leads to what society considers sex-appropriate or inappropriate behavior.

Symptoms

The onset of puberty increases the difficulties for people with gender identity disorder. The subsequent development of unwanted secondary sex characteristics, especially in males, increases a person's anxiety and frustrations. In an effort to cope with their feelings, some men with gender identity disorder may engage in stereotypical, or even super-masculine, activities. For example, a man struggling with the disorder may engage in such "macho" sports as wrestling and football in order to feel more "male." Unfortunately, the result is usually an increase in anxiety.

This anxious state is characterized by feelings of confusion, shame, guilt, and fear. These individuals are confused over their inability to handle their problem. They feel shame over their inability to control what society considers "perverse" activities. Even though cross-dressing and cross-gender fantasies provide relief, the respite is temporary. These activities often leave individuals with a profound shame over their thoughts and activities.

Closely associated with shame is guilt, particularly about being dishonest with family and friends. Sometimes people with gender identity disorder get married and have children without telling their spouse about their disorder. Typically, it is kept secret because they have the mistaken conviction that participation in marriage and parenting will eliminate or cure their gender identity problems. The fear of being discovered further raises their anxiety. With some justification, people with gender identity disorder fear being labeled "sick," and being rejected and abandoned by people they love.

If an individual's gender identity disorder is profound, a lifestyle change such as occasional cross-dressing may be insufficient. In such a case, gender expression may move from a lifestyle problem to a life-threatening imperative. The result can be extreme depression that requires medical treatment. If sufficiently severe, the imperative may result in gender reassignment surgery. If an individual lacks the psychological commitment to undertake surgery, the result may be suicide.

Demographics

Gender identity disorder is more prevalent in males than in females. Accurate estimates of prevalence for either males or females are not available.

Diagnosis

A mental health professional makes a **diagnosis** of gender identity disorder by taking a careful personal history. He or she obtains the age of the patient and determines whether the patient's sexual attraction is to males, females, both, or neither. Laboratory tests are neither available nor required to make a diagnosis of gender identity disorder. However, it is very important not to overlook a physical illness such as a tumor that might mimic or contribute to a psychological disorder. If there is any question that a physical problem might be the underlying cause of an apparent gender identity disorder, a mental health professional should recommend a complete physical examination by a medical doctor. Laboratory tests might be necessary as components of the physical evaluation.

According to the clinician's handbook for diagnosing mental disorders, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition text revised *(DSM-IV-TR)*, the following criteria must be met to establish a diagnosis of gender identity disorder. More specific descriptions and examples of the first two criteria follow the list.

- A strong and persistent cross-gender identification.

- Persistent discomfort with his or her sex or having a sense of inappropriateness in the gender role of one's birth sex.
- The disturbance is not concurrent with a physical intersex condition, such as hermaphroditism in which a person is born with the genitalia of both male and female.
- The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

A strong and persistent cross-gender identification

In children, the disturbance is manifested by four (or more) of the following:

- Repeatedly stating a desire to be, or insistence that he or she is, a member of the other sex.
- Strong preference for wearing clothes of the opposite gender. In boys, displaying a preference for cross-dressing or simulating female attire; in girls, insistence on wearing only stereotypical masculine clothing.
- Displaying strong and persistent preferences for cross-sex roles in make-believe play or experiencing persistent fantasies of being a member of the other sex.
- Having an intense desire to participate in the games and pastimes that are stereotypical of the other sex.
- Exhibiting a strong preference for playmates of the other sex.

Among adolescents and adults, the disturbance is manifested by symptoms such as a stated desire to become a member of the other sex, frequent passing as a person of the other sex, a desire to live or be treated as the other sex, or the conviction that he or she has the typical feelings and reactions of the other sex. These characteristics cannot be merely from a desire for any perceived cultural advantages of being the other sex.

Persistent discomfort with his or her sex or having a sense of inappropriateness in the gender role of one's birth sex

Among children, the disturbance is manifested by any of the following:

- Among boys, asserting that his penis or testes are disgusting or will disappear, asserting that it would be better not to have a penis, or having an aversion toward rough-and-tumble play and rejecting male stereotypical toys, games, and activities.
- Among girls, rejecting the gender-typical practice of urinating in a sitting position, asserting that she has or will grow a penis, or stating that she does not want to grow breasts or menstruate, or having a marked aversion toward normative feminine clothing.

Among adolescents and adults, the disturbance is manifested by symptoms such as preoccupation with getting rid of primary and secondary sex characteristics (request for hormones, surgery, or other procedures to alter sexual characteristics to simulate the other sex, for example) or a belief that he or she was born the wrong sex.

Treatments

One common form of treatment for gender identity disorder is **psychotherapy**. The earlier the intervention, the greater likelihood of success. Early intervention can lead to reduced levels of transsexual behavior later in life. The initial aim of treatment is to help individuals function in their biologic sex roles to the greatest degree possible.

Adults who have had severe gender identity disorder for many years sometimes request reassignment of their sex, or sex-change surgery. Before undertaking such surgery, they usually undergo hormone therapy to suppress same-sex characteristics and to accentuate other-sex characteristics. For instance, the female hormone estrogen is given to males to make breasts grow, reduce facial hair, and widen hips. The male hormone testosterone is administered to females to suppress menstruation, deepen the voice, and increase body hair. Following the hormone treatments, pre-operative candidates are usually required to live in the cross-gender role for approximately a year before surgery is performed.

Prognosis

If gender identity disorder persists into adolescence, it tends to be chronic in nature. There may be periods of remission. However, adoption of characteristics and activities appropriate for one's birth sex is unlikely to occur.

Prevention

Providing gender-appropriate clothing and toys in infancy and early childhood is helpful in preventing or mitigating gender identity disorder. Avoiding derogatory comments about a child's toy, clothing, or activity preference reduces the potential for inadvertent psychic harm.

Most individuals with gender identity disorder require and appreciate support from several sources. Families, as well as the person with the disorder, need and appreciate both information and support. Local and national **support groups** and informational services exist, and health care providers and mental health professionals can provide referrals.

Resources

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ORGANIZATIONS

American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org>.

American Academy of Pediatrics. 141 Northwest Point Boulevard, Elk Grove Village, IL 60007-1098. Telephone: (847) 434-4000. FAX: (847) 434-8000. Web site: <http://www.aap.org/default.htm>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. FAX: (202) 682-6850.

American Psychological Association. 750 First Street NW, Washington, DC, 20002-4242. Phone: (800) 374-2721 or (202) 336-5500. Web site: <http://www.apa.org>.

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Gender issues in mental health

Defining gender

The term *gender* is often used to classify the anatomy of a person's reproductive system as either male or female. In the social sciences, however, the concept of gender means much more than biological sex. It refers to socially constructed expectations regarding the ways in which one should think and behave, depending on sexual classification. These stereotypical expectations are commonly referred to as gender roles. Attitudes toward gender roles are thought to result from complex interactions among societal, cultural, familial, religious, ethnic, and political influences.

Gender affects many aspects of life, including access to resources, methods of coping with **stress**, styles of interacting with others, self-evaluation, spirituality, and expectations of others. These are all factors that can influence mental health either positively or negatively. Psychological gender studies seek to better understand the relationship between gender and mental health in order to reduce risk factors and improve treatment methods.

Traditional gender roles define masculinity as having power and being in control in emotional situations, in the workplace, and in sexual relationships. Acceptable male behaviors include competitiveness, independence, assertiveness, ambition, confidence, toughness, anger, and even violence (to varying degrees). Males are expected to avoid such characteristics associated with femininity as emotional expressiveness, vulnerability (weakness, helplessness, insecurity, worry), and intimacy (especially showing affection to other males).

Traditional femininity is defined as being nurturing, supportive, and assigning high priority to one's relationships. Women are expected to be emotionally expressive, dependent, passive, cooperative, warm, and accepting of subordinate status in marriage and employment. Competitiveness, assertiveness, anger, and violence are viewed as unfeminine and are not generally tolerated as acceptable female behavior.

Gender theories

Differences in gender roles have existed throughout history. Evolutionary theorists attribute these differences to the physiological characteristics of men and women that prescribed their best function for survival of the species. In primitive societies, men adopted the roles of hunting and protecting their families because of their physical strength. Women's ability to bear and nurse children led them to adopt the roles of nurturing young, as well as the less physically dependent roles of gathering and preparing food. These gender-dependent labor roles continued into the period of written human history, when people began to live in cities and form the earliest civilized societies.

In the 1800s, the industrial movement marked a prominent division of labor into public and private domains. Men began leaving home to work, whereas women worked within the home. Previously, both men

KEY TERMS

Active coping strategies—Ways of handling stress that affect the problem or situation in some way.

Androgyny—A way of behaving that includes high levels of both masculinity and femininity.

Antisocial behavior—Behavior characterized by high levels of anger, aggression, manipulation, or violence.

Avoidant coping strategies—Ways of coping with stress that do not alter the problem in any way, but instead provide temporary relief or distraction.

Externalizing disorders—Mental disorders with primary symptoms that involve outward behavior as opposed to inner emotions.

Femininity—Prescribed behavior for females, characterized by interpersonal warmth, passivity, and lack of aggression.

Gender role conflict or stress—A negative psychological state resulting from a discrepancy between gender role expectations and how one actually thinks, feels, or behaves.

Gender roles—Stereotypical expectations regarding how one should think, behave, and feel depending on whether one is male or female.

Internalizing disorders—Mental disorders with primary symptoms that involve inner emotions as opposed to outward behavior.

Machismo—The Latino image of extreme masculinity that includes such qualities as concern for personal honor, virility, physical strength, heavy drinking, toughness, aggression, risk taking, authoritarianism, and self-centeredness.

Masculinity—Prescribed behavior for males, characterized by independence, strength, control, and avoidance of emotional expressiveness.

Masochistic tendencies—Tendencies to direct harm or hatred towards oneself.

Object-relations theory—An approach to psychological development that includes Nancy Chodorow's stating that children develop according to interactions with their primary caregivers.

Psychoanalytic theory—A psychological theory proposed by Sigmund Freud involving unconscious conflicts and specific stages of development; central themes include sexuality and male superiority.

Socialization—The process whereby social influences and demands shape one's values, beliefs, or behavior.

and women frequently engaged in comparably respected, productive activities on their homestead. When men began working in the public domain, they acquired money, which was transferable for goods or services. Women's work, on the other hand, was not transferable. Men's relative economic independence contributed to their power and influence, while women were reduced to an image of frailty and emotionality deemed appropriate only for domestic tasks and child-rearing.

Sigmund Freud's psychoanalytic theory of human development, which emerged from Freud's late-nineteenth-century European setting and medical training, reflected an attitude of male superiority. Freud asserted that as children, boys recognize they are superior to girls when they discover the difference in their genitals. Girls, on the other hand, equate their lack of a penis with inferiority. This feeling of inferiority causes girls to idolize and desire their fathers, resulting in passivity, masochistic tendencies, jealousy and vanity—all seen by Freud as feminine characteristics.

Other developmental theorists rejected Freud's notions. Eric Erikson (in 1950) and Lawrence Kohlberg (in 1969) theorized that all humans begin as dependent on caregivers and gradually mature into independent and autonomous beings. Such theories, however, still favored males because independence has historically been considered a masculine trait. By such a standard, males would consistently achieve greater levels of maturity than females.

Nancy Chodorow's object-relations theory (in 1978) favored neither sex. She proposed that children develop according to interactions with their primary caregivers, who tend to be mothers. Mothers identify with girls to a greater extent, fostering an ability to form rich interpersonal relationships, as well as dependency traits. Mothers push boys toward independence, helping them to adjust to the male-dominated work environment, but rendering them unaccustomed to emotional connection. Chodorow's theory suggests both strengths and weaknesses inherent in male and female development, with neither deemed superior. Around that same time (1974), Sandra Bem advocated for

androgyny, or high levels of both masculinity and femininity, as the key to mental health.

In the 1980s, such psychologists as Carol Gilligan sought to build respect for stereotypically feminine traits. They introduced the notion that women function according to an ethic of care and relatedness that is not inferior to men—just different. In 1985, Daniel Stern's developmental theory favored traditional femininity, suggesting that humans start out as unconnected to others and gradually form more complex interpersonal connections as they mature.

Current gender studies appear less concerned with establishing male or female superiority. The general consensus seems to be that gender is socially constructed rather than biologically determined. The process of learning gender roles is known as socialization. Children learn which behaviors are acceptable or not acceptable for their sex by observing other people. They may also be shamed by caregivers or peers when they violate gender role expectations. As a result, gender roles usually become an internal guide for behavior early in childhood. Current studies focus on the ways in which extreme notions of masculinity or femininity affect mental health, and the social processes that shape one's concept of maleness or femaleness.

Gender role conflict

In current research, gender is viewed as an artificial (humanly constructed) concept that may not be related to biological sex at all. For example, masculinity and femininity may simply be sets of personality traits that can be exhibited by either sex. Individuals vary in degree of adherence to gender roles, resulting in large amounts of behavioral variation within the sexes.

Although attitudes toward gender roles are now much more flexible, different cultures retain varying degrees of expectations regarding male and female behavior. An individual may personally disregard gender expectations, but society may disapprove of his or her behavior and impose external social consequences. On the other hand, an individual may feel internal shame if he or she experiences emotions or desires characteristic of the opposite sex. Gender role conflict, or gender role stress, results when there is a discrepancy between how one believes he or she should act—based on gender role expectations learned in childhood—and how one actually thinks, feels, or behaves. If these discrepancies are unresolved, gender role conflict contributes to poor mental health.

Women's issues

Typical stressors

Women are often expected to occupy a number of roles at the same time: wife, mother, homemaker, employee, or caregiver to an elderly parent. Meeting the demands of so many roles simultaneously leads to stressful situations in which choices must be prioritized. Women are often forced to choose whether to pursue or further a career versus whether to devote more time to home and family.

Many women prefer to work outside the home because it gives them a greater sense of life satisfaction. For other women, such as those who run single-parent households, employment is not an option—it is a necessity. Compared with men, women are frequently given jobs with less autonomy or creativity, which decreases their level of job satisfaction. Women may also have more difficultly being accepted in the workplace because of hierarchical structures preferring men. Documentation repeatedly shows that women's salaries are lower than those of men in comparable positions; women tend to be paid less even when performing the same job as a man.

When women do choose or are required to work outside the home, they continue to perform the bulk of household duties as well. Sarah Rosenfield reported that compared to men, women perform 66% more of the domestic work, sleep one-half hour less per night, and perform an extra month of work each year. Needless to say, increased workloads and decreased attention to rest and relaxation are stressful and pose obstacles to women's mental health.

Divorce results in more severe consequences for women who choose or are able to stay home in deference to child-rearing. Such women depend on marriage for financial security. Such domestic skills as childcare and housecleaning are not highly valued by society, and thus are poorly compensated in terms of money. Women who have never been employed and then experience divorce often have few options for securing adequate income.

Although women's ability to form meaningful relationships is a buffer against stress, it can also be a source of stress. Caring about another person can be stressful when that person is not doing well physically or emotionally. Many families take for granted that the female members will care for elderly parents who are no longer self-sufficient. As a result, many women in their forties or fifties are caught between the needs of their college-age offspring and the needs of dependent parents or parents-in-law. Interpersonal conflicts resulting from these heavy burdens may cause stress or lower self-esteem. Women may also view unsuccessful relationships as rep-

resenting failure on their part to fulfill traditional feminine qualities such as nurturance, warmth, and empathy.

Additional sources of stress common to women include victimization, assertiveness, and physical unattractiveness. Victimization is a constant concern due to the power differential between men and women. Assertiveness may be stressful for women who have had little experience in competitive situations. Physical unattractiveness may cause some women who adhere to unrealistic standards of feminine beauty to experience shame, or place them at risk for developing eating disorders. Women considered unattractive may also suffer discrimination in the workplace or in admission to higher education. In addition, the double standard of aging in contemporary society means that all women will eventually have to cope with the **stigma** of unattractiveness simply through growing older.

Typical coping strategies

Studies suggest that women typically react to stress by seeking social support, expressing feelings, or using distraction. These strategies might include praying, worrying, venting, getting advice, or engaging in behaviors that are not related to the problem at all (including such antisocial behaviors as drinking alcohol). Seeking social support and distraction are considered avoidant coping strategies because they do not focus on solving or overcoming a problem, only on alleviating the stress associated with the problem. Research is inconclusive regarding whether men or women are more likely to use problem-solving, which is considered an active coping strategy.

Typical patterns of psychopathology

Women are more likely than men to experience internalizing disorders. Primary symptoms of internalizing disorders involve negative inner emotions as opposed to outward negative behavior. Depression (both mild and severe) and anxiety (generalized or "free-floating" anxiety, phobias, and panic attacks) are internalizing disorders common to women. Symptoms include sadness; a sense of loss, helplessness, or hopelessness; doubt about one's ability to handle problems; high levels of worry or nervousness; poor self-esteem; guilt, self-reproach, and self-blame; decreased energy, motivation, interest in life, or concentration; and problems with sleep or appetite.

Men's issues

Typical stressors

Situations that typically produce stress for men are those which challenge their self-identity and cause them to feel inadequate. If their identity closely matches a traditional male role, they will experience stress in situations requiring subordination to women or emotional expressiveness. They will also experience stress if they feel they are not meeting expectations for superior physical strength, intellect, or sexual performance. Research indicates that men who strictly adhere to extreme gender roles are at higher risk for mental disorders.

Certain cultures are thought to adhere more strictly to traditional male gender roles. In a study by Jose Abreu and colleagues, Latino men were identified as adopting the most exaggerated form of masculinity, followed by European Americans, and then African Americans. The Latino image of masculinity is often referred to as *machismo* and includes such qualities as concern for personal honor, virility, physical strength, heavy drinking, toughness, aggression, risk-taking, authoritarianism, and self-centeredness. African American males are also thought to have a unique image of masculinity; however, Abreu's study showed that African Americans are more egalitarian in terms of gender roles than European Americans.

Typical coping strategies

Men typically respond to stress by putting on a tough image, keeping their feelings inside, releasing stress through such activities as sports, actively attempting to solve the problem, denying the problem, abusing drugs or alcohol, or otherwise attempting to control the problem. As stated previously, research is inconclusive regarding whether males or females use problem-solving strategies more often. This type of coping strategy, however, has more frequently been attributed to males. Problem-solving is seen as an active coping strategy, which is more effective than such avoidant strategies as **denial**, abuse of drugs or alcohol, or refusing to talk about problems.

Typical patterns of psychopathology

Men are more likely than women to experience externalizing disorders. Externalizing disorders are characterized by symptoms involving negative outward behavior as opposed to internal negative emotions. Such externalizing disorders as substance abuse (both drugs and alcohol) and antisocial behavior (such as anger, hostility, aggression, violence, stealing, etc.) are common to men. Substance abuse results in such negative physical and social consequences as **hallucinations**, blackouts, physical dependency, job loss, divorce, arrests, organ and **brain** damage, and financial debt. Antisocial behavior impairs interpersonal relationships and can also result in negative consequences in other areas of life, such as run-ins with the criminal justice system.

Men are not exempt from such internalizing disorders as anxiety and depression. In fact, one study found that high levels of masculinity appear to be related to depression in males. Some researchers feel that men's abuse of substances could be considered the male version of depression. Because male gender roles discourage admitting vulnerability, men may resort to substance abuse as a way of covering their feelings.

Men who adhere to rigid gender roles are also at a disadvantage in interpersonal relationships, especially intimate relationships. They may avoid emotional expressiveness, or may behave in domineering and hostile ways. These behaviors increase their risk of social isolation, disconnection from nurturance, and participation in unhealthy relationships.

Mental health

Research indicates that, overall, neither males nor females are at greater risk for developing mental disorders as such. Being male or female may indicate susceptibility to certain types of disorders, however. Neither masculinity nor femininity is uniformly positive; both gender identifications have strengths and weaknesses. For example, femininity appears to be protective against antisocial behaviors and substance abuse, but is associated with high levels of avoidant coping strategies and low levels of achievement. Masculinity appears to be protective against depression, but is high in antisocial behavior and substance abuse.

Information about gender roles has implications for treatment. Women may not seek treatment because of lack of such resources as money, transportation, or time away from childcare duties. A treatment center sensitive to women's issues should seek to provide these resources in order to facilitate access to treatment. Men, on the other hand, may not seek treatment because it is incongruent with their image of masculinity. Therapists may need to offer men less threatening forms of treatment, such as those that focus on cognitive problem-solving rather than on emotions.

The focus of therapy may differ according to one's gender issues. Therapists should recognize the potential for shame and defensiveness when exploring gender norms. Externalizing behaviors may point to underlying hidden shame. For women, the importance placed on various roles in their lives and how closely those roles are tied to their self-identity is relevant. Men may be encouraged to connect to the spiritual aspects of their being and to consider less stringent views of masculinity. Therapists should also consider the associated influences of generation, culture, class, occupation, and educational level when exploring gender role issues.

Mental health is best achieved by maintaining a balance between masculine and feminine qualities. Taking either set of qualities to an extreme and to the exclusion of the other is detrimental. A non-traditional gender role orientation would combine the best of both genders: a social focus (reciprocally supportive relationships and a balance between interests of self and others) and active coping strategies.

Flexibility in using coping strategies is also important. Active, problem-focused coping strategies help to change the situation that is causing the problem. Avoidant or emotion-focused coping strategies manage or reduce emotional distress. Avoidant and emotion-focused strategies may be helpful for the immediate crisis, but should be used in combination with more active strategies for complete problem resolution.

See also Stress

Resources

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Society for the Psychology of Women. Division 35 Administrative Office. American Psychological Association, 750 First Street, NE, Washington, DC 2002-4242. (202) 336-6013. < http://www.apa.org/about/division/div35.html>.

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Sandra L. Friedrich, M.A.

Generalized anxiety disorder

Definition

Generalized anxiety disorder, or GAD, is a disorder characterized by diffuse and chronic worry. Unlike people with phobias or post-traumatic disorders, people with GAD do not have their worries provoked by specific triggers; they may worry about almost anything having to do with ordinary life. It is not unusual for patients diagnosed with GAD to shift the focus of their anxiety from one issue to another as their daily circumstances change. For example, someone with GAD may start worrying about finances when several bills arrive in the mail, and then fret about the state of his or her health when it is noticed that one of the bills is for health insurance. Later in the day he or she may read a newspaper article that moves the focus of the worry to a third concern.

A manual commonly used by mental health professionals is the ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM*. This manual may also be identified more specifically by edition, such as the *DSM,* fourth edition text revised, or *DSM-IV-TR*. The *DSM-IV-TR* classifies GAD as an anxiety disorder.

Description

Generalized anxiety disorder is characterized by persistent worry that is excessive and that the patient finds hard to control. Common worries associated with generalized anxiety disorder include work responsibilities, money, health, safety, car repairs, and household chores. The *ICD-10,* which is the European equivalent of *DSM-IV-TR,* describes the anxiety that typifies GAD as "free-floating," which means that it can attach itself to a wide number of issues or concerns in the patient's environment.

DSM-IV-TR specifies that the worry must occur "more days than not for a period of at least six months"; *ICD-10* states only that the patient "must have primary symptoms of anxiety most days for at least several weeks at a time, and usually for several months." The patient usually recognizes that his or her worry is out of proportion in its duration or intensity to the actual likelihood or impact of the feared situation or event. For example, a husband or wife may worry about an accident happening to a spouse who commutes to work by train, even though the worried partner knows objectively that rail travel is much safer than automobile travel on major highways. The anxiety level of a patient with GAD may rise and fall somewhat over a period of weeks or months but tends to become a chronic problem. The disorder typically becomes worse during stressful periods in the patient's life.

DSM-IV-TR specifies interference with work, family life, social activities, or other areas of functioning as a criterion for generalized anxiety disorder; *ICD-10* does not mention interference with tasks or other activities as a criterion for the disorder. Both diagnostic manuals mention such physical symptoms as **insomnia**, sore muscles, headaches, digestive upsets, etc. as common accompaniments of GAD, but only *DSM-IV-TR* specifies that an adult patient must experience three symptoms out of a list of six (restlessness, being easily fatigued, having difficulty concentrating, being irritable, high levels of muscle tension, and sleep disturbances) in order to be diagnosed with the disorder.

Patients diagnosed with GAD have a high rate of concurrent mental disorders, particularly major depres-

KEY TERMS

Anxiolytic—A preparation or substance given to relieve anxiety; a tranquilizer.

Ataque de nervios—A culture-specific anxiety syndrome found among some Latino groups in the United States and in Latin America. It resembles panic disorder in some respects but also includes dissociative symptoms, and frequently occurs in response to a stressful event.

Autonomic nervous system—The part of the nervous system that governs the heart, involuntary muscles, and glands.

Double anxiety—Acute anxiety from a recent stressful event combined with underlying persistent anxiety associated with generalized anxiety disorder.

Free-floating—A term used in psychiatry to describe anxiety that is unfocused or lacking an apparent cause or object.

Insidious—Proceeding gradually and inconspicuously but with serious effect.

Social modeling—A process of learning behavioral and emotional response patterns from observing one's parents or other adults. Some researchers think that social modeling plays a part in the development of generalized anxiety disorder in women.

Temperament—A person's natural disposition or inborn combination of mental and emotional traits.

Temporomandibular joint dysfunction—Condition resulting in pain in the head, face, and jaw. Muscle tension or abnormalities of the bones in the area of the hinged joint (the temporomandibular joint) between the lower jaw and the temporal bone are usually the cause.

Trait anxiety—A type of persistent anxiety found in some patients with generalized anxiety disorder. Trait anxiety is regarded as a feature (trait) of a person's temperament.

sion disorder, other anxiety disorders, or a substance abuse disorder. They also frequently have or develop such stress-related physical illnesses and conditions as tension headaches, irritable bowel syndrome (IBS), temporomandibular joint dysfunction (TMJ), bruxism (grinding of the teeth during sleep), and hypertension. In addition, the discomfort or complications associated with arthritis, diabetes, and other chronic disorders are often intensified by GAD. Patients with GAD are more likely to seek help from a primary care physician than a **psychiatrist**; they are also more likely than patients with other disorders to make frequent medical appointments, to undergo extensive or repeated diagnostic testing, to describe their health as poor, and to smoke tobacco or abuse other substances. In addition, patients with anxiety disorders have higher rates of mortality from all causes than people who are less anxious.

In many cases, it is difficult for the patient's doctor to determine whether the anxiety preceded the physical condition or followed it; sometimes people develop generalized anxiety disorder after being diagnosed with a chronic organic health problem. In other instances, the wear and tear on the body caused by persistent and recurrent worrying leads to physical diseases and disorders. There is an overall "vicious circle" quality to the relationship between GAD and other disorders, whether mental or organic.

Children diagnosed with GAD have much the same anxiety symptoms as adults. The mother of a six-year-old boy with the disorder told his pediatrician that her son "acted like a little man" rather than a typical first-grader. He would worry about such matters as arriving on time for school field trips, whether the family had enough money for immediate needs, whether his friends would get hurt climbing on the playground jungle gym, whether there was enough gas in the tank of the family car, and similar concerns. The little boy had these worries in spite of the fact that his family was stable and happy and had no serious financial or other problems.

GAD often has an insidious onset that begins relatively early in life, although it can be precipitated by a sudden crisis at any age above six or seven years. The idea that GAD often begins in the childhood years even though the symptoms may not become clearly noticeable until late adolescence or the early adult years is gaining acceptance. About half of all patients diagnosed with the disorder report that their worrying began in childhood or their teenage years. Many will say that they cannot remember a time in their lives when they were not worried about something. This type of persistent anxiety can be regarded as part of a person's temperament, or inborn disposition; it is sometimes called trait anxiety. It is not unusual, however, for people to develop the disorder in their early adult years or even later in reaction to chronic **stress** or anxiety-producing situations. For example, there are instances of persons developing GAD after several years of taking care of a relative with **dementia**, living with domestic violence, or living in close contact with a friend or relative with **borderline personality disorder**.

The specific worries of a person with GAD may be influenced by their ethnic background or culture. *DSM-IV-TR's* observation that being punctual is a common concern of patients with GAD reflects the value that Western countries place on using time as efficiently as possible. One study of worry in college students from different ethnic backgrounds found that Caucasian and African American students tended to worry a variable amount about a wider range of concerns whereas Asian Americans tended to worry more intensely about a smaller number of issues. Another study found that GAD in a community sample of older Puerto Ricans overlapped with a culture-specific syndrome called *ataque de nervios*, which resembles **panic disorder** but has features of other anxiety disorders as well as dissociative symptoms. (People experience dissociative symptoms when their perception of reality is temporarily altered—they may feel as if they were in a trance, or that they were observing activity around them instead of participating.) Further research is needed regarding the relationship between people's ethnic backgrounds and their outward expression of anxiety symptoms.

Causes and symptoms

Causes

The causes of generalized anxiety disorder appear to be a mixture of genetic and environmental factors. It has been known for some years that the disorder runs in families. Recent twin studies as well as the ongoing mapping of the human genome point to a genetic factor in the development of GAD. A gene related to panic disorder was identified in late 2001, which increases the likelihood that there is a gene or genes that govern susceptibility to generalized anxiety. The role of the family environment (social **modeling**) in an individual's susceptibility to GAD is uncertain. Social modeling, the process of learning behavioral and emotional response patterns from observing one's parents or other adults, appears to be a more important factor for women than for men.

Another factor in the development of GAD is social expectations related to gender roles. A recent Swiss study corroborated earlier findings that women have higher levels of emotional distress and lower quality of life than men. The higher incidence of GAD in women has been linked to the diffuse yet comprehensive expectations of women as caregivers. Many women assume responsibility for the well-being and safety of other family members in addition to holding a job or completing graduate or professional school. The global character of these responsibilities as well as their unrelenting nature has been described as a mirror image of the persistent but nonspecific anxiety associated with GAD.

Socioeconomic status may also contribute to generalized anxiety. One British study found that GAD is more closely associated with an accumulation of minor stressors than with any demographic factors. Persons of lower socioeconomic status, however, have fewer resources for dealing with minor stressors and so appear to be at greater risk for generalized anxiety.

One additional factor may be the patient's level of muscle tension. Several studies have found that patients diagnosed with GAD tend to respond to physiological stress in a rigid, stereotyped manner. Their autonomic reactions (reactions in the part of the nervous system that governs involuntary bodily functions) are similar to those of people without GAD, but their muscular tension shows a significant increase. It is not yet known, however, whether this level of muscle tension is a cause or an effect of GAD.

Symptoms

The symptomatology of GAD has changed somewhat over time with redefinitions of the disorder in successive editions of *DSM.* The first edition of *DSM* and *DSM-II* did not make a sharp distinction between generalized anxiety disorder and panic disorder. After specific treatments were developed for panic disorder, GAD was introduced in *DSM-III* as an anxiety disorder without panic attacks or symptoms of major depression. This definition proved to be unreliable. As a result, *DSM-IV* constructed its definition of GAD around the psychological symptoms of the disorder (excessive worrying) rather than the physical (muscle tension) or autonomic symptoms of anxiety. *DSM-IV-TR* continued that emphasis.

According to the *DSM-IV-TR,* the symptoms of GAD are:

- excessive anxiety and worry about a number of events or activities, occurring more days than not for at least six months
- worry that cannot be controlled
- worry that is associated with several symptoms such as restlessness, **fatigue**, irritability, or muscle tension
- worry that causes distress or impairment in relationships, at work, or at school

In addition, to meet the diagnostic criteria for GAD, the content or focus of the worry cannot change the **diagnosis** from GAD to another anxiety disorder such as panic disorder, **social phobia**, or **obsessive-compulsive disorder**, and the anxiety cannot be caused by a substance (a drug or a medication).

One categorization of GAD symptoms that some psychiatrists use in addition to the *DSM* framework consists of three symptom clusters:

- symptoms related to high levels of physiological arousal: muscle tension, irritability, fatigue, restlessness, insomnia
- symptoms related to distorted thinking processes: poor concentration, unrealistic assessment of problems, recurrent worrying
- symptoms associated with poor coping strategies: procrastination, avoidance, inadequate problem-solving skills

Demographics

It is difficult to compare present statistics for generalized anxiety disorder with those of the 1980s and early 1990s because of changes in the diagnostic criteria for GAD in successive editions of *DSM*. The National Institute of Mental Health (NIMH) states that as of 2000, 2.8% of the general United States population, or about four million people, have GAD during the course of a given year. One study that used *DSM-III-R* criteria concluded that 5% of the United States population, or one person in every 20, will develop GAD at some point in their lives. Another range of figures given for the lifetime prevalence of GAD in the American population is 4.1%–6.6%. The figure given for children in the United States is also 5%. Women develop generalized anxiety disorder more frequently than men; the sex ratio is variously given as 3:2 or 2:1. Prevalence across races and ethnic groups is more difficult to determine because of cultural influences on expressions of anxiety.

Some psychiatrists think that generalized anxiety disorder is overdiagnosed in both adults and children. One reason for this possibility is that diagnostic screening tests used by primary care physicians for mental disorders produce a large number of false positives for GAD. One study of the PRIME-MD, a screening instrument for mental disorders frequently used in primary care practices, found that 7% of patients met the criteria for GAD. Follow-up in-depth interviews with the patients, however, revealed that only a third of the GAD diagnoses could be confirmed.

Diagnosis

Diagnosis of GAD, particularly in primary care settings, is complicated by several factors. One is the high level of comorbidity (co-occurrence) between GAD and other mental or physical disorders. Another is the considerable overlap between anxiety disorders in general and depression. Some practitioners believe that depression and GAD may not be separate disorders after all, because studies have repeatedly confirmed the existence and common occurrence of a "mixed" anxiety/depression syndrome.

Evaluating a patient for generalized anxiety disorder includes the following steps:

- Patient interview. The doctor will ask the patient to describe the anxiety, and will note whether it is acute (lasting hours to weeks) or persistent (lasting from months to years). If the patient describes a recent stressful event, the doctor will evaluate him or her for "double anxiety," which refers to acute anxiety added to underlying persistent anxiety. The doctor may also give the patient a diagnostic questionnaire to evaluate the presence of anxiety disorders. The **Hamilton Anxiety Scale** is a commonly used instrument to assess anxiety disorders in general. The Generalized Anxiety Disorder Questionnaire for DSM-IV (GAD-Q-IV) is a more recent diagnostic tool, and is specific to GAD.
- Medical evaluation. Nonpsychiatric disorders that are known to cause anxiety (hyperthyroidism, Cushing's disease, mitral valve prolapse, carcinoid syndrome, and pheochromocytoma) must be ruled out, as well as certain medications (steroids, digoxin, thyroxine, theophylline, and selective serotonin reuptake inhibitors) that may also cause anxiety as a side effect. The patient should be asked about his or her use of herbal preparations as well.
- Substance abuse evaluation. Because anxiety is a common symptom of substance abuse and withdrawal syndrome, the doctor will ask about the patient's use of caffeine, nicotine, alcohol, and other common substances (including prescription medications) that may be abused.
- Evaluation for other psychiatric disorders. This step is necessary because of the frequent overlapping between GAD and depression or between GAD and other anxiety disorders.

In some instances the doctor will consult the patient's family for additional information about the onset of the patient's anxiety symptoms, dietary habits, etc.

Treatments

There are several treatment types that have been found effective in treating GAD. Most patients with the disorder are treated with a combination of medications and **psychotherapy**.

Medications

Pharmacologic therapy is usually prescribed for patients whose anxiety is severe enough to interfere with daily functioning. Several different groups of medications have been used to treat generalized anxiety disorder.

These medications include the following:

- Benzodiazepines. This group of tranquilizers does not decrease worry, but lowers anxiety by decreasing muscle tension and hypervigilance. They are often prescribed for patients with double anxiety because they act very quickly. The benzodiazepines, however, have several disadvantages: they are unsuitable for long-term therapy because they can cause dependence, and GAD is a long-term-disorder; they cannot be given to patients who abuse alcohol; and they cause short-term memory loss and difficulty in concentration. One British study found that benzodiazepines significantly increased a patient's risk of involvement in a traffic accident.
- **Buspirone** (BuSpar). Buspirone appears to be as effective as benzodiazepines and antidepressants in controlling anxiety symptoms. It is slower to take effect (about two–three weeks), but has fewer side effects. In addition, it treats the worry associated with GAD rather than the muscle tension.
- Tricyclic antidepressants. **Imipramine** (Tofranil), **nortriptyline** (Pamelor), and **desipramine** (Norpramin) have been given to patients with GAD. They have, however, some problematic side effects; imipramine has been associated with disturbances in heart rhythm, and the other tricyclics often cause drowsiness, dry mouth, constipation, and confusion. They increase the patient's risk of falls and other accidents.
- Selective serotonin reuptake inhibitors. **Paroxetine** (Paxil), one of the SSRIs, was approved by the Food and Drug Administration (FDA) in 2001 as a treatment for GAD. **Venlafaxine** (Effexor) appears to be particularly beneficial to patients with a mixed anxiety/depression syndrome; it is the first drug to be labeled by the FDA as an antidepressant as well as an anxiolytic. Venlafaxine is also effective in treating patients with GAD whose symptoms are primarily somatic (manifesting as physical symptoms, or bodily complaints).

Psychotherapy

Some studies have found cognitive therapy to be superior to medications and **psychodynamic psychotherapy** in treating GAD, but other researchers disagree with these findings. As a rule, GAD patients who have **personality disorders**, who are living with chronic social stress (are caring for a parent with **Alzheimer's disease**, for example), or who don't trust psychotherapeutic approaches require treatment with medications. The greatest benefit of cognitive therapy is its effectiveness in helping patients with the disorder to learn more realistic ways to appraise their problems and to use better problem-solving techniques.

Family therapy is recommended insofar as family members can be helpful in offering patients a different perspective on their problems. They can also help the patient practice new approaches to problem-solving.

Alternative and complementary therapies

Several alternative and complementary therapies have been found helpful in treating patients with generalized anxiety disorder. These include **hypnotherapy**; music therapy; Ayurvedic medicine; **yoga**; religious practice; and guided imagery **meditation**.

Biofeedback and relaxation techniques are also recommended for GAD patients in order to lower physiologic arousal. In addition, massage therapy, hydrotherapy, shiatsu, and **acupuncture** have been reported to relieve muscle spasms or soreness associated with GAD.

One herbal remedy that has been used in clinical trials for treating GAD is **passionflower** (*Passiflora incarnata*). One team of researchers found that passionflower extract was as effective as **oxazepam** (Serax) in relieving anxiety symptoms in a group of 36 outpatients diagnosed with GAD according to *DSM-IV* criteria. In addition, the passionflower extract did not impair the subjects' job performance as frequently or as severely as the oxazepam.

Prognosis

Generalized anxiety disorder is generally regarded as a long-term condition that may become a lifelong problem. Patients frequently find their symptoms resurfacing or getting worse during stressful periods in their lives. It is rare for patients with GAD to recover spontaneously.

Prevention

As of 2002, the genetic factors involved in generalized anxiety disorder have not been fully identified. In addition, the many stressors of modern life that raise people's anxiety levels are difficult to escape or avoid. The best preventive strategy, given the early onset of GAD, is the modeling of realistic assessment of stressful events by parents, and the teaching of effective coping strategies to their children.

See also Bodywork therapies; Cognitive-behavioral therapy; Cognitive problem-solving skills training; Stress

Resources

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ORGANIZATIONS

Anxiety Disorders Association of America. 11900 Parklawn Drive, Suite 100, Rockville, MD 20852-2624. (301) 231-9350. <www.adaa.org>.

Anxiety Disorders Education Program, National Institute of Mental Health. 6001 Executive Blvd., Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

Freedom From Fear. 308 Seaview Avenue, Staten Island, NY 10305. (718) 351-1717. <www.freedomfromfear.com>.

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Rebecca J. Frey, Ph.D.

Genetic factors and mental disorders

Introduction and overview

In recent years, mental health professionals have become increasingly aware of the importance of genetic factors in the etiology (causes) of mental disorders. Since the Human Genome Project began its mapping of the entire sequence of human DNA in 1990, the implications of its findings for psychiatric **diagnosis** and treatment have accumulated rapidly. A new subspecialty known as biological psychiatry (also called physiological psychology or psychiatric genetics) has emerged from the discoveries of the last two decades. Biological psychiatry got its start in the late 1980s, when several research groups identified genes associated with manic depression and **schizophrenia** respectively. These studies ran into difficulties fairly quickly, however, because of the com-

plexity of the relationship between genetic factors and mental illness.

The ongoing search for genes related to psychiatric symptoms and disorders is complicated by several factors:

- Psychiatric diagnosis relies on a doctor's human judgment and evaluation of a patient's behavior or appearance to a greater degree than diagnosis in other fields of medicine. For example, there is no blood or urine test for schizophrenia or a personality disorder. Diagnostic questionnaires for mental disorders are helpful in trimming the list of possible diagnoses but do not have the same degree of precision or objectivity as laboratory findings.
- Mental disorders almost always involve more than one gene. Studies have shown that one mental disorder can be caused by different genes on different chromosomes in different populations. For example, one study in the late 1980s found two genes on two different chromosomes among two populations that caused manic depression. Studies of schizophrenia done in the late 1980s and early 1990s revealed the same finding— different genes on different chromosomes produced schizophrenia in different populations. It now appears that specific mental disorders are related to different sets of genes that vary across family and ethnic groups.
- Genes associated with mental disorders do not always show the same degree of *penetrance*, which is defined as the frequency with which a gene produces its effects in a specific group of people. Penetrance is expressed as a percentage. For example, a gene for manic depression may have 20% penetrance, which means that 20% of the members of the family being studied are at risk of developing the disorder.
- Genetic factors in mental disorders interact with a person's family and cultural environment. A person who has a gene associated with susceptibility to alcohol abuse, for example, may not develop the disorder if he or she grows up in a family that teaches effective ways to cope with **stress** and responsible attitudes toward drinking.

There are several terms in biological psychiatry that are important to understand:

- Genotype: A person's *genotype* is the sum total of the genetic material transmitted from his or her parents.
- Phenotype: A person's *phenotype* is the observable signs, symptoms, and other aspects of his or her appearance. The term is also used sometimes to refer to a person's outward appearance and behavior as these result from the interaction between the person's genotype and his or her environment.
- Behavioral phenotype: The concept of a *behavioral phenotype* is used most often with reference to patterns of behavior found in certain developmental disorders of childhood, such as Down syndrome or Prader-Willi syndrome. Behavioral phenotype refers to the greater likelihood that people with a specific genetic syndrome will have certain behavioral or developmental characteristics compared to people who do not have the syndrome; it does not mean that every person diagnosed with a given genetic syndrome will invariably develop these characteristics.

Genetic causality in mental disorders

As of 2002, genes appear to influence the development of mental disorders in three major ways: they may govern the organic causes of such disorders as **Alzheimer's disease** and schizophrenia; they may be responsible for abnormalities in a person's development before or after birth; and they may influence a person's susceptibility to anxiety, depression, **personality disorders**, and substance abuse disorders.

One technological development that has contributed to the major advances in biological psychiatry in the last twenty years is high-speed computing. Faster computers have enabled researchers to go beyond rough estimates of the heritability of various disorders to accurate quantification of genetic effects. In some cases the data have led to significant reappraisals of the causes of specific disorders. As recently as the 1960s and 1970s, for example, schizophrenia was generally attributed to "refrigerator mothers" and a chilly emotional climate in the patients' extended families. As of 2002, however, the application of computer models to schizophrenia indicates that the heritability of the disorder may be as high as 80%. Another instance is **autism**, which was also blamed at one time on faulty parenting but is now known to be 90+% heritable.

Mental disorders with organic causes

The two most important examples of mental disorders caused by organic changes or abnormalities in the **brain** are late-onset Alzheimer's disease and schizophrenia. Both disorders are *polygenic*, which means that their expression is determined by more than one gene. Another disorder that is much less common, Huntington's disease, is significant because it is one of the few mental disorders that is *monogenic*, or determined by a single gene.

SCHIZOPHRENIA. Researchers have known for many years that first-degree biological relatives of patients with schizophrenia have a 10% risk of developing the disorder, as compared with 1% in the general population. The identical twin of a person with schizophrenia has a

KEY TERMS

Anticipation—In medicine, a phenomenon in which certain diseases manifest at earlier ages or in more severe phenotypes in each successive generation of an affected family.

Apolipoprotein E—A protein that transports cholesterol through the body. One form of this protein, apoE4, is associated with a 60% risk of late-onset AD.

Behavioral phenotype—A term that refers to the greater likelihood that people with a specific genetic syndrome will have certain behavioral or developmental characteristics, compared to people who do not have the syndrome.

Beta amyloid protein—A starchy substance that builds up in the brains of people with AD to form the plaques that are characteristic of the disease. Beta amyloid is formed when amyloid precursor protein, or APP, is not broken down properly by the body.

Bleomycin hydrolase—An enzyme involved in the body's processing of amyloid precursor protein. If the gene that governs production of BH mutates, the APP accumulates, producing the plaques in the brains of patients with AD.

Codon—A three-member nucleotide sequence in messenger RNA that codes for a specific amino acid in synthesizing protein molecules.

Cytogenetics—The branch of biology that combines the study of genetic inheritance with the study of cell structure.

Dizygotic—Developed from two fertilized ova. Dizygotic twins are sometimes called fraternal twins.

Down syndrome—A genetic disorder characterized by an extra chromosome 21 (trisomy 21), mental retardation, and susceptibility to early-onset Alzheimer's disease.

Epidemiology—The study of the causes, incidence, transmission, and control of diseases.

Etiology—The cause or origin of a disease or disorder. The word is also used to refer to the study of the causes of disease.

Exon—A segment of DNA that is transcribed to RNA and encodes information about the protein sequence.

Expansion mutation—A genetic mutation caused by additional repetitions of a triplet, or trinucleotide sequence, during the process of genetic transmission. In Huntington's disease, the expansion mutation produces more of a toxic gene product.

Genome—The total genetic makeup of a cell or organism. The human genome is the complete genetic constitution of a human being.

40%–50% risk. The first instance of a specific genetic linkage for schizophrenia, however, was discovered in 1987 by a group of Canadian researchers at the University of British Columbia. A case study that involved a Chinese immigrant and his 20-year-old nephew, both diagnosed with schizophrenia, led the researchers to a locus on the short arm of chromosome 5. In 1988, a study of schizophrenia in several Icelandic and British families also pointed to chromosome 5. Over the course of the next decade, other studies of families with a history of schizophrenia indicated the existence of genes related to the disorder on other chromosomes. In late 2001, a multidisciplinary team of researchers reported positive associations for schizophrenia on chromosomes 15 and 13. Chromosome 15 is linked to schizophrenia in European American families as well as some Taiwanese and Portuguese families. A recent study of the biological pedigrees found among the inhabitants of Palau (an isolated territory in Micronesia) points to chromosomes 2 and 13. Still another team of researchers has suggested that a disorder known as 22q deletion syndrome may actually represent a subtype of schizophrenia, insofar as people with this syndrome have a 25% risk of developing schizophrenia.

ALZHEIMER'S DISEASE. Late-onset Alzheimer's disease (AD) is unquestionably a polygenic disorder. It has been known since 1993 that a specific form of a gene for apolipoprotein E (apoE4) on human chromosome 19 is a genetic risk factor for late-onset Alzheimer's. People who have the apoE4 gene from one parent have a 50% chance of developing AD; a 90% chance if they inherited the gene from both parents. They are also likely to develop AD earlier in life. One of the remaining puzzles about this particular gene, however, is that it is not a consistent marker for AD. In other words, some people who have the apoE4 gene do not develop Alzheimer's, and some who do not have the gene do develop the disorder.

KEY TERMS CONTINUED

Genomic imprinting—The process in which specific genes or DNA segments are modified during the development of sperm or egg cells in a parent-specific fashion. The modification is reversible and appears to include the addition or removal of methyl groups to specific areas within the DNA sequence.

Genotype—The genetic makeup of an organism or a set of organisms.

Huntington's disease—A hereditary disorder that appears in middle age and is characterized by gradual brain deterioration, progressive dementia, and loss of voluntary movement. It is sometimes called Huntington's chorea.

Hyperphagia—An abnormally large appetite for food. Hyperphagia is one of the symptoms of Prader-Willi syndrome.

Intron—A segment of DNA that interrupts an exon and that does not encode any information about the protein sequence.

Monogenic—Determined or controlled by a single gene. Huntington's disease is one of the few psychiatric disorders that is monogenic.

Monozygotic—Developed from a single fertilized ovum. Monozygotic twins are sometimes called identical twins.

Nosology—The branch of medicine that deals with the systematic classification of diseases and disorders.

Nucleotide—One of the molecules that form the building blocks of DNA or RNA. The nucleotides of DNA include a phosphate group, four chemical bases (adenine, cytosine, guanine, and thymine), and a sugar containing five carbon atoms. In RNA the thymine base is replaced by uracil.

Penetrance—In genetics, the frequency with which a specific gene produces its effects in a group of people or other organisms. Penetrance is expressed as a percentage.

Phenotype—The observable signs, symptoms, and other aspects of the makeup of an organism. The term is also used sometimes to refer to the appearance of an organism resulting from the interaction between its genotype and its environment.

Polygenic—A trait or disorder that is determined by a group of genes acting together. Most human characteristics, including height, weight, and general body build, are polygenic. Schizophrenia and late-onset Alzheimer's disease are considered polygenic disorders.

Prader-Willi syndrome—A developmental disorder of childhood characterized by mental retardation; poor muscle tone; delayed growth and sexual maturation; and childhood onset of an abnormally large appetite for food.

In 1998, another gene on chromosome 12 that controls the production of bleomycin hydrolase (BH) was identified as a second genetic risk factor that acts independently of the apoE gene. In December 2000, three separate research studies reported that a gene on chromosome 10 that may affect the processing of amyloid-beta protein is also involved in the development of late-onset AD.

There are two other forms of AD, early-onset AD and familial Alzheimer's disease (FAD), which have different patterns of genetic transmission. Early-onset AD is caused by a defect in one of three genes known as APP, presenilin-1, and presenilin-2, found on human chromosomes 21, 14, and 1, respectively. Early-onset AD is also associated with Down syndrome, in that persons with trisomy 21 (three forms of human chromosome 21 instead of a pair) often develop this form of Alzheimer's. The brains of people with Down syndrome age prematurely, so that those who develop early-onset AD are often only in their late 40s or early 50s when the symptoms of the disease first appear. Familial Alzheimer's disease appears to be related to abnormal genes on human chromosomes 21 and 14.

HUNTINGTON'S DISEASE. Huntington's disease, or Huntington's chorea, is a neurological disorder that kills the cells in the caudate nucleus, the part of the brain that coordinates movement. It also destroys the brain cells that control cognitive functions. In 1983, the gene that causes Huntington's disease was discovered on the short arm of human chromosome 4. Ten years later, the gene was identified as an instance of a triplet or trinucleotide repeat. Nucleotides are the molecular "building blocks" of DNA and RNA. Three consecutive nucleotides form a codon, or triplet, in messenger RNA that codes for a specific amino acid. In 1991, researchers discovered not only that nucleotide triplets repeat themselves, but that these repetitions sometimes expand in number during the

process of genetic transmission. This newly discovered type of mutation is known as a dynamic or expansion mutation. Since 1991, more than a dozen diseases have been traced to expansion mutations. Eight of them are caused by repeats of the triplet cytosine-adenine-guanine (CAG), which codes for an amino acid called glutamine. In 1993, Huntington's disease was identified as a CAG expansion mutation disorder. Where the genetic material from a normal chromosome 4 has about 20 repeats of the CAG triplet, the Huntington's gene has a minimum of 45 repeats, sometimes as many as 86. The higher the number of CAG triplet repeats in a Huntington's gene, the earlier the age at which the symptoms will appear. The expansion mutation in Huntington's disease results in the production of a toxic protein that destroys the cells in the patient's brain that control movement and cognition.

Childhood developmental disorders

Developmental disorders of childhood are another large category of mental disorders caused by mutations, deletions, translocations (rearrangements of the arms of chromosomes) and other alterations in genes or chromosomes.

TRIPLET REPEAT DISORDERS. Since 1991, expansion mutations have been identified as the cause of thirteen different diseases. Some, like Huntington's disease, are characterized by long expansion mutations of the trinucleotide sequence CAG, which in effect adds so much glutamine to the protein being synthesized that it becomes toxic to the nervous system. A second category of triplet repeat disorders contains extra triplets that add an amino acid called alanine to the protein. The sequence of nucleotides is cytosine-guanine-N, where N stands for any of the four basic nucleotides. Although the proteins produced by this type of expansion mutation are not toxic, their normal function in the body is disrupted. The developmental disorders related to these CGN triplets are characterized by abnormalities of the skeleton. One of these disorders is synpolydactyly, in which the patient has more than the normal number of fingers or toes. Another is cleidocranial dysplasia, a disorder marked by abnormal development of the skull.

Other developmental disorders are caused by expansion mutations outside the regions of the gene that code for proteins. The segments of DNA that specify the sequence of a portion of a protein are known as *exons*, while the stretches of DNA that lie between the exons and do not code for proteins are called *introns*. The CAG and CGN groups of triplet disorders described in the preceding paragraph are expansion mutations that occur within exons. A third group of triplet disorders results from expansion mutations in introns. Expansions in this third group are usually much longer than those in the first two categories; some repeat several hundred or even several thousand times. The best-known expansion mutation in this group causes the disorder known as fragile X syndrome. Fragile X syndrome is the most common inherited form of **mental retardation** and should be considered in the differential diagnosis of any child with developmental delays, mental retardation, or learning difficulties. The syndrome is caused by a large expansion of a cytosine-guanine-guanine (CGG) repeat which interferes with normal protein transcription from a gene called the FMR1 gene on the X chromosome. Males with the mutation lack a second normal copy of the gene and are more severely affected than females who have a normal FMR1 gene on their second X chromosome. In both sexes there is a correlation between the length of the expansion mutation and the severity of the syndrome.

The discovery of expansion mutations was the solution to a long-standing genetic riddle. Clinicians had noticed as early as 1910 that some disorders produce a more severe phenotype or occur at earlier and earlier ages in each successive generation of an affected family. This phenomenon is known as *anticipation*, but its biological basis was not understood until recently. It is now known that triplet repeats that are long enough to cause disorders are unstable and tend to grow longer from generation to generation. For example, an expansion mutation of the cytosine-thymine-guanine (CTG) triplet causes a potentially life-threatening developmental disorder known as myotonic dystrophy. Repeats of the CTG triplet that are just above the threshold for myotonic dystrophy itself may produce a relatively mild disorder, namely eye cataracts in later life. Within two to three generations, however, the CTG repeats become longer, producing a fatal congenital illness. In addition to developmental disorders of childhood, expansion mutations may also be involved in other psychiatric disorders. Anticipation has been found in some families affected by **bipolar disorder** and schizophrenia, and some researchers think that it may also be present in some forms of autism.

GENOMIC IMPRINTING. Another recent discovery in the field of biological psychiatry is the phenomenon of genomic imprinting, which distinguishes between chromosomes derived from a person's father and those derived from the mother. Genomic imprinting was discovered in the late 1980s as an exception to Gregor Mendel's laws of biological inheritance. A small subset of human genes are expressed differently depending on the parent who contributes them to a child's genetic makeup. This phenomenon has helped researchers understand the causation of three well-known genetic disorders— Prader-Willi, Angelman, and Beckwith-Wiedemann syndromes.

In the 1980s, researchers studying Prader-Willi syndrome and Angelman syndrome noticed that both disor-

ders were caused by a deletion on the long arm of chromosome 15 in the very same region, extending from 15q11 to 15q13. This finding was surprising, because the two syndromes have markedly different phenotypes. Children with Prader-Willi syndrome have severe mental retardation, poor muscle tone, small hands and feet, and a voracious appetite (hyperphagia) that begins in childhood. As a result, they are often obese by adolescence. Children with Angelman syndrome, on the other hand, do not speak, are often hyperactive, and suffer from **seizures** and sleep disturbances. In the late 1980s, advances in molecular genetics revealed that the different expressions of the same deletion on the same chromosome were determined by the sex of the parent who contributed that chromosome. Children with Prader-Willi syndrome had inherited their father's copy of chromosome 15 while the children with Angelman syndrome had inherited their mother's. Highly specific diagnostic tests for these two disorders have been developed within the past decade.

Beckwith-Wiedemann syndrome is an overgrowth condition in which patients develop abnormally large bodies. They often have low blood sugar at birth and are at high risk for developing Wilms tumor, a childhood form of kidney cancer. Beckwith-Wiedemann syndrome is caused by several different genetic mutations that affect imprinted genes on chromosome 11p15. One of these imprinted genes governs the production of a growth factor that is responsible for the children's large body size.

BEHAVIORAL PHENOTYPES. Although medical professionals are familiar with the physical phenotypes associated with genetic disorders, the notion of behavioral phenotypes is still controversial. A behavioral phenotype is the characteristic set of behaviors found in patients with a genetic disorder. Behavioral phenotypes include patterns of language usage, cognitive development, and social adjustment as well as behavioral problems in the narrow sense. It is important for psychiatrists who treat children and adolescents to understand behavioral phenotypes, because they are better able to identify problem behaviors as part of a genetic syndrome and refer children to a geneticist for an accurate genetic diagnosis.

Examples of behavioral phenotypes are those associated with Down, Prader-Willi, and Williams syndromes. Children with Down syndrome have an increased risk of developing early-onset Alzheimer's disease. They are usually quiet and good-tempered, but may also be hyperactive and impulsive. Their behavioral phenotype includes delayed language development and moderate to severe mental retardation.

Children with Prader-Willi syndrome are often quiet in childhood but develop stubborn, aggressive, or impulsive patterns of behavior as they grow older. The onset of their hyperphagia is often associated with temper tantrums and other behavioral problems. They are typically obsessed with food, frequently hoarding it, stealing it, or stealing money to buy food. About 50% of children diagnosed with Prader-Willi syndrome meet the criteria for **obsessive-compulsive disorder** (OCD).

Williams syndrome is a genetic disorder that results from a deletion of locus 23 on chromosome 7q11. Children with this syndrome often have an "elf-like" face with short upturned noses and small chins. Their behavioral phenotype includes talkativeness, friendliness, and a willingness to follow strangers. They are also hyperactive and easily distracted from tasks. The personality profile of children with Williams syndrome is so distinctive that many are diagnosed on the basis of the behavioral rather than the physical phenotype.

Psychological/behavioral vulnerability in adults

Although psychiatrists at one time regarded emotional wounds in early childhood as the root cause of anxiety and depressive disorders in later life, inherited vulnerability to these disturbances is the subject of intensive study at the present time. In the past two decades, genetic factors have been shown to influence the likelihood of a person's developing mood disorders or post-traumatic syndromes in adult life. A study done in 1990 showed that first-degree relatives of a person diagnosed with major depression were two to four times as likely to develop depression themselves as people in the general population. As of 2002, however, the genetic patterns involved in depression appear to be quite complex; there is some evidence that both genomic imprinting and the phenomenon of anticipation may be present in some families with multigenerational histories of depression. In addition, the evidence indicates that susceptibility to major depression is governed by several different genes on several different chromosomes. At present, genetic factors are thought to account for about 40% of a person's risk of depression, with environmental factors and personal temperament accounting for the remaining 60%.

With regard to manic depression, twin studies have shown that the twin of a patient diagnosed with manic depression has a 70%–80% chance of developing the disorder. As of January 2002, a team of German researchers studying 75 families with a total of 275 members diagnosed with manic depression (out of 445 persons) has narrowed its search for genes for manic depression to one locus on human chromosome 10 and another on the long arm of chromosome 8.

POST-TRAUMATIC SYNDROMES. Researchers have found that some persons are more vulnerable than others to developing dissociative and anxiety-related symptoms

following a traumatic experience. Vulnerability to trauma is affected by such inherited factors as temperament as well as by family or cultural influences; shy or introverted persons are at greater risk for developing **post-traumatic stress disorder** (PTSD) than their extroverted or outgoing peers. In addition, twin studies indicate that certain abnormalities in brain hormone levels and brain structure are inherited, and that these increase a person's susceptibility to developing **acute stress disorder** (ASD) or PTSD following exposure to trauma.

ANXIETY DISORDERS. It has been known for some time that anxiety disorders tend to run in families. Recent twin studies as well as the ongoing mapping of the human genome point to a genetic factor in the development of **generalized anxiety disorder** (GAD). One study determined the heritability of GAD to be 0.32.

Recent research has also confirmed earlier hypotheses that there is a genetic component to **agoraphobia**, and that it can be separated from susceptibility to **panic disorder** (PD). In 2001 a team of Yale geneticists reported the discovery of a genetic locus on human chromosome 3 that governs a person's risk of developing agoraphobia. Panic disorder was found to be associated with two loci, one on human chromosome 1 and the other on chromosome 11q. The researchers concluded that agoraphobia and PD are common, heritable anxiety disorders that share some but not all of their genetic loci for susceptibility.

BEHAVIORAL TRAITS. There has been considerable controversy in the past decade concerning the mapping of genetic loci associated with specific human behaviors, as distinct from behavioral phenotypes related to developmental disorders. In 1993 a group of Dutch researchers at a university-affiliated hospital in Nijmegen reported that a mutation in a gene that governs production of a specific enzyme (monoamine oxidase A or MAOA) appeared to be the cause of violent antisocial behavior in several generations of males in a large Dutch family. At least fourteen men from this family had been in trouble with the law for unprovoked outbursts of aggression, ranging from arson and attacks on employers to sexual assaults on female relatives. Tests of the men's urine indicated that **neurotransmitters** secreted when the body responds to stress were not being cleared from the bloodstream, which is the normal function of MAOA. In other words, the genetic mutation resulted in an overload of stress-related neurotransmitters in the men's bodies, which may have primed them to act out aggressively. As of 2002, however, the Dutch findings have not been replicated by other researchers.

Another controversial study in the early 1990s concerned the possible existence of "gay genes" as a factor in human homosexuality. A researcher at the Salk Institute found that cells in the hypothalamus, a structure in the brain associated with the regulation of temperature and sleep cycles, were over twice as large in heterosexual males as in homosexual men. Although the researcher acknowledged that the structural differences may have arisen in adult life and were not necessarily present at birth, he raised the possibility that sexual orientation may have a genetic component. Another study of affected sibling pairs reported a possible locus for a "gay gene" on the X chromosome, but as of 2002 the results have not been replicated elsewhere.

In general, however, research into the genetic component of human behavior is presently conducted with one eye, so to speak, on the social and political implications of its potential results. Given contemporary concerns about the misuse of findings related to biological race or sex, investigators are usually careful to acknowledge the importance of environmental as well as genetic factors.

Genetic epidemiology

Genetic epidemiology is the branch of medicine that investigates the incidence and prevalence of genetic disorders in specific populations. Researchers in this field make use of specific types of studies in order to assess the relative importance of genetic and environmental factors in families with a history of inherited disorders.

Twin studies

Twin studies are based on the assumption that twins reared in the same family share a common environment. Monozygotic (identical) twins have all their genes in common, whereas dizygotic (fraternal) twins share only half their genes. If a certain disorder appears more frequently in monozygotic twins of affected persons than in dizygotic twins, one may assume that the difference is due to genetic factors rather than the family environment. Some phenotypes show clear differences between identical and fraternal twins, including schizophrenia, childhood autism, **attention-deficit/hyperactivity disorder**, unipolar depression, manic depressive disorder, and cognitive abilities as measured by IQ tests.

Twin studies have proved to be particularly important in genetic research into autism. Until the early 1970s, autism was assumed to be caused primarily by parental coldness toward the child. In part, the lack of interest in genetic aspects of the disorder was due to the fact that cytogenetic research (research that studies the links between genetic inheritance and cell structure) was not sufficiently advanced in the late 1960s to have demonstrated any chromosomal abnormalities in people diag-

nosed with autism. The first small-scale twin study of children with autism was done in 1977; its findings showed, first, that there is a significant difference between monozygotic and dizygotic twin pairs with regard to the appearance of the disorder in siblings. More importantly, the study showed that the similarities within monozygotic pairs of twins included a range of social and cognitive disabilities, not just autism itself. This finding implied that the phenotype of autism is broader than the older diagnostic categories implied. In the 1970s and 1980s, advances in cytogenetic techniques led to the discovery that autism is associated with several different chromosomal abnormalities, including the defect that produces fragile X syndrome. A much larger British twin study done in 1995 confirmed earlier findings in the United States: a monozygotic twin of a child diagnosed with autism was 12 times more likely to have the disorder than a dizygotic twin (60% vs 5%). Secondly, the British study confirmed the hypothesis that the genetic risk of autism extends to a broader phenotype; over 90% of the monozygotic twin pairs in the British study shared social and intellectual disabilities similar to those found in patients with autism, but less severe.

Adoption studies

Adoption studies are used less frequently than twin studies, but are crucial in researching such conditions as schizophrenia and alcoholism. Studies of schizophrenia done in Denmark, for example, showed that the frequency of schizophrenia was 16% in the biological relatives of patients with schizophrenia who had been adopted as infants, compared with 1.8% in the adoptive relatives and the relatives of a group of adopted children who did not have schizophrenia.

Family studies

Family studies are important tools for evaluating environmental effects on children with genetic disorders— and also for evaluating the impact of the disorder on the family environment. Family studies have indicated that families may develop problems in response to a child's illness as well as affecting the child's prognosis for recovery.

Family factors fall into three categories: shared genetic material; shared environment; and nonshared environment. These three categories are complicated, however, by the fact that genetic as well as environmental factors affect interactions between parents and children. For example, a parent's behavior toward a child diagnosed with depression is partly shaped by the parent's genetic vulnerability to depression.

In general, much of the impact of a family's environment on a child with a mental disorder is due to nonshared rather than shared interactions. A clinical research measurement called expressed emotion, or EE, originally developed to study young adults with schizophrenia, is now used to study families with younger children with mental disorders. EE measures three primary aspects of family members' attitudes toward the child with the illness: criticism, hostility, and emotional overinvolvement. A growing number of research studies indicate that EE is a good predictor of the outcome of the child's illness; high EE is a marker of a more difficult course of the disorder and a poorer prognosis.

Clinical applications of biological psychiatry

As of 2002, recent advances in genetics have affected the practice of psychiatry in several ways:

- Genetic counseling. Genetic counseling is recommended when a couple has already produced a child with mental retardation, dysmorphic (malformed) features, or developmental delays; when either parent is suspected or known to have a genetic disorder; when the mother is over 35; when there is a family history of a genetic disorder, especially if several members are affected; or if the mother has been exposed during pregnancy to drugs or environmental toxins known to cause birth defects. Genetic counselors do not try to control the couple's decision about a present or future pregnancy; rather, they offer information about the disorder, including treatment options as well as the risk of recurrence. They discuss possible reproductive choices available to the couple and help them adjust to caring for a child who is already affected.
- Medication selection and dosage. Preliminary studies of patients with schizophrenia indicate that DNA testing of the gene for a specific serotonin receptor can predict the patient's response to antipsychotic drugs. A similar form of gene testing can predict which children with asthma will respond to an inhaled medication known as albuterol and which will not. In the near future, researchers hope to devise genetic tests that will measure patients' responsiveness to specific antidepressant and anti-anxiety medications. Such tests would greatly simplify the present process of trial-and-error prescribing of drugs for psychiatric disorders.
- Psychiatric nosology. Nosology is the branch of psychiatry that deals with the classification of mental disorders. Some current diagnostic labels, including autism and attention-deficit/hyperactivity disorder, may represent groups of related syndromes rather than a single diagnostic entity. In other instances, genetic

studies may lead to eventual reclassification of certain disorders. Some studies, for example, suggest that **body dysmorphic disorder** is more closely related to obsessive-compulsive disorder than to the somatoform disorders with which it is presently grouped.

- Diagnosis of disorders with major psychological consequences. As of 2002, it is possible for people to find out whether they have the gene for Huntington's disease or the BRCA1 or BRCA2 genes for breast cancer. Although some people may choose not to know, others may prefer the possibility of bad news to years of chronic uncertainty and anxiety.

Ethical concerns

As the number of tests available for determining genetic markers for mental disorders continues to increase, ethical issues are being debated. These concerns include:

- Regulation of genetic testing. Some companies have started to market tests for the apoE4 Alzheimer's gene even though the present benefits of such testing are not clear. The Department of Health and Human Services has established an advisory committee to study the question of government regulation of genetic testing.
- Confidentiality. The fear of losing health insurance is a major barrier to acceptance of genetic testing in the general population. Many people do not trust hospitals or research laboratories to keep test results confidential.
- Discrimination. Others are concerned that genetic findings could be used to deny college or graduate school admission to persons at risk for certain disorders, or to restrict their access to employment opportunities.
- Reproductive issues. A recent controversy in the *British Medical Journal* erupted over the question of using genetic testing for "social engineering" by forcing couples to abort fetuses with "undesirable" psychological characteristics or restricting people's right to have children. As more and more human traits are found to have a genetic component, questions inevitably arise regarding the possibility of government control over reproduction. But while few people would want to preserve the gene for Huntington's disease, for example, they are likely to disagree about the desirability of other human traits, such as a tendency toward short stature.

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National Human Genome Research Institute of the National Institutes of Health. <www.nhgri.nih.gov>.

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Geodon *see* **Ziprasidone**

Geriatric Depression Scale

Definition

The Geriatric Depression Scale (GDS) is a 30-item self-report assessment designed specifically to identify depression in the elderly. The items may be answered yes or no, which is thought to be simpler than scales that use a five-category response set. It is generally recommended as a routine part of a comprehensive geriatric assessment. One point is assigned to each answer and corresponds to a scoring grid. A score of 10 or 11 or lower is the usual threshold to separate depressed from nondepressed patients. However, a **diagnosis** of clinical depression should not be made on the GDS results alone. Although the test has well-established reliability and validity, responses should be considered in conjunction with other results from a comprehensive diagnostic work-up. A short version of the GDS containing 15 questions has been developed. The GDS is also available in a number of languages other than English.

Purpose

Depression is widespread among elderly persons, affecting one in six patients treated in general medical practice and an even higher percentage of those in hospitals and nursing homes. Older people have the highest **suicide** rate of any group, and many medical problems common to older people may be related to, or intensified by, a depressive disorder. Recognition of the prevalence of depression among older people prompted the development of the geriatric depression scale in 1982-83. Yes/no responses are thought to be more easily used than the graduated responses found on other standard assessment scales such as the **Beck Depression Inventory**, the

KEY TERMS

Low affect—Severe lack of interest and emotions; emotional numbness.

Somatic concern—Excessive concern about the body, particularly in relation to illness.

Hamilton rating scale for depression, or the Zung self-rating depression scale.

While it is not found in the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*) produced by the American Psychiatric Association, the GDS is widely recommended for clinical use and is included as a routine part of a comprehensive geriatric assessment. It is also increasingly being used in research on depression in the elderly.

Precautions

Depression scales are either interviewer-administered or by self-report means. The GDS is a self-report assessment developed in 1982 by J. A. Yesavitch and colleagues. A self-report assessment is easier and quicker to administer, though an interviewer-administered test is generally more sensitive and specific—another reason for using more than one tool to obtain an accurate diagnosis.

There is some controversy over whether the GDS is reliable for depression screening in individuals with mild or moderate **dementia**. Several studies have shown good agreement with observer ratings of depression, whether or not the patient had dementia. However, persons with dementia may deny symptoms of depression. It also appears that less educated people are more likely to score in the depressed range on the GDS 15-item short form. These caveats notwithstanding, the GDS can be usefully applied in general medical settings in combination with other clinical assessments, observation, and interviews with elder patient and their families.

Both symptom pattern and symptom severity must be considered when trying to identify depression. These dimensions are taken into account in the development of symptom scales and, while clinical judgment takes priority, a scale such as the GDS can help in identifying persons with depression, whether they are making satisfactory progress with treatment, or when they may need further assessment or referral.

Description

Yesavitch and his coworkers chose 100 statements that they determined were related to seven common characteristics of depression in later life. These included:

- somatic concern
- lowered **affect** (affect is the outward expression of emotion)
- cognitive impairment
- feelings of discrimination
- impaired motivation
- lack of future orientation
- lack of self-esteem

The best 30 items were selected after administration of the 100 items to 46 depressed and normal elders. Those items were then administered to 20 elders without depression and 51 who were in treatment for depression. The test was 84% sensitive and 95% specific for a depression diagnosis. Repeated studies have demonstrated the value of GDS.

Examples of the questions in the GDS include:

- Are you basically satisfied with your life?
- Have you dropped many of your activities and interests?
- Are you hopeful about the future?
- Do you often get restless and fidgety?
- Do you frequently get upset over little things?
- Do you enjoy getting up in the morning?

A time frame should be specified for administration of the test, for example, "Answer these questions by thinking of how you've felt the past two weeks."

Results

A scoring grid accompanies the GDS. One point is given for each respondent's answer that matches those on the grid. For example, the grid response to "Are you basically satisfied with your life?" is "no." If the elderly person responds in the negative one point is scored; if the response is "yes," then no point is scored. For the 30-item assessment, a score of 0–9 is considered normal; 10–19 indicates mild depression, and a score over 20 is suggestive of severe depression. The maximum number of points that can be scored is 30.

See also Depression and depressive disorders

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Judy Leaver, M.A.

Gestalt therapy

Definition

Gestalt therapy is a complex psychological system that stresses the development of client self-awareness and personal responsibility.

Purpose

The goal of Gestalt therapy is to raise clients' awareness regarding how they function in their environment (with family, at work, school, friends). The focus of therapy is more on what is happening (the moment-to-moment process) than what is being discussed (the content). Awareness is being alert to what are the most important events in clients' lives and their environment with full sensorimotor, emotional, cognitive, and energy support. Support is defined as anything that makes contact with or withdrawal from with the environment possible, including energy, body support, breathing, information, concern for others, and language, for example.

In therapy, clients become aware of what they are doing, how they are doing it, and how they change themselves, and at the same time, learn to accept and value themselves. Individuals, according to this approach, define, develop, and learn about themselves in relationship to others, and that they are constantly changing.

Gestalt therapy is "unpredictable" in that the therapist and client follow moment-to-moment experience and neither knows exactly where this will take them. Gestalt therapy is complex and intuitive, but it is based on the following principles:

- Holism. Gestalt therapy takes into account the whole person including thoughts, feelings, behavior, body sensations, and dreams. The focus is on integration, that is, how the many parts of the person fit together, and how the client makes contact (interacts) with the environment.
- Field theory. According to this theory, everything is related, in flux, interrelated, and in process. The therapist focuses on how the client makes contact with the environment (family, work, school, friends, authority figures).
- The figure-formation process describes how individuals organize or manipulate their environment from moment to moment.
- Organismic self-regulation is the creative adjustment that the organism (person) makes in relation to the environment. The person's equilibrium with his or her environment is "disturbed" by the emergence of a client need, sensation, or interest and is related to the figure-formation process in that the need of the person organizes the field. For example, if an individual wants coffee, this coffee need is what comes out of the defused background and becomes "figural" (comes to the forefront of the client's environment or field) and when the individual enters a room, the "figural" will be related to the coffee need. The therapist is interested in what is "figural" for a person because it may provide insight into the person's need(s).
- The Now. The concept of the here and now is what is being done, thought, and felt at the moment, and not in the past or the future.
- Unfinished business is defined as the unexpressed feelings that are associated with distinct memories and fantasies. These feelings may be resentment, rage, hatred, pain, anxiety, **grief**, guilt, and abandonment that are not fully experienced in awareness, linger in the background, and are carried into the present life and cause preoccupations, compulsive behaviors, wariness, and other self- defeating behaviors. Unfinished business will persist until the person faces and deals with these denied or alienated feelings.

The current practice of Gestalt therapy includes treatment of a wide range of problems and has been successfully employed in the treatment of a wide range of "psychosomatic" disorders including migraine, ulcerative colitis, and spastic neck and back. Therapists work with couples and families, and with individuals who have difficulties coping with authority figures. In addition, Gestalt therapy has been used for brief **crisis intervention**, to help persons with **post-traumatic stress disorders**, alcohol and drug abuse, depression, or anxiety disorders; with adults in a poverty program; with seriously mentally ill individuals with psychotic disorders; and those with borderline **personality disorders**.

Description

The relationship between the therapist and the client is the most important aspect of **psychotherapy** in Gestalt therapy. In Gestalt therapy, the interaction between therapist and client is an ever changing dialogue marked by straightforward caring, warmth, acceptance, and self-responsibility. There are four characteristics of dialogue:

- Inclusion, in which the therapist puts him- or herself, as much as is possible, into the experience of the client. The therapist does not judge, analyze, or interpret what he or she observes.
- Presence refers to the therapist expressing his or her observations, preferences, feelings, personal experience, and thoughts to the client.
- Commitment to dialogue allows a feeling of connection (contact) between the therapist and the client.
- Dialogue is active and can be nonverbal as well as verbal. It can be dancing, song, words, or any modality that expresses and moves the energy between the therapist and the client.

Gestalt therapy holds the view that people are endlessly remaking or discovering themselves; therefore, individuals are always in constant transformation. The therapist's approach is to help clients: to increase or deepen their awareness of themselves and with aspects of themselves and their relationship with others, by attending and engaging with the client; to explore the client's experience; and to describe what is. All techniques used within the therapeutic relationship help clients to work through and move beyond painful emotional blocks and is an ongoing process. This allows the client to explore new behavior, first, in the context of the therapeutic relationship and then, as appropriate, in the outside world.

The therapeutic process begins at the first contact between client and therapist. The assessment method for the Gestalt therapist has been unique to Gestalt therapy theory, as well as some psychodynamic treatments, and other humanistic treatments. Assessment and screening are usually done as part of the ongoing relationship with the client and not as a separate period of diagnostic testing and history taking. Assessment information is obtained by beginning the therapeutic work and includes: the client's willingness and support for work in the Gestalt therapy framework, the match between the client and the therapist, diagnostic and personality information, the decision regarding the frequency of sessions, the need for adjunctive treatment (such as day treatment, **biofeedback** training), and the need for medication and medical consultation.

Gestalt therapists now make use of the traditional diagnostic categories to obtain necessary information to help patients with serious mental illnesses (such as psychotic disorders and borderline disorders) and because of administrative and insurance reimbursement procedures. Despite these changes, it is believed that Gestalt therapy assessment techniques will continue to be varied since Gestalt therapists draw on other therapeutic systems.

In therapy, the Gestalt therapist is active and sessions are lively and characterized by warmth, acceptance, caring, and self-responsibility and promote direct experiencing of a situation or event rather than passively talking about the event. Events recalled from the past are explored and felt in the here and now of the therapy session. Clients can see, hear, and be told how they are seen, what is seen, how the therapist feels, what the therapist is like as a person, and how client awareness is limited by how they and the therapist interact with or engage each other— that is, make contact.

The Gestalt therapist has a wide range of active interventions (cognitive and behavioral) at his or her disposal and may use any technique or method as long as it is (a) aimed toward increasing awareness, (b) arises out of the dialogue and the therapist's perception of what is going on with the client (sensing, feeling, thinking) in the immediate therapy session), and (c) within the parameters of ethical practice.

Exercises and experiments

Many therapeutic interventions called exercises and experiments have been developed to enhance awareness and bring about client change. Exercises are defined as ready-made techniques that are sometimes used to evoke certain emotions (such as the expression of anger) in clients. Experiments, on the other hand, grow out of the immediate interaction (dialogue) between client and therapist. They are spontaneous, one-of-a-kind, and relevant to a particular moment and the particular development of an emerging issue such as the client's reports of a need, dream, fantasy, and body awareness. Experiments are

done with full participation and collaboration with clients and are designed to expand clients' awareness and to help them to try out new ways of behaving rather than to achieve a particular result. These experiments may take many forms. According to Gerald Corey, some are: "imagining a threatening future event; setting up a dialogue between a client and some significant person in his and her life; dramatizing the memory of a painful event; reliving a particularly profound early experience in the present; assuming the identity of one's mother or father through role-playing; focusing on gestures, posture, and other nonverbal signs of inner expression; carrying on a dialogue between two conflicting aspects within the person."

While participating in experiments, clients actually experience the feelings associated with their conflicts or issues in the here and now. Experiments are tailored to each individual client and used in a timely manner; they are to be carried out in a context that offers safety and support while encouraging the client to risk trying out new behavior. The Gestalt therapy focus is on the entire person and all parts—verbal and nonverbal behaviors, emotional feelings— all are attended to.

Gestalt therapists are said to rely on spontaneity, inventiveness, and "present-centeredness" and a range of possible therapeutic encounters, interactions that leads to exercises and experiments that are potentially infinite but can be categorized as follows.

THE USE OF STATEMENTS AND QUESTIONS TO FOCUS AWARENESS. Many interventions have to do with simply asking "what the client is aware of experiencing;" or asking simple and direct questions as, "What are you feeling?" "What are you thinking?" The client may be instructed to start a sentence with "Now, I am aware..." or is asked to repeat a behavior, as in, "Please wring your hands together again." A frequent technique is to follow the client's awareness report with the instruction, "Stay with it!" or "Feel it out!"

CLIENT'S VERBAL BEHAVIOR OR LANGUAGE. Awareness can be enhanced and emphasized through the client's verbal behavior or language since client speech patterns are considered to be an expression of their feelings, thoughts, and attitudes. Some aspects of language that might indicate the clients' avoidance of strong emotions or of self-responsibility are the general pronouns such as "it" and "you." Clients are instructed to substitute, when appropriate, the personal pronoun "I" for these pronouns to assume a sense of responsibility for his or her feelings or thoughts (ownership). Sometimes clients may be asked to change their questions into direct statements in order to assume responsibility for what they say. Other examples of helping clients to be more in control using language are to have them omit qualifiers and disclaimers such as "maybe," "perhaps," or "I guess" from their language patterns. This changes ambivalent and weak statements into more clear and direct statements; to substitute "I won't" for "I can't" because often "can't" gives the feeling of being unable to do something. It may be more accurate to say "I won't" meaning "I choose not to do this for any of various reasons," or use the word, "want" instead of "need" which is considered an indication of urgency and anxiety, and is less accurate. Other changes might be to change "should" and "ought" to "I choose to" or "I want to" increasing the clients' power and control of their lives.

NONVERBAL BEHAVIOR. Awareness can also be enhanced by focusing on nonverbal behavior and may include any technique that makes the clients more aware of their body functioning or helps them to be aware of how they can use their bodies to support excitement, awareness, and contact. The parts of the body that therapists may attend to include the mouth, jaw, voice, eyes, nose, neck, shoulders, arms, hands, torso, legs, feet, and the entire body. The therapist, for example, may point out to and explore with the client how he or she is smiling while at the same time expressing anger.

SELF-DIALOGUE. Self-dialogue by clients is an **intervention** used by Gestalt therapists that allows clients to get in touch with feelings that they may not be unaware of and, therefore, increase the integration of different parts of clients that do not match or conflicts in clients. Examples of some common conflicts include "the parent inside versus the child inside, the responsible one versus the impulsive one, the puritanical side versus the sexual side, the 'good side' versus the 'bad side,' the aggressive self versus the passive self, the autonomous side versus the resentful side, and the hard worker versus the goof-off." The client is assisted in accepting and learning to live with his or her polarities and not necessarily getting rid of any one part or trait.

The client is engaged in the self-dialogue by using what is called the empty-chair technique. Using two chairs, the client is asked to take one role (for example, the parent inside) in one chair and then play the other role (for example, the child inside) in the second chair. As the client changes roles and the dialogue continues between both sides of the client he or she moves back and forth between the two chairs. Again according to Corey, other examples of situations in which dialogues can be used include "one part of the body versus the other (one hand versus the other), between a client and another person, or between the self and object such as a building or an accomplishment."

ENACTMENT AND DRAMATIZATION. Enactment increases awareness through the dramatizing of some part of the client's existence by asking him or her to put

his or her feelings or thoughts into action such as instructing the client to "Say it to the person (when in group therapy)," or to role-play using the empty chair technique. "Put words to it" is also often said to the client. Exaggeration is a form of enactment in which clients are instructed to exaggerate a feeling, thought or a movement in order to provide more intensity of feelings. Enactment can be therapeutic and give rise to creativity.

GUIDED FANTASY. Guided fantasy (visualization) is a technique some clients are able to use more effectively than using enactment to bring an experience into the here and now. Clients are asked to close their eyes (if comfortable) and, with the guidance of the therapist, slowly imagine a scene of the past or future event. More and more details are used to describe the event with all senses and thoughts.

DREAM WORK. Dream work is most important in Gestalt therapy. The aim is to "bring dreams back to life and relive them as though they are happening now." Working with the clients' dreams requires developing a list of all the details of the dream, remembering each person, event, and mood in it and then becoming each of these parts through role-playing, and inventing dialogue. Each part of the dream is thought to represent the clients' own contradictory and inconsistent sides. Dialogue between these opposing sides leads clients toward gradual insight into the range of their feelings and important themes in their lives.

AWARENESS OF SELF AND OTHERS. An example of how this technique is used by the Gestalt therapist is having the client to "become" another person such as asking "the client to be his mother and say what his mother would say if the client came in at 2:00 A.M." This provides more insight for the client rather just asking what the client thinks his mother would say if he came home at 2:00 A.M.

AVOIDANCE BEHAVIORS. Awareness of and the reintegration the client's avoidance behaviors are assisted by the interventions used to increase and enhance awareness of feelings, thought, and behaviors.

HOMEWORK. Homework assignments between therapy sessions may include asking clients to write dialogues between parts of themselves or between parts of their bodies, gather information, or do other tasks that are related to and fit with what is going on in the therapy process. Homework may become more difficult as the awareness develops.

Therapy sessions are generally scheduled once a week and individual therapy is often combined with **group therapy**, marital or **family therapy**, movement therapy, **meditation**, or biofeedback training. Sessions can be scheduled from five times a week to every other week and session frequency depends on how long the client can go between sessions without loss of continuity or relapsing. Meetings less frequent than once a week are thought to diminish the intensity of the therapy unless the client attends weekly group with the same therapist. More than twice a week in not usually indicated except with clients who have psychotic disorders, and is contraindicated with those who have a **borderline personality disorder**.

Weekly group therapy may vary from one and one-half to three hours in length, with the average length of two hours. A typical group is composed of ten members and usually balanced between males and females. Any age is thought to be appropriate for Gestalt therapy. There are groups for children as well.

Gestalt therapy is considered by its proponents to have a greater range of styles and modalities than other therapeutic systems and is practiced in individual therapy, groups, workshops, couples, families, and with children, and in agencies such as clinics, family service agencies, hospitals, private practice, growth centers. According to Corey, "The therapeutic style of therapists in each modality vary drastically on many dimensions including degree and type of structure; quantity and quality of techniques used; frequency of sessions, abrasiveness and ease of relating, focus on body, cognitions, feelings; interpersonal contact; knowledge of work within psychodynamic themes; and degree of personal encountering."

Risks

Gestalt therapy is considered to have pioneered the development of many useful and creative innovations in psychotherapy theory and practice. However, there is some concern regarding abusing power by therapist, as well as the high-intensity interaction involved. The concern is in the nature of therapists being enchanted with and using the techniques of Gestalt therapy with other theories of therapy without having the appropriate training in Gestalt therapy theory. Gestalt therapists are very active and directive within the therapy session and therefore, care must be taken that they have characteristics that include sensitivity, timing, inventiveness, empathy, and respect for the client. These characteristics, along with ethical practice, are dependent on the skill, training, experience, and judgment of the therapist. The intensity of the therapy might not be suitable for all patients, and even disruptive for some, despite the competence of the therapist. In addition, there is a lack of monitored, scientific research evidence supporting the effectiveness of Gestalt therapy.

Normal results

Gestalt therapists expect that as result of their involvement in the Gestalt process clients will improve

in the following ways: have increased awareness of themselves; assume ownership of their experience rather than making others responsible for what they are thinking, feeling, or doing; develop skills and acquire values that will allow them to satisfy their needs without violating the rights of others; become aware of all their senses (smelling, tasting, touching, hearing, and seeing); accept responsibility for their actions and the resulting consequences; move toward internal self-support from expectations for external support; to be able to ask for and get help from others and be able to give to others.

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American Psychological Association. 750 First Street,N.E., Washington,D.C. 20002. (202) 336-5800. <http://helping.apa.org>.

The Association for the Advancement of Gestalt Therapy. 7861 Spring Avenue,Elkins Park, PA 19027. (215) 782-1484. Fax: (215) 635-2391. <http://www.aagt.org>.

National Institute of Mental Health. 6001 Executive Boulvard, RM8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

National Mental Health Association. 1021 Prince Street, Alexandria,VA 22314-2971. (703)-684-7722. <http://www.nmha.org>.

Janice VanBuren, Ph.D.

Ginkgo biloba

Definition

Ginkgo biloba is an herbal remedy that has been utilized for thousands of years in China and elsewhere. It is obtained from the leaves and seeds of a plant that is commonly known as the maiden hair tree, believed to be the oldest living species of tree.

KEY TERMS

Alzheimer's disease—An incurable dementia marked by the loss of cognitive ability and memory over a period of 10–15 years. Usually affects elderly people.

Anticoagulant—A medication (such as warfarin, Coumadin, or Heparin) that decreases the blood's clotting ability preventing the formation of new clots. Although anticoagulants will not dissolve existing clots, they can stop them from getting larger. These drugs are commonly called blood thinners.

Thiazide diuretic—Also called water pill, helps the body get rid of excess fluids. Examples include diuril, hydrodiuril, and microzide.

Purpose

Ginkgo preparations have been used to treat such conditions as asthma, inflammation, dizziness, memory problems, and circulatory problems throughout the **brain** and body. As of 2002, research has been concentrating on the possibility that Ginkgo biloba may be a helpful adjunct therapy for memory deficits occurring in **Alzheimer's disease**. Ginkgo is also being explored as a possible treatment for impotence and other circulatory disorders.

Description

Recent research into how Ginkgo biloba affects memory suggests that Ginkgo improves blood flow to the brain by preventing blockages in small blood vessels. These blockages can occur when platelets (blood components that aid in clotting) clump together. Ginkgo seems to decrease platelet stickiness, thus preventing clumping.

The active ingredients of Ginkgo biloba appear to include flavone glycosides and terpene lactones. Flavone glycosides have antioxidant properties. They prevent damage to the cells in the brain by chemicals called free radicals. Terpene lactones improve memory by improving the uptake of the neurotransmitter component choline in the nerve synapses. Terpene lactones also help guard against blood clots within the brain, and may provide some protection against metabolic injury. Improved bloodflow throughout the brain seems to help preserve/improve memory.

Ginkgo biloba is available in a variety of forms, including extracts, capsules, and tinctures.

Leaves of the ginkgo tree. *(Robert J. Huffman. Field Mark Publications. Reproduced by permission.) See color insert for color photo.*

Recommended dosage

As with other herbal supplements, standardization issues sometimes make it difficult to verify the actual dose being administered. In general, efficacious preparations appear to contain at least 24% gingko flavone glycosides and 6% terpene lactones. This is the standardized extract that is commonly used in research about this remedy.

Adults may take between 120 and 240 mg of Ginkgo biloba daily, divided into two or three doses.

Precautions

Because of Ginkgo's effects on platelets, there has been some concern regarding interactions between Ginkgo biloba and anticoagulant medicines, such as warfarin (Coumadin) and aspirin. Studies so far have indicated that Ginkgo does decrease platelet function occasionally. For patients taking Ginkgo, their physician can monitor their platelet function. Rare case reports exist of patients experiencing hemmorhage (including cerebral) while taking Ginkgo.

Side effects

Most reports on Ginkgo biloba suggest that side effects are relatively rare. However, some people may experience stomach upset, including nausea and/or diarrhea. Others who have taken Ginkgo biloba report headache, dizziness, and weakness.

Interactions

Ginkgo biloba may interact with more medications than are listed below. People should notify their health care team of all medications and herbals they take.

To avoid the possibility of increased bleeding, Ginkgo biloba preparations should not be used by patients who are also taking blood thinners (anticoagulants), such as aspirin, warfarin (Coumadin), clopidogrel, dipyridamole, heparin, or ticlopidine.

Ginkgo preparations may interfere with the efficacy of anticonvulsants, such as **carbamazepine** and **valproic acid**.

Caution should be used when taking Ginkgo with thiazide diuretics or with the antidepressant, **trazodone**.

Resources

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Rosalyn Carson-DeWitt, M.D.

Ginseng

Definition

Ginseng is an herbal preparation derived from the aromatic root of a plant of the genus *Panax*, which is native to East Asia. Ginseng belongs to the Araliaceae family of plants. Siberian ginseng belongs to a different genus, *Eleutherococcus senticosus.* The English name of the plant is a modification of its Chinese name, *ren shen,* which means "man" and "herb." The Chinese name comes from the ginseng root's resemblance to the shape of the human body, whence the plant's traditional use as a tonic for male sexual vigor and potency. The Latin name for the species, *Panax*, is derived from the Greek word *panacea*, which means "cure-all," or, "all-healer."

There are three species of ginseng in common use in the United States: American ginseng, Korean ginseng, and Siberian ginseng. All are regarded as adaptogens that normalize immune functions and are preparations that help the body adapt to change, thus lowering the risk of stress-related illness. American ginseng, whose botanical name is *Panax quinquefolius*, has recently been evaluated as a treatment for high blood sugar in patients with type 2 (adult-onset) diabetes. It is considered to be less stimulating than the Korean or Siberian varieties. Korean

ginseng, or *Panax ginseng*, is the species most often studied in Western as well as Asian trials of botanical preparations. Siberian ginseng, or *Eleutherococcus senticosus*, has been used in Russian sports medicine to boost athletic performance and strengthen the immune system.

As of 2002, ginseng is one of the most expensive herbs in the world, costing as much as $20 per ounce, or more for red ginseng with the root, which is over 10,000 years old. It is one of the top three herbal products sold in the United States.

KEY TERMS

Adaptogen—A remedy that helps the body adapt to change, and thus lowers the risk of stress-related illnesses.

Aphrodisiac—A medication or preparation given to stimulate sexual desire.

Douche—A jet or current of water, often with a medication or cleansing agent dissolved in it, applied to a body cavity for medicinal or hygienic purposes.

Ginseng abuse syndrome—A group of symptoms recognized by Chinese physicians as the result of excessive use of ginseng. The symptoms include dizziness, high blood pressure, restlessness, nausea, possible bleeding from the digestive tract, and skin rashes.

Panacea—A medicine or other substance regarded as a cure for all ills. Ginseng should not be considered a panacea.

Qi—The Chinese term for energy, life force, or vital force.

Purpose

In traditional Chinese medicine (TCM), ginseng is regarded as having a "sweet" and "neutral" nature. It is thought to have a particular affinity for the spleen and lungs. It is used as an aphrodisiac, a tonic for the spleen, kidney and adrenal functions, and lungs, and a general restorative for the qi or vital energy in the body. TCM also recommends ginseng for asthma, weak pulse, indigestion, lack of appetite, rectal prolapse, hypertension, diabetes, **insomnia**, angina, congestive heart failure, and heart palpitations. It is important to note that ginseng is an exception to the rule that Chinese herbal medicine rarely uses a single herb in the manner of Western herbalism. Ginseng is often listed as one ingredient among several in Chinese medicines; it is, however, one of the few herbs in TCM that is sometimes prescribed by itself.

In the West, ginseng is frequently advertised as an energy booster, a memory aid, a sexual stimulant, a treatment for impotence and gastrointestinal disorders, and a promoter of longevity. Many Western researchers consider these claims inflated; some studies have found no difference between ginseng and a placebo in terms of the energy levels or general well-being reported by test subjects. Most studies nevertheless have shown improved energy, memory function and performance especially when fatigued, though most of the studies have been short-term. Ginseng's association with the male reproductive system is sufficiently strong that Western feminist herbalists frequently advise women against taking ginseng for any reason.

Description

The part of the ginseng plant that is used medicinally is the root. Ginseng roots are not harvested until the plant is four to six years old. The active ingredients in ginseng root are saponin triterpenoid glycosides, or chemicals commonly called ginsenosides. Other compounds found in Asian ginseng include glycans (panaxans); polysaccharide fraction DPG-3-2; peptides; maltol; and volatile oil. The active compounds in Siberian ginseng are called eleutherosides. Eleutherosides are somewhat different from the ginsenosides found in the *Panax* varieties of ginseng. There has been some debate among herbalists whether Siberian ginseng should be considered a true ginseng at all, due to this difference in active ingredients. Ginseng root from any of the three varieties is dried and can then be made into powder, capsules, or a liquid tincture. American ginseng is also available in the United States as whole roots.

Recommended dosages

Dosages of Korean ginseng used in traditional Chinese medicine are given as 2–8 g as a tonic and 15–20 g for acute conditions.

Researchers who studied the potenial effectiveness of ginseng as a treatment for diabetes found that 1–3 g of American ginseng taken 40 minutes before a meal was effective in reducing blood sugar levels. Because dried ginseng root is hard and brittle, it must be simmered for about 45 minutes to extract the ginsenosides. Two to three teaspoonsful of dried root are used per cup. Powder made from American ginseng can be made into tea or taken with water or juice. One-half to one teaspoon is recommended per serving. American ginseng is usually taken two to three times per day between meals.

For Siberian ginseng, the recommended dosage for the powdered form is 1–2 g daily, taken in capsules or mixed with water or juice. The dose should be divided

and taken two or three times per day between meals. The recommended dosage for liquid extract of Siberian ginseng is 1–2 mL twice daily.

Precautions

Because ginseng is considered a dietary supplement rather than a drug, it is not regulated by the Food and Drug Administration (FDA). Studies done between 1999 and 2001 found that many ginseng products for sale in the United States contain little or no ginseng. There have been no recent reports of contaminated products.

It is important for patients with Type 2 diabetes who are taking oral prescription medications to lower blood sugar levels to tell their physician if they are using any products containing ginseng. One Chinese-American physician reported several incidents of patients developing hypoglycemia (low blood sugar) from taking ginseng preparations alongside their regular prescription drugs.

People who use ginseng should discontinue it prior to abdominal or dermatologic surgery, or dental extraction. It has been associated with bleeding problems following surgery.

The American Herbal Products Association (AHPA) states that ginseng should not be taken by people with hypertension (high blood pressure). Data suggests variable effects on blood pressure. Some patients experience hypertension and some experience hypotension.

Ginseng should not be given to children. In addition, pregnant or lactating women should not use ginseng, as it may lower estrogen production.

Ginseng should not be used uninterruptedly for long periods of time. In Asian medicine, it is customary to take ginseng for two months and then stop for a full month before taking it again, but the basis for this is uncertain.

Side effects

Ginseng can have serious side effects. The American Herbal Products Association, or AHPA, classifies ginseng as a Class 2d herb, which means that its use is subject to restrictions.

Contemporary Chinese practitioners recognize a condition known as ginseng abuse syndrome, caused by taking ginseng incorrectly or excessively. In China, ginseng is almost always used for longevity by people over the age of 60; it is not given to younger people unless they are severely debilitated. Chinese medicine also recommends ginseng for use in winter only; it is not taken year round. The symptoms of ginseng abuse syndrome include include heart palpitations, heaviness in the chest, high blood pressure, dizziness, insomnia, agitation, restlessness, nausea, vomiting, abdominal pain and/or bloating, diarrhea, possible upper digestive tract bleeding, edema, and a red skin rash that is most noticeable on the face. Western herbalists recommend that anyone taking ginseng who develops these symptoms should stop taking the herb at once and contact a licensed practitioner of TCM to determine whether ginseng abuse is the cause of the problem.

A number of case studies involving severe side effects from habitual use of ginseng have been reported in American medical journals. These studies include a case of Stevens-Johnson syndrome (a disorder of the skin and mucosa usually caused by reactions to corticosteroids and a few other systemic drugs) in a Chinese student; a case of cerebral arthritis in a 28-year-old woman following a large dose of ginseng extract; a case of metrorrhagia (uterine hemorrhage) following two months of steady use of ginseng; and a case of hemorrhagic bleeding from the vagina following habitual use of ginseng douches.

Interactions

Ginseng has been reported to interact with caffeine to cause overstimulation and insomnia in some people. It has also been reported to increase the effects of digoxin, a medication used to treat congestive heart failure; and to interact with **phenelzine**, an antidepressant. Its interactions with phenelzine cause symptoms ranging from manic episodes to headaches. It also may alter the effects of the drug coumadin, and any anticoagulant therapies.

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American Association of Oriental Medicine. 433 Front Street, Catasaqua, PA 18032. (610) 266-1433. Fax: (610) 264-2768. <www.aaom.org>.

American Botanical Council. PO Box 144345. Austin, TX 78714-4345.<www.herbalgram.org>.

Herb Research Foundation. 1007 Pearl Street. Suite 200. Boulder, CO 80302. <www.herbs.org>.

National Center for Complementary and Alternative Medicine (NCCAM) Clearinghouse. P.O. Box 7923, Gaithersburg, MD 20898. (888) 644-6226. TTY: (866) 464-3615. Fax: (866) 464-3616. <www.nccam.nih.gov>.

Rebecca J. Frey, Ph.D

Grief

Definition

Grief, which is also known as bereavement, is a term used to describe the intense and painful emotions experienced when someone or something a person cares about either dies or is lost. The emotional pain from losing a loved one, whether it is a spouse, child, parent, sibling, friend, or pet, can be the most severe suffering a person must endure. At its most intense, grief can dominate every facet of a person's life, making the carrying out of ordinary responsibilities impossible. Loss and subsequent grief, however, are an inevitable part of life and loving other people or companion animals. Painful as it is, grief is a normal response to loss and generally resolves with the passage of time.

Description

Grief is usually characterized by numbness, tearfulness, physical feelings of emptiness in the pit of the stomach, weak knees, shortness of breath, a tendency to sigh deeply, a sense of unreality, and overall emotional distress. Anxiety and longing may alternate with depression and despair. **Insomnia** and loss of appetite are common. Initially, people often feel numb and unable to accept their loss. Numbness is followed by shock as reality begins to penetrate.

There is generally a disorganization of normal behavior patterns that may make it impossible for a bereaved person to return to work immediately or take social initiatives. Such acute symptoms usually begin to subside after several months, with emotional balance being regained within a year. Studies using instruments developed to measure symptoms of grief and bereavement demonstrate wide individual variations in specific symptoms and their intensity. Long after the immediate period of mourning, bereaved persons may continue to feel upset, empty, or tearful. In addition, further losses, additional stressors, or dates of such important anniversaries as a wedding, birthday, or the date of death can reactivate the acute symptoms of grief.

Dimensions of grief

Grief and mourning are important life experiences in that they permit a bereaved person to accept the reality of loss and begin to find ways of filling the resultant emptiness. Loss is a significant part of the aging process and can contribute to emotional problems in older people. The impact of loss and resulting grief and mourning is not limited to the death of a loved one. It is also present to a lesser extent in the loss of physical acuity and agility and the loss of social of status as a result of retirement and/or growing older.

Unfortunately, people in the United States do not generally receive cultural support for the losses they experience and the need to mourn those losses. Unlike other cultures with specific rituals for grief and mourn-

Columbine High School students in Littleton, Colorado grieving for their lost classmates. *(Photo by David Zalubowski. AP/Wide World Photos. Reproduced by permission.)*

ing, there is often subtle but insistent pressure on Americans— particularly males— to stop crying and move forward with resumption of regular activities. Onlookers may try to divert the mourner's attention to other topics or discourage crying or talking about the loved one. These responses suggest that grief isn't healthy or that it should be minimized or avoided. If the grief is associated with the loss of a pet, the person may be shamed for grieving because "it was just an animal." Women who have had a pregnancy ended by miscarriage also encounter responses that minimize or trivialize the loss of their expected child. Social insensitivity may drive the mourner to grieve in secret or feel guilty because of continued intense feelings of loss.

Stages of grief

Elizabeth Kubler-Ross, the noted researcher on death and dying, identified five stages of acceptance in the process of dying. While her work initially referred to the person who is dying, the five stages are also applied to people who are grieving a loss. The stages are sometimes collapsed into three, but the general grieving process includes these components:

- Shock/denial. This stage comprises the initial period after receiving news of the loss. The affected person may say, "There must be a mistake," "This can't be true," or similar expressions of disbelief. People often describe feeling numb or cold in this stage.
- Bargaining. This stage represents an attempt to persuade God or a higher power to change the reality of loss in exchange for improved behavior or some sacrifice on the part of the bereaved person. The mourner may offer, for example, to take better care of their relationship with the loved one if God will only bring them back.
- Anger. This emotion may be directed toward the medical establishment, family members, God, or even the person who has died.
- Depression. In this stage, the person's body begins to absorb the reality of the loss. The bereaved person may be unable to eat, sleep, or talk normally with people. They may have episodes of spontaneous crying and such physical symptoms as nausea, headaches, chills or trembling.
- Acceptance. This is the phase in which the mourner comes to terms with the loss and begins to look ahead once more. Energy returns and the bereaved person is able to reconnect with others, engage in enjoyable activities, and make plans for the future.

There is, however, no "normal" pattern for grief; it is a highly variable experience. People pass through the stages outlined by Kubler-Ross at their own rate, depending on the significance of the loss, number of previous losses, individual resiliency, presence of a support system, and permission to grieve from those around them. Grieving is not a linear process. There is movement back and forth between the stages until acceptance is reached. Occasionally, a person may remain "stuck" in one stage, particularly anger or depression, and may benefit from professional help in order to move on. Remaining in one of the stages indefinitely can create emotional and occupational difficulties.

Bereavement and marriage

Studies show that some widowed people have **hallucinations** or **delusions** of contact with the lost spouse that may last for years. These hallucinations are more likely to occur in people who were happily married. The most common hallucination reported is a sense of the dead spouse's presence. Others report seeing, hearing, or being touched by or spoken to by the spouse.

The interplay of grief and marital quality has led to research findings that contradict earlier widespread beliefs. A study by Deborah Carr and her colleagues in 2000 found that anxiety was greater in those who had been highly dependent on their spouses than in those who were less dependent. People who had been in conflicted relationships reported lower levels of yearning for the spouse than those who had enjoyed high levels of marital closeness. Women who had relied on their husbands to do the driving and perform other similar tasks had much higher levels of yearning than men who depended on their wives. This finding contradicts the common belief that grief is more severe if the marriage was conflicted,

suggesting a more complex relationship between bereavement and characteristics of the marriage.

Another suggestion of the complex relationship between bereavement and marriage is reflected in studies of sudden and anticipated loss among older widowed people. The sudden death of a spouse was associated with slightly higher levels of yearning among women, but significantly lower yearning among men. Forewarning of the death (extended illness, advancing age) did not affect depression, anger, shock or overall grief six or 18 months after the loss. Prolonged forewarning was associated with increased anxiety at six- and 18-month follow-up interviews after the death.

Grief and mourning may also occur when the loss of a partner occurs through divorce or the end of a dating relationship. Some researchers think that moving to the stage of acceptance is more difficult in such cases because the partner can still be contacted, especially if there are children involved. Seeing a former partner involved in a new relationship can cause the partner mourning the loss to re-experience acute symptoms of grief. Some research evidence suggests that grief related to the breakup of an intimate relationship is more intense for the individual who was left behind than for the person who ended the relationship.

Grieving may be particularly prolonged and intense when certain unexpected losses occur that are outside the ordinary progression of life events. The loss of a parent before a child reaches adulthood or a parent's loss of a child inflict deep emotional wounds for an extended period of time. Similarly, the loss of a loved one to murder, terrorism, or other acts of intentional violence is harder to bear than death resulting from natural causes or accidents. Death from **suicide** complicates grief by adding shame to the other painful emotions associated with bereavement. The opportunity to fully grieve such significant losses, however, enables survivors to move forward despite the magnitude of their loss.

See also Adjustment disorder; Suicide

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The Compassionate Friends, Inc. P.O. Box 3696, Oak Brook, IL 60522. <http://wwwcompassionatefriends.org>.

GROWW [Grief Recovery Online (founded by) Widows & Widowers]. 931 N. State Road 434, Suite 1201-358, Altamonte Springs, FL 32714. <http://www.groww.org>.

Judy Leaver, M.A.

Grief counseling

Definition

Grief counseling refers to a specific form of therapy, or a focus in general counseling with the goal of helping the individual grieve and address personal loss in a healthy manner. Grief counseling is offered individually by psychologists, clergy, counselors or **social workers**, in groups led by professionals, as well as informal **support groups** offered by churches, community groups, or organizations devoted to helping individuals grieve specific losses.

Specific tasks of grief counseling include emotional expression about the loss (which can include a wide range of feelings), accepting the loss, adjusting to life after the loss, and coping with the changes within oneself and the world after the loss. Typical feelings experienced by individuals, and addressed in grief counseling, include sadness, anxiety, anger, loneliness, guilt, relief, isolation, confusion, or numbness. Behavioral changes may also be noticed, such as being disorganized, feeling tired, having

KEY TERMS

Therapeutic letter—A letter written to the deceased in order to help the survivors express feelings and thoughts they may not have been able to before the loss.

trouble concentrating, sleep problems, appetite changes, vivid dreams, or daydreaming about the deceased.

Purpose

The purpose of grief counseling is to help individuals work through the feelings, thoughts, and memories associated with the loss of a loved one. Although grieving can occur for other types of loss as well (such as loss of goals, ideals, and relationships), grief counseling is generally directed toward positive adjustment following loss after the death of a loved one.

Grief counseling helps the individual recognize normal aspects of the grieving or mourning process, cope with the pain associated with the loss, feel supported through the anxiety surrounding life changes that may follow the loss, and develop strategies for seeking support and self-care.

Precautions

Grieving is a normal life process—an adjustment reaction to a loss. Grief counseling is meant to facilitate that normal process. No specific precautions are warranted. However, there are certain circumstances in which complications to the normal grieving process may occur. These circumstances may involve the loss of a child, or the loss of a loved one due to an accident or homicide, for example.

In these cases of complicated grieving, more extreme responses to the loss may be observed, depending on the individual's capacity for coping, personal resiliency, and support system. For example, if the individual feels isolated, he may be at greater risk for severe depressive symptoms or a **suicide** attempt. Alternatively, if the survivors feel rage or anger over the loss, there may be a risk of harm to others.

Description

Grief counseling helps the individual work through the feelings associated with the loss of another, accept that loss, determine how life can go on without that person, and consolidate memories in order to be able to move forward. Grief counseling also provides information about the normal grieving process, to help individuals understand that many of the symptoms and changes they are experiencing are a normal, temporary reaction to loss. For some individuals, the primary focus of grief counseling is to help identify ways to express feelings about the loss that the person has been unable to express on his or her own. Individuals who seek grief counseling may be experiencing an emotional numbness, or a residual shock in reaction to the loss, and need assistance to return to a normal life. In those cases, grief counseling will focus on helping the individual get in touch with those feelings and become more active in the daily routine. This often requires accepting the loss as a reality.

For some people, grieving may initially be so extreme that physical and psychological symptoms may be experienced, while other people appear to experience no symptoms whatsoever, similar to the numbness described above. Activities of daily living may feel overwhelming to an individual who has experienced a loss. In these cases, grief counseling may focus on specific coping skills to help the individual resume some normalcy in his or her daily routine. For example, if sleep patterns are disrupted, grief counseling may include consultation with the individual's physician to assist with temporary strategies to increase sleep. If the individual is having trouble getting to work on time, behavioral strategies may be used as an interim measure to help the person return to aspects of normal daily life.

Additional work in grief counseling may involve identifying ways to let go or say good-bye if the individual has not been able to do so successfully. Therapeutic letters may be a helpful mechanism to express thoughts that were not conveyed prior to the death. Dreams are frequently experienced by survivors, and these can be a focus in grief counseling as well. The dreams can often be a way of consolidating the memories about the deceased.

Preparation

No specific preparation is required by the participant; however, a need for grief counseling is indicated by prolonged symptoms (such as crying spells, preoccupation with the deceased, lack of motivation, or suicidal thoughts), and the severity of personal distress over the loss. A patient seeking grief counseling would most likely undergo a clinical evaluation by a therapist, before the grief counseling began, so that the therapist could understand the patient's personal history and goals for treatment.

Aftercare

Aftercare is usually provided through informal support systems, which may include family and friends, as well as support groups.

Risks

A slight risk exists regarding treatment of complicated grief. Such circumstances include chronic, prolonged grieving or unexpected loss (particularly due to a violent accident, suicide, homicide, or the death of a child). These factors complicate the grieving process due to the unexpected, sometimes violent nature of the loss, that feels inconsistent with expectations and desires for loved ones. In these cases, an initial adverse effect may be seen from participation in treatment, due to the increased focus on the loss. This reaction improves over time, as adjustment is facilitated. Two other factors impacting individual adjustment include the type of relationship the individual had with the deceased, and the resiliency of the individual.

Normal results

Normal results from grief counseling include being able to move on with one's life, recognizing and accepting the physical loss of the individual, and being able to bridge that loss with positive memories of the deceased. Successful coping will be characterized by a return to normal routines, although some symptoms may be experienced periodically throughout the year or so following the loss.

Abnormal results

Abnormal results would include an unsuccessful outcome of prolonged grief, exhibited by continued preoccupation with the loss of the individual, crying spells, and depressive symptoms being the most likely complications. Some disruption of the daily routine would persist, and there may be extreme emotional responses, that could include no apparent reaction to difficulty containing feelings. Other complications include "unfinished business," or feelings of unresolved issues with the deceased. Sometimes the feelings of unresolved issues can be as simple as wishing they had communicated their love and affection for the person the last time they saw them, or may be as complicated as unresolved feelings about a history of **abuse** by the deceased.

See also Creative therapies; Support groups

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The Compassionate Friends. P. O. Box 3696, Oak Brook, IL 60522. (630) 990-0010. <http://www.compassionate-friends.org>.

SHARE Pregnancy and Infant Loss Support. St. Joseph Health Center, 300 First Capital Drive, St. Charles, MO 63301. (800) 821-6819.

SIDS Alliance. 1314 Bedford Ave., Suite 230, Baltimore, MD 21208. (800-221-SIDS). <http://www.sidsalliance.org>.

Widowed Persons Service, AARP. 601 E. Street, NW, Washington, DC 20049. (202) 434-2260.

OTHER

<http://www.griefnet.org>.

Deanna Pledge, Ph.D.

Group homes

Definition

Group homes are small, residential facilities located within a community and designed to serve children or adults with chronic disabilities. These homes usually have six or fewer occupants and are staffed 24 hours a day by trained caregivers.

Description

Most group homes are standard, single-family houses, purchased by group home administrators and adapted to meet the needs of the residents. Except for any adaptive features such as wheelchair ramps, group homes are virtually indistinguishable from other homes in the surrounding neighborhood. Group homes may be located in neighborhoods of any socioeconomic status.

Residents of group homes usually have some type of chronic mental disorder that impairs their ability to live

KEY TERMS

Ambulation—Ability to walk.

Case manager—A professional who designs and monitors implementation of comprehensive care plans (i.e., services addressing medical, financial, housing, psychiatric, vocational, social needs) for individuals seeking mental health or social services.

Community mental health centers—Organizations that manage and deliver a comprehensive range of mental health services, education, and outreach to residents of a given community.

Community Mental Health Centers Act of 1963—Federal legislation providing grants for the operation of community mental health centers and related services.

Deinstitutionalization—The process of moving people out of mental hospitals into treatment programs or halfway houses in local communities. With this movement, the responsibility for care shifted from large (often governmental) agencies to families and community organizations.

Fair Housing Act of 1968—Federal legislation regarding access to housing that prohibits discrimination based on race, color, national origin, sex, religion, disability, or familial status.

Least restrictive environment—Refers to care options that involve the least amount of restraint and the greatest degree of independence possible, while still meeting the individual's needs and maintaining safety.

Medicaid Home and Community-Based Waiver—Legislation regarding the use of Medicaid funds for care services; allows certain federal requirements to be bypassed so that states can use the funds more flexibly for accessing home- and community-based services rather than using hospitals or intermediate-care facilities.

NIMBY phenomenon—Acronym for Not In My Backyard, describing the common opposition displayed by citizens toward the placement of group homes or other social service facilities in their neighborhoods.

Non-ambulatory—Unable to walk.

independently. Many residents also have physical disabilities such as impairments of vision communication, or ambulation. These individuals require continual assistance to complete daily living and self-care tasks. Some also require supervision due to behavior that may be dangerous to self or others, such as aggression or a tendency to run away.

Although most group homes provide long-term care, some residents eventually acquire the necessary skills to move to more independent living situations. Group homes for children are usually temporary placements, providing care until a foster family can be secured. Others may return to their natural families. Occasionally, halfway homes for people recently released from prison or discharged from a substance abuse program may also be referred to as group homes. These types of group homes are also transitory in nature.

History and mission

The development of group homes occurred in response to the **deinstitutionalization** movement of the 1960s and 1970s. As psychiatric hospitals closed, discharged individuals needed places to live. Group homes were designed to provide care in the least restrictive environment and to integrate individuals with disabilities into the community, reducing **stigma** and improving quality of life. The environment of a group home was intended to simulate typical family life as much as possible.

Since the passage of the **Community Mental Health** Centers Act in 1963, grants have been available to group homes. State and federal funds such as the Medicaid Home and Community-Based Waiver continue to support the majority of group homes. However, some homes operate on donations from private citizens or civic and religious organizations. Most group homes are owned by private rather than governmental organizations, and can be either non-profit or for-profit organizations. Group homes are considered more cost effective compared to institutional care. Unfortunately, the number of available group homes has not always matched need, resulting in **homelessness** or re-hospitalization for some individuals.

One of the goals of group home living is to increase the independence of residents. Group home staff members teach residents daily living and self-care skills, providing as little assistance as possible. Daily living skills include meal preparation, laundry, housecleaning, home maintenance, money management, and appropriate social interactions. Self-care skills include bathing or showering, dressing, toileting, eating, and taking prescribed medications.

Staff also assure that residents receive necessary services from community service providers, including medical care, physical therapy, occupational therapy, vocational training, education, and mental health services. Most group home residents are assigned a case manager from a community mental health center or other government agency who oversees their care. Case managers review group home documentation regarding skills learned and services received, and make recommendations for adjustments in care.

The NIMBY phenomenon

Unfortunately, group homes have received much opposition from communities. NIMBY (acronym for Not In My Backyard) describes the common reaction of community residents when they discover that a group home is targeted for their neighborhood. Current research suggests that protests frequently involve concerns over personal security, declining property values, or a generalized threat to the neighborhood's quality. Some researchers believe that prejudiced attitudes such as ignorance, fear, and distrust are the true reasons for protest.

Usually, neighborhood opposition is unsuccessful due to provisions of the Fair Housing Act of 1968. However, such opposition can be detrimental to the goal of integrating residents into the community. The NIMBY phenomenon is also a concern because as deinstitutionalization continues, the need for additional group homes increases. Statistics show that between 1987 and 1999, the use of group homes serving individuals with developmental disabilities and containing six residents or less increased by 240%.

Social service workers are constantly looking for ways to address the NIMBY phenomenon. Some research has suggested that community concerns decrease with time as community members become familiar with group home residents. A recent study proposed that opposition can be decreased by providing advanced notice of plans for a group home, as well as adequate information and discussion about expectations.

Factors affecting group home success

Initially, many people were skeptical about the adequacy of group home care compared to psychiatric hospitals or other institutions. Over the past 25 years, many studies have examined the impact of group home care on residents. These studies have consistently shown increases in adaptive behavior, productivity, community integration, and level of independence.

Risks involved in successfully transitioning an individual to a group home include psychological deterioration such as severe cognitive or physical impairments, physical deterioration that includes being non-ambulatory, or mortality issues such as being age 70 or older.

Group homes are facilities in residential communities that house people with physical or mental disabilities or other challenges. The group home residents pictured above live in a teen group home in Massachusetts for low-income mothers and their children. *(AP Photo/Lisa Poole. Photo reproduced by permission.)*

Before considering group home placement—especially for those in the high-risk category—extensive planning should be conducted. A complete assessment plan of the individual's needs should specify which agency will be responsible for meeting medical needs, particularly in the event of a crisis. The individual's strengths should be incorporated into the plan whenever possible. For example, if a supportive family is an identified strength, the preferred group home should be close in proximity to facilitate family visits.

Other factors that contribute to group home success are a small staff-to-resident ratio, well-trained staff, and a home-like atmosphere. As with any type of organization, some group homes are better run than others. A careful investigation into a home's procedures is recommended. Research suggests that individuals with severe cognitive impairments often experience a period of disorientation, and may need additional support or supervision for the first few months while adjusting to their new surroundings. Pre-placement visits and discussion can reduce anxiety for the future resident.

See also Case management

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ORGANIZATIONS

The ARC National Headquarters. 1010 Wayne Avenue, Suite 650, Silver Spring, Maryland 20910. (301) 565-3842. <http://www.thearc.org>.

Child Welfare League of America-Headquarters. 440 First Street, NW, Third Floor, Washington, DC 20001-2085. (202) 638-2952. < http://cwla.org>.

National Institute of Mental Health. 600 Executive Boulevard, Room 8184, MSC 9663, Bethesda, Maryland 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

Office of Fair Housing and Equal Opportunity. Room 5116, Department of Housing and Urban Development, 451 Seventh Street, SW, Washington, DC 20410-2000. (202) 708-2878. <http://www.hsh.com>.

Sandra L. Friedrich, M.A.

Group therapy

Definition

Group therapy is a form of **psychotherapy** in which a small, carefully selected group of individuals meets regularly with a therapist.

Purpose

The purpose of group therapy is to assist each individual in emotional growth and personal problem solving.

Description

Group therapy encompasses many different kinds of groups with varying theoretical orientations that exist for varying purposes. All therapy groups exist to help individuals grow emotionally and solve personal problems. All utilize the power of the group, as well as the therapist who leads it, in this process.

Unlike the simple two-person relationship between patient and therapist in individual therapy, group therapy offers multiple relationships to assist the individual in growth and problem solving. The noted **psychiatrist** Dr. Irvin D. Yalom in his book *The Theory and Practice of Group Therapy* identified 11 "curative factors" that are the "primary agents of change" in group therapy.

Instillation of hope

All patients come into therapy hoping to decrease their suffering and improve their lives. Because each member in a therapy group is inevitably at a different point on the coping continuum and grows at a different rate, watching others cope with and overcome similar problems successfully instills hope and inspiration. New members or those in despair may be particularly encouraged by others' positive outcomes.

Universality

A common feeling among group therapy members, especially when a group is just starting, is that of being isolated, unique, and apart from others. Many who enter group therapy have great difficulty sustaining interpersonal relationships, and feel unlikable and unlovable. Group therapy provides a powerful antidote to these feelings. For many, it may be the first time they feel understood and similar to others. Enormous relief often accompanies the recognition that they are not alone; this is a special benefit of group therapy.

KEY TERMS

Altruism—An unselfish willingness to help others.

Behavior therapies—Numerous techniques all having their roots in principles of learning.

Catharsis—A powerful emotional release followed by a feeling of great relief.

Cognitive-behavior therapy—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

Existential factors—Realities of life including death, isolation, freedom, and meaninglessness that must be faced by all individuals.

Gestalt therapy—A therapeutic approach that focuses on increasing awareness of feelings and impulses in the present.

Group cohesiveness—The degree to which a group functions well in its assigned task; the importance the group develops to each of its members.

Group psychotherapy—A form of therapy in which a small, carefully selected group of individuals meets regularly with a therapist to assist each individual in emotional growth and personal problem solving.

Imitative behavior—Behaviors of a therapist or group member that are imitated, consciously or unconsciously, by other group members.

Individual psychotherapy—A relationship between therapist and patient designed to foster the patient's emotional growth and personal problem-solving skills.

Information giving—Imparting of information about a disease or condition as part of the therapeutic process.

Interpersonal learning—Learning that takes place via feedback from others.

Person-centered therapy—A therapeutic approach that believes the client's own drive towards growth and development is the most important factor in healing.

Phenomenological therapy—A therapeutic approach that focuses on the interpretations individuals place on their experiences.

Psychodrama—A form of group therapy in which group members act out parts of important people in the lives of individual group members.

Psychodynamic groups—Psychotherapy groups that utilize the principles of unconscious needs and motivations developed by Sigmund Freud.

Self-help groups—Groups that fall outside the realm of psychotherapy groups, but that offer help to individuals around a particular problem or concern. These groups typically are not professionally led.

Termination—The process of ending a therapy group; an important part of a therapy group.

Universality—The feeling of being isolated, unique, and separate from others, often experienced by therapy group members.

Information giving

An essential component of many therapy groups is increasing members' knowledge and understanding of a common problem. Explicit instruction about the nature of their shared illness, such as **bipolar disorders**, depression, panic disorders, or bulimia, is often a key part of the therapy. Most patients leave the group far more knowledgeable about their specific condition than when they entered. This makes them increasingly able to help others with the same or similar problems.

Altruism

Group therapy offers its members a unique opportunity: the chance to help others. Often patients with psychiatric problems believe they have very little to offer others because they have needed so much help themselves; this can make them feel inadequate. The process of helping others is a powerful therapeutic tool that greatly enhances members' self-esteem and feeling of self-worth.

Corrective recapitulation of the primary family

Many people who enter group therapy had troubled family lives during their formative years. The group becomes a substitute family that resembles—and improves upon—the family of origin in significant ways. Like a family, a therapy group consists of a leader (or coleaders), an authority figure that evokes feelings similar to those felt toward parents. Other group members substitute for siblings, vying for attention and affection from the leader/parent, and forming subgroups and coali-

tions with other members. This recasting of the family of origin gives members a chance to correct dysfunctional interpersonal relationships in a way that can have a powerful therapeutic impact.

Improved social skills

According to Yalom, social learning, or the development of basic social skills, is a therapeutic factor that occurs in all therapy groups. Some groups place considerable emphasis on improving social skills, for example, with adolescents preparing to leave a psychiatric hospital, or among bereaved or divorced members seeking to date again. Group members offer feedback to one another about the appropriateness of the others' behavior. While this may be painful, the directness and honesty with which it is offered can provide much-needed behavioral correction and thus improve relationships both within and outside the group.

Imitative behavior

Research shows that therapists exert a powerful influence on the communication patterns of group members by **modeling** certain behaviors. For example, therapists model active listening, giving nonjudgmental feedback, and offering support. Over time, members pick up these behaviors and incorporate them. This earns them increasingly positive feedback from others, enhancing their self-esteem and emotional growth.

Interpersonal learning

Human beings are social animals, born ready to connect. Our lives are characterized by intense and persistent relationships, and much of our self-esteem is developed via feedback and reflection from important others. Yet we all develop distortions in the way we see others, and these distortions can damage even our most important relationships. Therapy groups provide an opportunity for members to improve their ability to relate to others and live far more satisfying lives because of it.

Group cohesiveness

Belonging, acceptance, and approval are among the most important and universal of human needs. Fitting in with our peers as children and adolescents, pledging a sorority or fraternity as young adults, and joining a church or other social group as adults all fulfill these basic human needs. Many people with emotional problems, however, have not experienced success as group members. For them, group therapy may make them feel truly accepted and valued for the first time. This can be a powerful healing factor as individuals replace their feelings of isolation and separateness with a sense of belonging.

Catharsis

Catharsis is a powerful emotional experience—the release of conscious or unconscious feelings—followed by a feeling of great relief. Catharsis is a factor in most therapies, including group therapy. It is a type of emotional learning, as opposed to intellectual understanding, that can lead to immediate and long-lasting change. While catharsis cannot be forced, a group environment provides ample opportunity for members to have these powerful experiences.

Existential factors

Existential factors are certain realities of life including death, isolation, freedom, and meaninglessness. Becoming aware of these realities can lead to anxiety. The trust and openness that develops among members of a therapy group, however, permits exploration of these fundamental issues, and can help members develop an acceptance of difficult realities.

History of group therapy

Group therapy in the United States can be traced back to the late nineteenth and early twentieth centuries, when millions of immigrants moved to American shores. Most of these immigrants settled in large cities, and organizations such as Hull House in Chicago were founded to assist them adjust to life in the United States. Known as settlement houses, these agencies helped immigrant groups lobby for better housing, working conditions, and recreational facilities. These early social work groups valued group participation, the democratic process, and personal growth.

In 1905, a Boston physician named Joseph Pratt formed groups of impoverished patients suffering from a common illness—tuberculosis. Pratt believed that these patients could provide mutual support and assistance. Like settlement houses, his early groups were another forerunner of group therapy.

Some early psychoanalysts, especially Alfred Adler, a student of Sigmund Freud, believed that many individual problems were social in origin. In the 1930s Adler encouraged his patients to meet in groups to provide mutual support. At around the same time, social work groups began forming in mental hospitals, child guidance clinics, prisons, and public assistance agencies. A contemporary descendant of these groups is today's support group, in which people with a common problem come together, without a leader or therapist, to help each other solve a common problem. Groups such as Alcoholics Anonymous, Narcotics Anonymous, and

Group therapy offers multiple relationships to assist an individual in growth and problem solving. In group therapy sessions, members are encouraged to discuss the issues that brought them into therapy openly and honestly. The therapist works to create an atmosphere of trust and acceptance that encourages members to support one another. *(Richard T. Nowitz. Photo Researchers, Inc. Reproduced by permission.)*

Survivors of Incest all have their roots in this early social work movement.

Types of therapy groups

PSYCHODYNAMIC THERAPIES. Psychodynamic theory was conceived by Sigmund Freud, the father of **psychoanalysis**. Freud believed that unconscious psychological forces determine thoughts, feelings, and behaviors. By analyzing the interactions among group members, psychodynamic therapies focus on helping individuals become aware of their unconscious needs and motivations as well as the concerns common to all group members. Issues of authority (the relationship to the therapist) and affection (the relationships among group members) provide rich sources of material that the therapist can use to help group members understand their relationships and themselves.

PHENOMENOLOGICAL THERAPIES. Until the 1940s virtually all psychotherapy was based on psychoanalytic principles. Several group therapy approaches were developed by psychoanalytically trained therapists looking to expand their focus beyond the unconscious to the interpretations individuals place on their experiences. Underlying this focus is the belief that human beings are capable of consciously controlling their behavior and taking responsibility for their decisions. Some phenomenological therapies include:

- *Psychodrama*—developed by Jacob Moreno, an Austrian psychiatrist, this technique encourages members to play the parts of significant individuals in their lives to help them solve interpersonal conflicts. Psychodrama brings the conflict into the present, emphasizing dramatic action as a way of helping group members solve their problems. Catharsis, the therapeutic release of emotions followed by relief, plays a prominent role. This approach is particularly useful for people who find it difficult to express their feelings in words.
- *Person-centered therapy*—a therapeutic approach developed by the **psychologist** Carl Rogers. Rather than viewing the therapist as expert, Rogers believed that the client's own drive toward growth and development is the most important healing factor. The therapist empathizes with the client's feelings and perceptions,

helping him or her gain insight and plan constructive action. Rogers's **person-centered therapy** became the basis for the intensive group experience known as the encounter group, in which the leader helps members discuss their feelings about one another and, through the group process, grow as individuals. Rogers emphasized honest feedback and the awareness, expression, and acceptance of feelings. He believed that a trusting and cohesive atmosphere is fundamental to the therapeutic effect of the group.

- *Gestalt therapy*—In the 1940s Fritz Perls challenged psychoanalytic theory and practice with this approach. Members take turns being in the "hot seat," an empty chair used to represent people with whom the person is experiencing conflicts. The therapist encourages the client to become aware of feelings and impulses previously denied.

BEHAVIOR THERAPIES. Behavior therapies comprise a number of techniques based upon a common theoretical belief: maladaptive behaviors develop according to the same principles that govern all learning. As a result, they can be unlearned, and new, more adaptive behaviors learned in their place. The emergence of behavior therapies in the 1950s represented a radical departure from psychoanalysis.

Behavior therapies focus on how a problem behavior originated, and on the environmental factors that maintain it. Individuals are encouraged to become self-analytical, looking at events occurring before, during, and after the problem behavior takes place. Strategies are then developed and employed to replace the problem behavior with new, more adaptive behaviors.

An important offshoot of behavior therapy is **cognitive-behavioral therapy**, developed in the 1960s and 1970s, which is the predominant behavioral approach used today. It emphasizes the examination of thoughts with the goal of changing them to more rational and less inflammatory ones. Albert Ellis, a psychologist who believed that we cause our own unhappiness by our interpretations of events, rather than by the events themselves, is a major figure in cognitive-behavior therapy. By changing what we tell ourselves, Ellis believes we can reduce the strength of our emotional reactions, as well.

Who belongs in a therapy group?

Individuals that share a common problem or concern are often placed in therapy groups where they can share their mutual struggles and feelings. Groups for bulimic individuals, victims of sexual **abuse**, adult children of alcoholics, and recovering drug addicts are some types of common therapy groups.

Individuals that are suicidal, homicidal, psychotic, or in the midst of a major life crisis are not typically placed in group therapy until their behavior and emotional states have stabilized. People with organic **brain** injury and other cognitive impairments may also be poor candidates for group therapy, as are patients with sociopathic traits, who show little ability to empathize with others.

How are therapy groups constructed?

Therapy groups may be homogeneous or heterogeneous. Homogeneous groups, described above, have members with similar diagnostic backgrounds (for example, they may all suffer from depression). Heterogeneous groups contain a mix of individuals with different emotional problems. The number of group members typically ranges from five to 12.

How do therapy groups work?

The number of sessions in group therapy depends upon the group's makeup, goals, and setting. Some are time limited, with a predetermined number of sessions known to all members at the beginning. Others are indeterminate, and the group and/or therapist determines when the group is ready to disband. Membership may be closed or open to new members. The therapeutic approach used depends on both the focus of the group and the therapist's orientation.

In group therapy sessions, members are encouraged to discuss the issues that brought them into therapy openly and honestly. The therapist works to create an atmosphere of trust and acceptance that encourages members to support one another. Ground rules may be set at the beginning, such as maintaining confidentiality of group discussions, and restricting social contact among members outside the group.

The therapist facilitates the group process, that is, the effective functioning of the group, and guides individuals in self-discovery. Depending upon the group's goals and the therapist's orientation, sessions may be either highly structured or fluid and relatively undirected. Typically, the leader steers a middle course, providing direction when the group gets off track, yet letting members set their own agenda. The therapist may guide the group by reinforcing the positive behaviors they engage in. For example, if one member shows empathy and supportive listening to another, the therapist might compliment that member and explain the value of that behavior to the group. In almost all group therapy situations, the therapist will emphasize the commonalities among members to instill a sense of group identity.

Self-help or **support groups** like Alcoholics Anonymous and Weight Watchers fall outside of the psy-

chotherapy realm. These groups offer many of the same benefits, including social support, the opportunity to identify with others, and the sense of belonging that makes group therapy effective for many. **Self-help groups** also meet to share their common concern and help one another cope. These groups, however, are typically leaderless or run by a member who takes on the leader role for one or more meetings. Sometimes self-help groups can be an adjunct to psychotherapy groups.

How are patients referred for group therapy?

Individuals are typically referred for group therapy by a psychologist or psychiatrist. Some may participate in both individual and group therapy. Before a person begins in a therapy group, the leader interviews the individual to ensure a good fit between their needs and the group's. The individual may be given some preliminary information before sessions begin, such as guidelines and ground rules, and information about the problem on which the group is focused.

How do therapy groups end?

Therapy groups end in a variety of ways. Some, such as those in drug rehabilitation programs and psychiatric hospitals, may be ongoing, with patients coming and going as they leave the facility. Others may have an end date set from the outset. Still others may continue until the group and/or the therapist believe the group goals have been met.

The termination of a long-term therapy group may cause feelings of **grief**, loss, abandonment, anger, or rejection in some members. The therapist attempts to deal with these feelings and foster a sense of closure by encouraging exploration of feelings and use of newly acquired coping techniques for handling them. Working through this termination phase is an important part of the treatment process.

Who drops out of group therapy?

Individuals who are emotionally fragile or unable to tolerate aggressive or hostile comments from other members are at risk of dropping out, as are those who have trouble communicating in a group setting. If the therapist does not support them and help reduce their sense of isolation and aloneness, they may drop out and feel like failures. The group can be injured by the premature departure of any of its members, and it is up to the therapist to minimize the likelihood of this occurrence by careful selection and management of the group process.

Results

Studies have shown that both group and individual psychotherapy benefit about 85% of the patients who participate in them. Ideally, patients leave with a better understanding and acceptance of themselves, and stronger interpersonal and coping skills. Some individuals continue in therapy after the group disbands, either individually or in another group setting.

See also Abuse; Addiction; Alcohol and related disorders; Amphetamines and related disorders; Anxiety and anxiety disorders; Bulimia nervosa; Cannabis and related disorders; Cocaine and related disorders; Cognitive-behavioral therapy; Grief counseling and therapy; Modeling; Nicotine and related disorders; Obesity; Opioids and related disorders; Peer groups; Psychodynamic therapy; Rational emotive therapy; Reinforcement; Self-help groups; Social skills training; Substance abuse and related disorders; Support groups

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ORGANIZATIONS

American Psychiatric Association. 1400 K Street NW, Washington DC 20005. (888) 357-7924. <http://www.psych.org>.

American Psychological Association (APA). 750 First Street NE, Washington, DC 20002-4242. (202) 336-5700. <//www.apa.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

Barbara S. Sternberg, Ph.D.

Guided imagery therapy

Definition

Guided imagery therapy is a cognitive-behavioral technique in which a client is guided in imagining a relaxing scene or series of experiences.

KEY TERMS

Rational emotive therapy—A form of psychotherapy developed by Albert Ellis and other psychotherapists based on the theory that emotional response is based on the subjective interpretation of events, not on the events themselves.

Purpose

Numerous clinical observations suggest that an individual visualizing an imagined scene reacts as though it were actually occurring; therefore, "induced" images can have a profound effect on behavior. The usefulness of guided imagery techniques have been shown to be effective in helping individuals learn or modify behaviors such as:

- learning to relax
- changing or controlling their negative emotions in response to a particular situation, event (loss of a job), or belief
- preparing themselves for changes they are likely to have to deal with in the future (children leaving home, parent moving)
- eliminating or reducing undesirable behaviors (smoking, obesity)
- increasing effective pain management
- coping with difficult situations (a difficult boss)
- learning new and desirable behaviors (assertiveness)
- becoming more motivated (doing homework between therapy sessions) in dealing with their problems
- coping with how they behaved in an earlier situation (had a temper tantrum) in order to feel less shame or guilt
- experimenting with ways to manage stressful or anxiety-producing situations (giving a presentation in public) by mentally rehearsing the needed behavior(s)

Guided imagery techniques have been applied to—and found to be effective or show promise with—a variety of populations, including individuals with:

- phobias (including **agoraphobia**, **social phobia**, and **specific phobias**)
- mild to moderate depression
- generalized anxiety disorders
- **post-traumatic stress disorder**
- **obsessive-compulsive disorder**
- sexual difficulties
- habit disorders
- chronic fatigue syndrome
- children's behavioral disorders
- stuttering
- acute and chronic pain (and other physical disorders)

Guided imagery has also contributed to the achievement of skills and overcoming anxiety in normal life situations that include learning or improving motor skills, test taking, and public speaking. In addition, visualization and imagery, along with other behavioral techniques, have been applied to the fields of business, industry, child rearing, education, behavioral medicine, and sports.

Description

Imagery techniques have been combined with a wide range of behavioral and cognitive procedures and treatment methods of some psychotherapeutic approaches, including **behavior modification**, cognitive processing therapy, **rational emotive therapy**, multimodal therapy, and **hypnotherapy**. Combinations of treatment methods among these approaches leads to the following general uses of imagery:

- antifuture shock imagery (preparing for a feared future event)
- positive imagery (using pleasant scenes for relaxation training)
- aversive imagery (using an unpleasant image to help eliminate or reduce undesirable behavior)
- associated imagery (using imagery to track unpleasant feelings)
- coping imagery (using images to rehearse to reach a behavioral goal or manage a situation
- "step-up" technique (exaggerating a feared situation and using imagery to cope with it)

An assessment of the individual's presenting problems is an essential part of treatment, both at the beginning of therapy and throughout the entire process. This is to ensure that the therapist has sufficient understanding of the client's situation and **diagnosis** of the problem(s). The assessment generally covers a variety of areas, such as developmental history (including family, education, employment, and social relationships), past traumatic experiences, medical and psychiatric treatments, and client goals. Often, clients have several problems, and both the therapist and the client work together on prioritizing specific treatment goals.

Following the assessment, the therapist will present a general rationale for the use of imagery. The therapist might explain that the client will learn techniques in which he or she imagines they or another person are performing a particular behavior. To enhance visualization, it is important to involve all senses in the image. For example, if the client is to be walking down a busy street, he or she is encouraged to imagine hearing sounds from traffic and other people, smell exhaust fumes from buses and aromas from a nearby bakery, and observe body movements and wind in the face. It is stressed to the client that the most critical aspect of imagining is the feeling of actually experiencing the scene—of being in it rather than just seeing oneself in it.

Both the therapist and the client construct a relaxing scene by discussing exactly what the client finds pleasant. It is better if the client chooses all images (positive or negative) and the therapist trains the client to visualize the selected images as vividly as possible.

Once a pleasant scene is decided upon, the client is asked to assume a relaxed position and with closed eyes, if this is comfortable, before being guided in visualization. A common beginning instruction may be: "Imagine you are lying on a warm sandy beach." The therapist continues to guide the relaxation by saying such phrases as: "Notice the texture of the sand and the color of the sky. Focus on the sounds you hear, and the smells..." The client is asked to practice the image at home between sessions. A tape of the guided imagery in the familiar voice of the therapist can be helpful to some clients in practicing at home.

During visualization, clients are given permission to take control if they need to by changing the image or stopping the activity completely. To help clients maintain control of the image, the therapist may also say to the client: "Take as long as you need to relax," and "Do whatever you need to do in order to feel safe." This empowers clients in using such techniques.

Length of treatment

Treatments using behavioral techniques tend to be relatively brief. However, many factors determine the length of therapy. Generally, treatment takes longer if target behaviors are more numerous and more difficult to specify. Some types of treatments require more sessions than others. For example, techniques using imagery require more sessions than treatments in which the client is exposed to the actual feared situations in real life.

Other factors that determine the length of treatment are the types of presenting disorders, the client's willingness to do homework, how long the client has had the problem, client financial resources, and whether there are supportive family members and friends. The therapist's style and experience may also affect the length of therapy. Clients may be seen several times (two to five times) a week at the start of therapy and then once weekly for several months, and every other month for follow-up for a few more months.

Normal results

Guided imagery techniques have been taken from behavior therapy and are used by different psychological theories and systems of counseling and **psychotherapy**, including **cognitive-behavioral therapy**. Research has shown these techniques to be effective when applied to specific problems.

Depending on the combination of visualization and imagery techniques used, the therapeutic approach, and client problem(s), it is expected that clients will have positive changes in specifically defined target behaviors; a reduction in biases or distortions in thinking, resulting in more effective functioning that, in turn, leads to more positive feelings, behavior, and thinking; and experience less emotional disturbances, increased effective coping skills, decreased self-defeating behaviors, and less tension.

Abnormal results

Guided imagery is not used in isolation but as a part of a therapeutic formulation and is appropriate for a range of problems and disorders. It is, however, thought that some techniques—such as imagery used in rational-emotive therapy—can trigger high levels of anxiety in some clients. Therefore, caution should be taken when using these techniques if clients have the following conditions:

- asthma attacks triggered by stress or anxiety
- seizures triggered by stress or anxiety
- cardiac condition or related conditions
- depression with suicidal ideation
- hysteria
- pregnancy
- severe psychiatric disorders

In these instances, other strategies and techniques that do not trigger high levels of anxiety, such as relaxation exercises or coping imagery, should be considered. When working with clients with these conditions, the therapist should be in consultation with their medical provider.

See also Aversion therapy; Covert sensitization

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Anxiety Disorders Association of America. 11900 Parklawn Dr., Suite 100,Rockville MD 20852-2624. <http://www.adaa,org/>.

National Institute of Mental Health. 6001 Excutive Boulevard, Room 8184,MSC 9663, Bethesda MD 20892-9663. <http://www.nimh.nih.gov/>.

National Mental Health Association. 1021 Prince Street, Alexander, VA 22314-2071. <http://www.nmha.org>.

Janice VanBuren, Ph.D.

H

Halcion *see* **Triazolam**

Haldol *see* **Haloperidol**

Hallucinations

Description

A hallucination is a false perception occurring without any identifiable external stimulus and indicates an abnormality in perception. The false perceptions can occur in any of the five sensory modalities. Therefore, a hallucination essentially is seeing, hearing, tasting, feeling, or smelling something that is not there. The false perceptions are not accounted for by the person's religious or cultural background, and the person experiencing hallucinations may or may not have insight into them. Therefore, some people experiencing hallucinations may be aware that the perceptions are false, whereas others may truly believe that what they are seeing, hearing, tasting, feeling, or smelling is real. In cases when the person truly believes the hallucination is real, the individual may also have a delusional interpretation of the hallucination.

Hallucinations must be distinguished from illusions, which are misperceptions of actual external stimuli. In other words, an illusion is essentially seeing, hearing, tasting, feeling, or smelling something that is there, but perceiving it or interpreting it incorrectly. An example of an illusion might be hearing one's name called when the radio is playing. There is an external auditory stimulus, but it is misperceived. True hallucinations do not include false perceptions that occur while dreaming, while falling asleep, or while waking up. Unusual perceptual experiences one may have while falling asleep are referred to as hypnagogic experiences. Unusual perceptual experiences one may have while waking up are referred to as hypnopompic experiences. Hallucinations also do not include very vivid experiences one may have while fully awake (such as especially vivid daydreaming or imaginative play).

Hallucinations are a symptom of either a medical (e.g., epilepsy), neurological, or mental disorder. Hallucinations may be present in any of the following mental disorders: psychotic disorders (including **schizophrenia**, **schizoaffective disorder**, **schizophreniform disorder**, **shared psychotic disorder**, **brief psychotic disorder**, **substance-induced psychotic disorder**), **bipolar disorder**, major depression with psychotic features, **delirium**, or **dementia**. Auditory hallucinations, in particular, are common in psychotic disorders such as schizophrenia.

Use of certain recreational drugs may induce hallucinations, including **amphetamines** and cocaine, hallucinogens (such as lysergic acid diethylamide or LSD), phencyclidine (PCP), and cannabis or marijuana. For example, visual hallucinations are commonly associated with substance use. Individuals may report false perceptions of little people or animals (sometimes referred to as Lilliputian hallucinations). In addition, withdrawal from some recreational drugs can produce hallucinations, including withdrawal from alcohol, sedatives, hypnotics, or anxiolytics. Withdrawal from alcohol, for instance, commonly causes visual hallucinations, especially at nighttime.

Types

Hallucinations are categorized according to which sensory modality is involved and, in addition, are categorized as either mood-congruent or mood-incongruent. The types of hallucinations are:

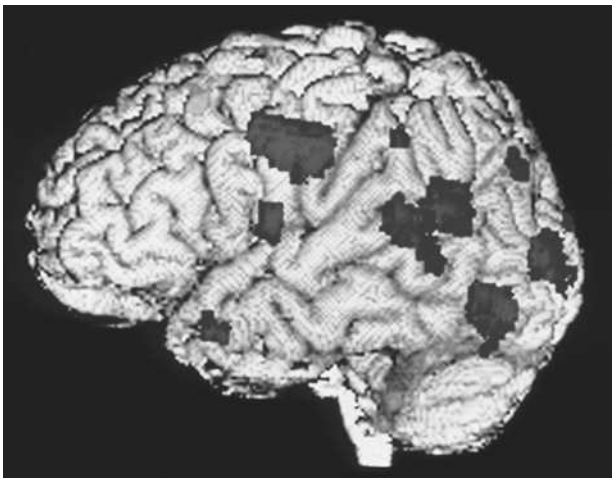
- Auditory: The false perception of sound, music, noises, or voices. Hearing voices when there is no auditory stimulus is the most common type of auditory hallucination in mental disorders. The voice may be heard either inside or outside one's head and is generally considered more severe when coming from outside one's head. The voices may be male or female, recognized as

KEY TERMS

Illusion—A misperception or misinterpretation in the presence of a real external stimulus.

the voice of someone familiar or not recognized as familiar, and may be critical or positive. In mental disorders such as schizophrenia, however, the content of what the voices say is usually unpleasant and negative. In schizophrenia, a common symptom is to hear voices conversing and/or commenting. When someone hears voices conversing, they hear two or more voices speaking to each other (usually about the person who is hallucinating). In voices commenting, the person hears a voice making comments about his or her behavior or thoughts, typically in the third person (such as, "isn't he silly"). Sometimes the voices consist of hearing a "running commentary" on the person's behavior as it occurs ("she is showering"). Other times, the voices may tell the person to do something (commonly referred to as "command hallucinations").

- Gustatory: A false perception of taste. Usually, the experience is unpleasant. For instance, an individual may complain of a persistent taste of metal. This type of hallucination is more commonly seen in some medical disorders (such as epilepsy) than in mental disorders.
- Olfactory hallucination: A false perception of odor or smell. Typically, the experience is very unpleasant. For example, the person may smell decaying fish, dead bodies, or burning rubber. Sometimes, those experiencing olfactory hallucinations believe the odor emanates from them. Olfactory hallucinations are more typical of medical disorders than mental disorders.
- Somatic/tactile hallucination: A false perception or sensation of touch or something happening in or on the body. A common tactile hallucination is feeling like something is crawling under or on the skin (also known as formication). Other examples include feeling electricity through one's body and feeling like someone is touching one's body but no one is there. Actual physical sensations stemming from medical disorders (perhaps not yet diagnosed) and hypochondriacal preoccupations with normal physical sensations, are not thought of as somatic hallucinations.
- Visual hallucination: A false perception of sight. The content of the hallucination may be anything (such as shapes, colors, and flashes of light) but are typically people or human-like figures. For example, one may perceive a person standing before them when no one is there. Sometimes an individual may experience the false perception of religious figure (such as the devil, or Christ). Perceptions that would be considered normal for an individual's religion or culture are not considered hallucinations.



Colored positron emission tomography scan (PET scan) of the brain of a patient with schizophrenia who is experiencing a hallucination. Highlighted areas show brain activity. The patient's hallucination consisted of heads that spoke to him. The active areas of the brain seen here (the auditory and visual areas) confirm that the patient "saw" and "heard" the heads in the hallucination. *(Wellcome Dept of Cognitive Neurology. Photo Researchers, Inc./Science Source. Reproduced by permission.) See color insert for color version of photo.*

- Mood-congruent hallucination: Any hallucination whose content is consistent with either the depressive or manic state the person may be in at the time. Depressive themes include guilt, death, disease, personal inadequacy, and deserved punishment. Manic themes include inflated self-worth, power, knowledge, skills, and identity and a special relationship with a famous person or deity. For example, a depressed person may hear voices saying that he or she is a horrible person, whereas a manic person may hear voices saying that he or she is an incredibly important person.
- Mood-incongruent hallucination: Any hallucination whose content is not consistent with either the depressed or manic state the person is in at the time, or is mood-neutral. For example, a depressed person may experience hallucinations without any themes of guilt, death, disease, personal inadequacy, or deserved punishment. Similarly, a manic person may experience hallucinations without any themes of inflated self-worth, power, knowledge, skills, or identity or a special relationship to a famous person or deity.

See also Alcohol and related disorders; Major depressive disorder; Substance abuse and related disorders; Substance-induced psychotic disorders

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Jennifer Hahn, Ph.D.

Hallucinogens and related disorders

Definition

Hallucinogens are a chemically diverse group of drugs that cause changes in a person's thought processes, perceptions of the physical world, and sense of time passing. Hallucinogens can be found naturally in some plants, and can be synthesized in the laboratory. Most hallucinogens are abused as recreational drugs. Hallucinogens are also called psychedelic drugs.

Description

Hallucinogens are as old as civilization. Many cultures recorded eating certain plants specifically to induce visions or alter the perception of reality. Often these **hallucinations** were part of a religious or prophetic experience. Shamans in Siberia were known to eat the hallucinogenic mushroom *Amanita muscaria*. The ancient Greeks and the Vikings also used naturally occurring plant hallucinogens. Peyote, a spineless cactus native to the southwestern United States and Mexico was used by native peoples, including the Aztecs, to produce visions.

Although several hundred plants are known to contain compounds that cause hallucinations, most hallucinogens are synthesized in illegal laboratories for delivery as street drugs. The best known hallucinogens are lysergic acid diethylamide (LSD), mescaline, psilocybin, and MDMA (ecstasy). Phencyclidine (PCP, angel dust) can produce hallucinations, as can **amphetamines** and marijuana, but these drugs are not considered classic hallucinogens and are discussed under separate entries. In addition, new designer drugs that are chemical variants of classic hallucinogens are apt to appear on the street at any time.

Although hallucinogens produce similar physical and psychological effects, they are a diverse group of compounds. However, all hallucinogens appear to affect the **brain** in similar ways. Although the mechanism of action of hallucinogens is not completely understood, researchers have shown that these drugs bind with one type of serotonin receptor ($5\text{-}HT_2$) in the brain.

Serotonin is a neurotransmitter that facilitates transmission of nerve impulses in the brain and is associated with feelings of well-being, as well as many physiological responses. When a hallucinogenic compound binds with serotonin receptors, serotonin is blocked from those receptor sites, and nerve transmission is altered. There is an increase in free (unbound) serotonin in the brain. The result is a distortion of the senses of sight, sound, and touch, disorientation in time and space, and alterations of mood. In the case of hallucinogen intoxication, however, a person is not normally delirious, unconscious, or dissociated. He or she is aware that these changes in perception are caused by the hallucinogen.

LSD

LSD is the best known and most potent of the hallucinogens. LSD was first synthesized by Alfred Hoffman for a pharmaceutical company in Germany in 1938 while searching for a headache remedy. Hoffman discovered the hallucinogenic properties of LSD accidentally in 1943. The drug became popular with hippies in the mid-1960s when its sense-altering properties were reputed to offer a window into enhanced creativity and self-awareness. LSD also occurs naturally in morning glory seeds.

Pure LSD is a white, odorless, crystalline powder that dissolves easily in water, although contaminants can cause it to range in color from yellow to dark brown. LSD was listed as a Schedule I drug under the Controlled Substance Act of 1970, meaning that it has no medical or legal uses and has a high potential for abuse. LSD is not easy to manufacture in a home laboratory, and some of its ingredients are controlled substances that are difficult to obtain. However, LSD is very potent, and a small amount can produce a large number of doses.

On the street, LSD is sold in several forms. Microdots are tiny pills smaller than a pinhead. Windowpane is liquid LSD applied to thin squares of gelatin. Liquid LSD can also be sprayed on sugar cubes. The

KEY TERMS

Dissociated—Feelings of experiencing an altered state of reality, similar to a trance state. During the period of dissociation, the affected person may feel as if he or she is an observer instead of a participant in events, and may feel as if surroundings are unreal or distorted.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

most common street form of the drug is liquid LSD sprayed onto blotter paper and dried. The paper, often printed with colorful or psychedelic pictures, is divided into tiny squares, each square being one dose. Liquid LSD can also be sprayed on the back of a postage stamp and licked off. Street names for the drug include acid, yellow sunshine, windowpane, cid, doses, trips, and boomers.

Mescaline

Mescaline is a naturally occurring plant hallucinogen. Its primary source is the cactus *Lophophora williamsii*. This cactus is native to the southwestern United States and Mexico. The light blue-green plant is spineless and has a crown called a peyote button. This button contains mescaline and can be eaten or made into a bitter tea. Mescaline is also the active ingredient of at least ten other cacti of the genus *Trichocereus* that are native to parts of South America.

Mescaline was first isolated in 1897 by the German chemist Arthur Hefftner and first synthesized in the laboratory in 1919. Some experiments were done with the drug to determine if it was medically useful, but no medical uses were found. However, peyote is culturally significant. It has been used for centuries as part of religious celebrations and vision quests of Native Americans. The Native American Church, which fuses elements of Christianity with indigenous practices, has long used peyote as part of its religious practices.

In 1970 mescaline was listed as a Schedule I drug under the Controlled Substances Act. However, that same year the state of Texas legalized peyote for use in Native American religious ceremonies. In 1995, a federal law was passed making peyote legal only for this use in all 50 states.

Psilocybin

Psilocybin is the active ingredient in what are known on the street as magic mushrooms, shrooms, mushies, or Mexican mushrooms. There are several species of mushrooms that contain psilocybin, including *Psilocybe mexicana, P. muscorumi,* and *Stropharia cubensis*. These mushrooms grow in most moderate, moist climates.

Psilocybin-containing mushrooms are usually cooked and eaten (they have a bitter taste), or dried and boiled to make a tea. Although psilocybin can be made synthetically in the laboratory, there is no street market for synthetic psilocybin, and virtually all the drug comes from cultivated mushrooms. In the United States, it is legal to possess psilocybin-containing mushrooms, but it is illegal to traffic in them, and psilocybin and psilocyn (another psychoactive drug found in small quantities in these mushrooms) are both Schedule I drugs.

MDMA

MDMA, short for 3,4-methylenedioxymethamphetamine, and better known as ecstasy, TXC, E, X, or Adam, has become an increasingly popular club drug since the 1980s. MDMA was first synthesized in 1912 by a German pharmaceutical company looking for a new compound that would stop bleeding. The company patented the drug, but never did anything with it. A closely related drug, methylenedioxyamphetimine or MDA, was tested by a pharmaceutical company as an appetite suppressant in the 1950s, but its use was discontinued when it was discovered to have hallucinogenic properties. In the 1960s, MDA was a popular drug of abuse in some large cities such as San Francisco.

During the early 1980s therapists experimented with MDMA, which was legal at the time, as a way to help patients open up and become more empathetic. Recreational use soon followed. The drug was declared an illegal Schedule I drug in 1985. For about a year between 1987 and 1988, the drug was again legal as the result of court challenges, but it permanently joined other Schedule I hallucinogens in March 1988.

MDMA is a popular club drug and can be obtained at all-night raves or dance parties. The drug, sold in tablets, is attractive because it combines stimulant effects that allow ravers to dance for hours with a feeling of empathy, reduced anxiety, and reduced inhibitions, and euphoria. Some authorities consider MDA and MDMA stimulant-hallucinogens and do not group them with classic hallucinogens such as LSD, but research indicates that MDA and MDMA affect the brain in the same way as classic hallucinogens. The American Psychiatric

Association considers MDMA as a drug that can cause hallucinogen-related disorders.

Causes and symptoms

A cause of hallucinogen use is that hallucinogens are attractive to recreational drug users for a number of reasons, including:

- They are minimally addictive and there are no physical withdrawal symptoms upon stopping use.
- They produce few serious or debilitating physical side effects.
- They do not usually produce a delusional state, excessive stupor, or excessive stimulation.
- They do not cause memory loss with occasional use.
- They are easily and cheaply available.
- They produce a high that gives the illusion of increasing creativity, empathy, or self-awareness.
- Deaths from overdoses are rare.

On the other hand, strong hallucinogens such as LSD can cause frightening and anxiety-evoking emotional experiences, known as bad trips. Flashbacks, where the sensations experienced while under the influence of a drug recur uncontrollably without drug use, can occur for months after a single drug use. During hallucinogen intoxication, reality may be so altered that a person may endanger himself by believing he is capable of feats such as flying off buildings. Hallucinogens also may induce or cause a worsening of latent psychiatric disorders such as anxiety, depression, and **psychosis**. Hallucinogens can also cause **paranoia**, long-term memory loss, personality changes (especially if there is a latent psychiatric disorder), and psychological drug dependence.

Psychological symptoms

Hallucinogens work primarily on the perception of reality. They usually do not create true hallucinations, which are imagined visions or sounds (voices heard in the head, for example) in the absence of any corresponding reality. Instead, classic hallucinogens alter the perception of something that is physically present. A face may appear to "melt" or colors may become brighter, move, and change shape. Sounds may be "seen," rather than heard.

More than with other drugs, the mental state of the hallucinogen user and the environment in which the drug is taken influence the experience a user has. LSD, especially, is known for symptoms that range from mellowness and psychedelic visions (good trips) to anxiety and panic attacks (bad trips). Previous good experiences with a drug do not guarantee continued good experiences. People with a history of psychiatric disorders are more likely to experience harmful reactions, as are those who are given the drug without their knowledge.

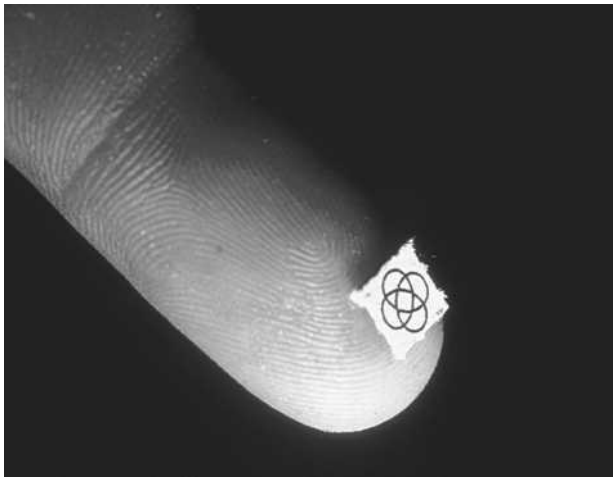
The main psychological symptoms of hallucinogens are listed below. Normally, mescaline and psilocybin produce uniformly milder symptoms than LSD. During a single drug experience, the user can experience a range of symptoms. Mood can shift from happy to sad or pleasant to frightening and back again several times. Some symptoms occur primarily with MDMA, as indicated. Psychological symptoms of hallucinogen intoxication include:

- distortion of sight, sound, and touch
- confusion of the senses—sounds are "seen" or vision is "heard"
- disorientation in time and space
- **delusions** of physical invulnerability (especially with LSD)
- paranoia
- unreliable judgment and increased risk taking
- anxiety attacks
- flashbacks after the drug has been cleared from the body
- blissful calm or mellowness
- reduced inhibitions
- increased empathy (MDMA)
- elation or euphoria
- impaired concentration and motivation
- long-term memory loss
- personality changes, especially if there is a latent psychiatric disorder
- psychological drug dependence

Physical symptoms

Although the primary effects of hallucinogens are on perceptions, some physical effects do occur. Physical symptoms include:

- increased blood pressure
- increased heart rate
- nausea and vomiting (especially with psilocybin and mescaline)
- blurred vision which can last after the drug has worn off
- poor coordination
- enlarged pupils



LSD on a small piece of blotter paper shown on the tip of a finger. *(Custom Medical Stock Photo, Inc. Reproduced by permission.)*

- sweating
- diarrhea (plant hallucinogens)
- restlessness
- muscle cramping (especially clenched jaws with MDMA)
- dehydration (MDMA)
- serious increase in body temperature leading to **seizures** (MDMA)

Demographics

Hallucinogen use, excluding MDMA, peaked in the United States late 1960s as part of the hippie movement. Hallucinogen use then gradually declined until the early 1990s, when it again picked up. Usage appears to have peaked around 1998, and may now be remaining steady or declining. Hallucinogens are drugs normally used by adolescents and young adults in social settings such as dance parties or raves. Even heavy users do not use these drugs more than two or three times a week. Casual or occasional use is common and many people outgrow their use.

Unlike LSD use, MDMA use has increased enormously since the 1980s. Between 1995 and 1999 its use by 18- to 25-year-olds increased 47%. In a survey of 400 hospitals, MDMA-related emergency room visits increased 58% between 1999 and 2000. One-third of these visits involved people under the age of 25. In 2000, the last year for which statistics are available, the National Institute of Drug Abuse found that about 11% of twelfth-grade students had used LSD and about 7% reported using other hallucinogens. People who use hallucinogens are also likely to abuse alcohol and marijuana.

Diagnosis

Although not all experts agree, the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*), which presents guidelines used by the American Psychiatric Association for **diagnosis** of mental disorders, recognizes two hallucinogen-related disorders: hallucinogen dependence and hallucinogen abuse. Hallucinogen dependence is the continued use of hallucinogens even when the substances cause the affected individual significant problems, or when the individual knows of adverse effects (memory impairment while intoxicated, anxiety attacks, flashbacks), but continues to use the substances anyway. "Craving" hallucinogens after not using them for a period of time has been reported. Hallucinogen abuse is repeated use of hallucinogens even after they have caused the user impairment that undermines his or her ability to fulfill obligations at work, school, or home, but the use is usually not as frequent as it is among dependent users. In addition to these two disorders, the American Psychiatric Association recognizes eight hallucinogen-induced disorders. These are:

- hallucinogen intoxication
- hallucinogen persistent perception disorder (flashbacks)
- hallucinogen intoxication **delirium**
- hallucinogen-induced psychotic disorder with delusions
- hallucinogen-induced psychotic disorder with hallucinations
- hallucinogen-induced mood disorder
- hallucinogen-induced anxiety disorder
- hallucinogen-related disorder not otherwise specified

Hallucinogen dependence and abuse are normally diagnosed from reports by the patient or person accompanying the patient of use of a hallucinogenic drug. Active hallucinations and accompanying physical symptoms can confirm the diagnosis, but do not have to be present. Routine drug screening does not detect LSD in the blood or urine, although specialized laboratory methods can detect the drug. Hallucinogen dependence differs from other drug dependence in that there are no withdrawal symptoms when the drug is stopped, and the extent of tolerance, (needing a higher and higher dose to achieve the same effect) appears minimal.

Hallucinogen intoxication is diagnosed based on psychological changes, perceptual changes, and physical

symptoms that are typical of hallucinogen use. These changes must not be caused by a general medical condition, other substance abuse, or another mental disorder.

Hallucinogen persisting perception disorder, better known as flashbacks, occur after hallucinogen use followed by a period of lucidity. Flashbacks may occur weeks or months after the drug was used, and may occur after a single use or many uses.

To be diagnosed as a psychiatric disorder, flashbacks must cause significant distress or interfere with daily life activities. They can come on suddenly with no warning, or be triggered by specific environments. Flashbacks may include emotional symptoms, seeing colors, geometric forms, or, most commonly, persistence of trails of light across the visual field. They may last for months. Flashbacks are most strongly associated with LSD.

Hallucinogen intoxication delirium is rare unless the hallucinogen is contaminated by another drug or chemical such as strychnine. In hallucinogen intoxication, the patient is still grounded in reality and recognizes that the experiences of altered perception are due to using a hallucinogen. In hallucinogen intoxication delirium, the patient is no longer grounded in reality. Hallucinogen-induced psychotic disorders are similar in that the patient loses touch with reality. Psychotic states can occur immediately after using the drug, or days or months later.

Hallucinogen-induced mood disorder and hallucinogen-induced anxiety disorder are somewhat controversial, as hallucinogen use may uncover latent or pre-existing anxiety or mood disorders rather than being the cause of them. However, it does appear that MDMA use can cause major depression.

Treatments

Acute treatment is aimed at preventing the patient from harming himself or anyone else. Since most people experiencing hallucinogen intoxication remain in touch with reality, "talking down" or offering reassurance and support that emphasizes that the bad trip, anxiety, **panic attack**, or paranoia will pass as the drug wears off is often helpful. Patients are kept in a calm, pleasant, but lighted environment, and are encouraged to move around while being helped to remain oriented to reality. Occasionally, drugs such as **lorazepam** are given for anxiety. Complications in treatment occur when the hallucinogen has been contaminated with other street drugs or chemicals. The greatest life-threatening risk is associated with MDMA. Users may develop dangerously high body temperatures. Reducing the patient's temperature is an essential acute treatment.

Treatment for long-term effects of hallucinogen use involve long-term **psychotherapy** after drug use has stopped. Many people find 12-step programs or group support helpful. In addition, underlying psychiatric disorders must be addressed.

Prognosis

Because hallucinogens are not physically addictive, many people are able to stop using these drugs successfully. However, users may be haunted by chronic problems such as flashbacks or mood and anxiety disorders either brought about or worsened by use of hallucinogens. It is difficult to predict who will have long-term complications and who will not.

Prevention

Hallucinogen use is difficult to prevent, because these drugs have a reputation for being non-addictive and "harmless." Drug education and social outlets that provide people with a sense of self-worth are the best ways to prevent hallucinogen and other substance abuse.

See also Amphetamines and related disorders; Cannabis and related disorders; Phencyclidine and related disorders

Resources

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National Clearinghouse for Alcohol and Drug Information. P. O. Box 2345, Rockville, MD 20852. (800) 729-6686. <http://www.health.org>.

National Institute on Drug Abuse. 5600 Fishers Lane, Room 10 A-39, Rockville, MD 20857. (888) 644-6432. <http://niad.nih.gov>.

Partnership for a Drug-Free America. 405 Lexington Avenue, New York, NY 10174. (212) 922-1560. <http://www.drugfreeamerica.org>.

Tish Davidson, A.M.

Haloperidol

Definition

Haloperidol is a major tranquilizer. It is used to treat psychoses, senile **dementia**, Tourette's syndrome, and certain serious behavioral disorders in children. In the United States it is sold under the brand name Haldol.

Purpose

Haloperidol is used to control symptoms of psychotic disorders, such as **schizophrenia**. It is also used for controlling tics and inappropriate vocalizations associated with Tourette's syndrome in both children and adults.

In children, haloperidol is occasionally used to treat severe behavior problems such as combativeness and extreme outbursts that that occur without immediate provocation. Occasionally it is used for short-term treatment of children who display excessive motor activity with accompanying difficulty in attention, aggression, impulse control, mood changes, and coping with frustration. Haloperidol is used only after **psychotherapy** and other medications have been tried and found to be unsuccessful.

Description

Haloperidol is a major tranquilizer, and can be administered as a pill or by intramuscular injection (a shot).

The precise way in which haloperidol helps control symptoms associated with psychoses or dementia has not yet been clearly established.

Recommended dosage

For adults, the recommended initial dosage of haloperidol is 0.5 –5.0 mg two or three times each day. The initial dosage depends on the severity of the symptoms in the person being treated. All people taking haloperidol must be carefully monitored to establish an individualized dosage. Physicians have found that there is great variability in the amount of haloperidol required to control symptoms.

Children require smaller dosages of haloperidol than do adults. The recommended initial dosage of haloperidol for controlling psychotic symptoms in children is 0.5–2.0 mg two or three times each day. The recommended dosage for controlling symptoms of Tourette's syndrome and other non-psychotic disorders is between 0.075 and 0.05 mg per kilogram of body weight per day. The total dosage is usually divided into two or three administrations per day. The goal of therapy is to use the smallest amount of haloperidol that will control symptoms. Children under age three should not take this drug.

KEY TERMS

Anticoagulant—A medication (such as warfarin, Coumadin, or Heparin) that decreases the blood's clotting ability preventing the formation of new clots. Although anticoagulants will not dissolve existing clots, they can stop them from getting larger. These drugs are commonly called blood thinners.

Anticonvulsant—A medication used to control abnormal electrical activity in the brain that causes seizures.

Psychosis—Severe state characterized by loss of contact with reality, and is usually one feature of an overarching disorder, not a disorder in itself. (Plural: psychoses)

Tic—A sudden involuntary behavior that is difficult or impossible for the person to suppress. Tics may be either motor (related to movement) or vocal, and may become more pronounced under stress.

Tourette syndrome—Neurological disorder characterized by multiple involuntary movements and uncontrollable vocalizations called tics that come and go over years, usually beginning in childhood and becoming chronic. Sometimes the tics include inappropriate language.

Tranquilizer—A medication that induces a feeling of calm and relaxation.

Precautions

Haloperidol may cause low blood pressure (hypotension). For this reason people with heart and blood pressure problems should be carefully monitored while taking the drug. Haloperidol also increases the possibility of having **seizures**. People with a history of seizures or who are taking anticonvulsants (medication to control seizures) should take lower dosages of haloperidol and be closely monitored by a physician until a safe dosage is established. Haloperidol also interferes with the action of the anticoagulant (blood-thinning) drug phenindione.

Haloperidol may increase the action of central nervous system depressants such as anesthetics, alcohol, and opiates (some pain killers and sleeping pills). It may also decrease the time required to change from mania to depression among persons with **bipolar disorder** (also known as manic-depressive disorder).

Side effects

Haloperidol has the potential to produce a serious side effect called **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements that may not disappear or may only partially improve after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. These side effects may appear after people have stopped taking haloperidol. The chance of developing tardive dyskinesia increases with increasing age and with increasing dosage of haloperidol. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period.

Haloperidol use may lead to the development of symptoms that resemble Parkinson's disease, but that are not caused by Parkinson's. These symptoms may include a taut or mask-like expression on the face, drooling, tremors, pill-rolling motions in the hands, cogwheel rigidity (abnormal rigidity in muscles, characterized by jerky movements when the muscle is passively stretched), and a shuffling gait. Taking the anti-Parkinson drugs **benztropine** mesylate or **trihexyphenidyl** hydrochloride along with haloperidol help to control these symptoms. Medication to control Parkinsonian-like symptoms may have to be continued after haloperidol is stopped. This is due to different rates of elimination of these drugs from the body.

Other side effects of haloperidol include anxiety, restlessness, agitation, **insomnia**, headache, euphoria, drowsiness, depression, confusion, dizziness, and seizures. Unwanted or unexpected effects associated with the use of haloperidol have been reported for virtually all organ systems in the body. Although numerous, such side effects are relatively uncommon.

Interactions

The simultaneous use of haloperidol and lithium, a common treatment for bipolar disorder, has been associated with an encephalopathic syndrome. People with this syndrome have symptoms of weakness, lethargy, fever, confusion, and high levels of white blood cells.

Haloperidol may increase the effect of central nervous system depressants such as anesthetics, opiates, and alcohol.

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American Academy of Clinical Toxicology. 777 East Park Drive, PO Box 8820, Harrisburg, PA 17105-8820. Phone: (717) 558-7750. Fax: (717) 558-7845. Web site: <http://www.clintox.org/index.html>.

American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Phone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Phone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850, Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

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Halstead-Reitan Battery

Definition

The Halstead-Reitan Neuropsychological Test Battery is a fixed set of eight tests used to evaluate **brain** and nervous system functioning in individuals aged 15 years and

KEY TERMS

Abstraction—Ability to think about concepts or ideas separate from specific examples.

Aphasia—Loss of previously acquired ability to understand or use written or spoken language, due to brain damage or deterioration.

Cutoff scores—In psychological testing, scores that indicate the borderline between normal and impaired functioning.

Dominant hand—The hand that one prefers to use when performing various tasks such as writing or throwing an object.

Lateralization—The control of specific neurological functions by one side of the brain or the other; for example, in most right-handed people, language functions are controlled by the left side of the brain and spatial and visual functions are controlled by the right side of the brain.

Localization—The control of specific neurological functions by specific areas in the brain.

Motor—Involving muscle movement.

Neurologic—Pertaining to the nervous system (brain and nerve cells).

Neuropsychological functioning—The ability of the nervous system and brain to process and interpret information received through the senses.

Non-dominant hand—The hand that one does not typically use when performing various tasks such as writing or throwing an object.

Tactile/tactual—Perceptible by touch.

older. Children's versions are the Halstead Neuropsychological Test Battery for Older Children (ages nine to 14) and the Reitan Indiana Neuropsychological Test Battery (ages five to eight).

Purpose

Neuropsychological functioning refers to the ability of the nervous system and brain to process and interpret information received through the senses. The Halstead-Reitan evaluates a wide range of nervous system and brain functions, including: visual, auditory, and tactual input; verbal communication; spatial and sequential perception; the ability to analyze information, form mental concepts, and make judgments; motor output; and attention, concentration, and memory.

The Halstead-Reitan is typically used to evaluate individuals with suspected brain damage. The battery also provides useful information regarding the cause of damage (for example, closed head injury, alcohol abuse, Alzheimer's disorder, **stroke**), which part of the brain was damaged, whether the damage occurred during childhood development, and whether the damage is getting worse, staying the same, or getting better. Information regarding the severity of impairment and areas of personal strengths can be used to develop plans for rehabilitation or care.

Precautions

Due to its complexity, the Halstead-Reitan requires administration by a professional examiner and interpretation by a trained **psychologist**. Test results are affected by the examinee's age, education level, intellectual ability, and—to some extent—gender or ethnicity, which should always be taken into account. Because the Halstead-Reitan is a fixed battery of tests, some unnecessary information may be gathered, or some important information may be missed. Overall, the battery requires five to six hours to complete, involving considerable patience, stamina, and cost. The battery has also been criticized because it does not include specific tests of memory; rather, memory is evaluated within the context of other tests.

Description

Ward Halstead and Ralph Reitan are the developers of the Halstead-Reitan battery. Based on studies of patients with neurologic impairments at the University of Chicago, Halstead recognized the need for an evaluation of brain functioning that was more extensive than intelligence testing. He began experimenting with psychological tests that might help identify types and severity of brain damage through observation of a person's behavior in various tasks involving neuropsychological abilities. Initially he chose a set of ten tests; all but three are in the current Halstead-Reitan battery.

Ralph Reitan, one of Halstead's students, contributed to the battery by researching the tests' ability to identify neurological problems. In a remarkable study, Reitan diagnosed 8,000 patients using only their test results—without meeting the patients or knowing anything about their background. This provided strong support for the battery's effectiveness. Reitan adapted the original battery by including additional tests.

The Halstead-Reitan has been researched more than any other neuropsychological test battery. Research continues to support its ability to detect impairment accu-

rately in a large range of neuropsychological functions. The eight core tests are described below, followed by a list of tests commonly used in combination with the basic battery.

Category Test

A total of 208 pictures consisting of geometric figures are presented. For each picture, individuals are asked to decide whether they are reminded of the number 1, 2, 3, or 4. They press a key that corresponds to their number of choice. If they chose correctly, a chime sounds. If they chose incorrectly, a buzzer sounds. The pictures are presented in seven subtests.

The key to this test is that one principle, or common characteristic, underlies each subtest. The numbers 1, 2, 3, and 4 represent the possible principles. If individuals are able to recognize the correct principle in one picture, they will respond correctly for the remaining pictures in that subtest. The next subtest may have the same or a different underlying principle, and individuals must again try to determine that principle using the feedback of the chime and buzzer. The last subtest contains two underlying principles. The test takes approximately one hour to complete, but individuals with severe brain damage may take as long as two hours.

The Category Test is considered the battery's most effective test for detecting brain damage, but does not help determine where the problem is occurring in the brain. The test evaluates abstraction ability, or the ability to draw specific conclusions from general information. Related abilities are solving complex and unique problems, and learning from experience. Children's versions consist of 80 items and five subtests for young children, and 168 items and 6 subtests for older children.

Scoring involves recording the number of errors. Based on traditional scoring using cutoff values (cutoff scores are scores that indicate the borderline between normal and impaired functioning), scores above 41 are considered indicative of brain impairment for ages 15 to 45. For ages 46 and older, scores above 46 indicate impairment. Reitan has suggested a cutoff of 50 or 51 errors. Recommended cutoffs also vary depending on age and education level.

Tactual Performance Test

A form board containing ten cut-out shapes, and ten wooden blocks matching those shapes are placed in front of a blindfolded individual. Individuals are then instructed to use only their dominant hand to place the blocks in their appropriate space on the form board. The same procedure is repeated using only the non-dominant hand, and then using both hands. Finally, the form board and blocks are removed, followed by the blindfold. From memory, the individual is asked to draw the form board and the shapes in their proper locations. The test usually takes anywhere from 15 to 50 minutes to complete. There is a time limit of 15 minutes for each trial, or each performance segment.

Other names for this test are the Form Board Test and the Seguin-Goddard Formboard. It evaluates sensory ability, memory for shapes and spatial location, motor functions, and the brain's ability to transfer information between its two hemispheres. In addition to simple detection of brain damage, this test also helps determine on which side of the brain damage may have occurred. For children under the age of 15, only six shapes are used.

Scoring involves recording the time to complete each of the three blindfolded trials and the total time for all trials combined (time score), the number of shapes recalled (memory score), and the number of shapes drawn in their correct locations (localization score). Generally, the trial for the non-dominant hand should be between 20 to 30 percent faster than the trial for the dominant hand, due to the benefit of practice. If the non-dominant hand is slower than the dominant hand or more than 30 percent faster than the dominant hand, brain damage is possible. However, some people without brain damage do not exhibit this typical improvement rate. Injuries of the arms, shoulders, or hands can also affect performance. Scores should be adjusted depending on education level and may vary depending on age.

Trail Making Test

This test consists of two parts. Part A is a page with 25 numbered circles randomly arranged. Individuals are instructed to draw lines between the circles in increasing sequential order until they reach the circle labeled "End." Part B is a page with circles containing the letters A through L and 13 numbered circles intermixed and randomly arranged. Individuals are instructed to connect the circles by drawing lines alternating between numbers and letters in sequential order, until they reach the circle labeled "End." If individuals make mistakes, the mistakes are quickly brought to their attention, and continue from the last correct circle. The test takes approximately five to 10 minutes to complete.

This test was originally known as Partingon's Pathways, or the Divided Attention Test, which was part of the Army Individual Test Battery. The test evaluates information processing speed, visual scanning ability, integration of visual and motor functions, letter and number recognition and sequencing, and the ability to maintain two different trains of thought. The test can be administered orally if an individual is incapable of writ-

ing. The Color Trails Test, designed for children and individuals of different cultures, uses colors instead of numbers and letters.

Scoring is simply the time to complete each part. Errors naturally increase the total time. Some have argued that the time taken to alert individuals of errors may vary depending on the person giving the test. For adults, scores above 40 seconds for Part A and 91 seconds for Part B have traditionally indicated brain impairment. Current research discourages the use of such traditional cutoffs, preferring ranges depending on age, education, and gender. For example, one study reported that for ages 15 to 19, the average time to complete Part A was 25.7 seconds and the time to complete Part B was 49.8 seconds. For ages 80 to 85, however, the average time to complete Part A was 60.7 seconds and the time to complete Part B was 152.2 seconds. This demonstrates the importance of considering other variables when scoring.

Finger Tapping Test

Individuals place their dominant hand palm down, fingers extended, with the index finger resting on a lever that is attached to a counting device. Individuals are instructed to tap their index finger as quickly as possible for ten seconds, keeping the hand and arm stationary. This trial is repeated five to 10 times, until the examiner has collected counts for five consecutive trials that are within five taps of each other. Before starting the test, individuals are given a practice session. They are also given brief rests between each 10-second trial, and one- to two-minute rests after every third trial. This entire procedure is repeated with the non-dominant hand. The test takes approximately ten minutes to complete.

This test is also called the Finger Oscillation Test. The children's version uses an electronic tapper instead of a manual one, which was difficult for children to operate. The test measures motor speed and helps determine particular areas of the brain that may be damaged. Scoring involves using the five accepted trials to calculate an average number of taps per trial for each hand. In general, the dominant hand should perform ten percent better than the non-dominant hand. Yet this is not always the case, especially with left-handed individuals. Males and younger people tend to perform better than females and older people. Interpretation should also consider education level, intelligence, **fatigue**, general weakness or lack of coordination, depression, and injuries to the shoulders, arms, or hands. This test should only be interpreted in combination with other tests in the battery.

Rhythm Test

Thirty pairs of tape-recorded, non-verbal sounds are presented. For each pair, individuals decide if the two sounds are the same or different, marking "S" or "D" respectively on their answer sheets. The pairs are grouped into three subtests. This test is also called the Seashore Rhythm Test, and is based on the Seashore Tests of Musical Ability. It evaluates auditory attention and concentration, and the ability to discriminate between non-verbal sounds. The test helps detect brain damage, but not the location of damage. Adequate hearing and visual abilities are needed to take this test. Scoring is based on number of correct items, with higher scores indicating less damage or good recovery. Scores should be interpreted along with information from other tests. Some researchers consider this test unreliable and simplistic. The children's version does not include this test.

Speech Sounds Perception Test

Sixty tape-recorded nonsense syllables containing the sound "ee" (for example, "meer" and "weem") are presented. After each syllable, individuals underline, from a set of four written syllables, the spelling that represents the syllable they heard. This test evaluates auditory attention and concentration and the ability to discriminate between verbal sounds. It provides some information regarding specific areas of brain damage, and may also indicate attention deficits or hearing loss. Scoring and interpretation are similar to that used for the Rhythm Test. The children's version contains fewer syllable choices.

Reitan-Indiana Aphasia Screening Test

Aphasia is the loss of ability to understand or use written or spoken language, due to brain damage or deterioration. In this test, individuals are presented with a variety of questions and tasks that would be easy for someone without impairment. Examples of test items include verbally naming pictures, writing the name of a picture without saying the name aloud, reading printed material of increasing length, repeating words stated by the examiner, simple arithmetic problems, drawing shapes without lifting the pencil, and placing one hand to an area on the opposite side of the body.

This test is a modification of the Halstead-Wepman Aphasia Screening Test. It evaluates language-related difficulties, right/left confusion, and non-verbal tasks. A typical scoring procedure is not used because this is a screening test; its purpose is to detect possible signs of aphasia that may require further evaluation. Subtle language deficits may not be detected.

Reitan-Klove Sensory-Perceptual Examination

This test detects whether individuals are unable to perceive stimulation on one side of the body when both sides are stimulated simultaneously. It has tactile, auditory, and visual components involving the ability to (a) specify whether touch, sound, or visible movement is occurring on the right, left, or both sides of the body; (b) recall numbers assigned to particular fingers (the examiner assigns numbers by touching each finger and stating the number with the individual's eyes closed); (c) identify numbers "written" on fingertips while eyes are closed; and (d) identify the shape of a wooden block placed in one hand by pointing to its shape on a form board with the opposite hand.

Ancillary tests

In addition to the core tests, examiners may choose to administer other tests based on the difficulties that an individual is experiencing. Tests commonly used in combination with the Halstead-Reitan battery include the Grip Strength Test, the Grooved Pegboard Test, the Reitan-Klove Lateral Dominance Examination, the Wechsler Memory Scale, the California Verbal Learning Test, the Buschke Selective Reminding Test, the Rey Auditory Verbal Memory Test, the Rey Complex Figure Test, the Test of Memory and Learning, the **Wide Range Achievement Test**, the **Minnesota Multiphasic Personality Inventory**, and the **Wechsler Adult Intelligence Scale** or **Wechsler Intelligence Scales for Children**.

Results

Interpretation of the Halstead-Reitan involves analysis of various factors:

- Overall performance on the battery. The Halstead Impairment Index (HII) and the General Neuropsychological Deficit Scale (GNDS) are commonly used to obtain an overall score. The HII is calculated by counting the total number of tests in the impaired range, and dividing that number by the total tests administered, resulting in a decimal between zero and one (0.0–0.2: normal functioning; 0.3–0.4: mild impairment; 0.5–0.7: moderate impairment; 0.8–1.0: severe impairment). The GNDS is calculated by assigning a value between zero and four to 42 variables contained in the tests, then summing those values (0–25: normal functioning; 26–40: mild impairment; 41–67: moderate impairment; 68+: severe impairment).
- Performance on individual tests. Each test must be interpreted in relation to other tests in the battery. Significantly poor performance on one test may be due to various factors. However, if a pattern of poor performance occurs on three or more tests, or if significant discrepancies occur on two or more tests, impairment is likely.
- Indications of lateralization and localization. This refers to the particular region of the brain that is damaged. Performance on sensory and motor tasks provides the necessary clues.

With the above information, a psychologist can diagnose the type of condition present, predict the course of the impairment (staying the same, getting better, or getting worse), and make recommendations regarding treatment, care, or rehabilitation.

See also Assessment and Diagnosis; Brain; Dementia; Executive function; Luria-Nebraska Inventory; Mini-Mental State Exam; Neuropsychological Testing

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Division of Clinical Neuropsychology. Division 40, American Psychological Association, 750 First Street, NE, Washington DC 20002-4242. (202) 336-6013. <http://www.div40.org>.

International Neuropsychology Society, 700 Ackerman Road, Suite 550, Columbus, Ohio 43202. (614) 263-4200. <http://www.osu.edu/ins>.

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Hamilton Anxiety Scale

Definition

The Hamilton Anxiety Scale (HAS or HAMA) is a 14-item test measuring the severity of anxiety symptoms. It is also sometimes called the Hamilton Anxiety Rating Scale (HARS).

Purpose

The HAS is used to assess the severity of anxiety symptoms present in children and adults. It is also used as an outcome measure when assessing the impact of anti-anxiety medications, therapies, and treatments and is a standard measure of anxiety used in evaluations of psychotropic drugs. The HAS can be administered prior to medication being started and then again during follow-up visits, so that medication dosage can be changed in part based on the patient's test score.

The HAS was developed by Max Hamilton in 1959. It provides measures of overall anxiety, psychic anxiety (mental agitation and psychological distress), and somatic anxiety (physical complaints related to anxiety). Hamilton developed the HAS to be appropriate for adults and children; although it is most often used for younger adults, there has been support for the test's use with older adults as well. Hamilton also developed the widely used **Hamilton Depression Scale** (HDS) to measure symptoms of depression.

Hamilton developed the scale by utilizing the statistical technique of factor analysis. Using this method, he was able to generate a set of symptoms related to anxiety and further determine which symptoms were related to psychic anxiety and which were related to somatic anxiety.

KEY TERMS

Factor analysis—A statistical method for summarizing relationships between variables. For the HAS, factor analysis was utilized to determine the specific sets of symptoms relating to overall anxiety, somatic anxiety, and psychic anxiety.

Psychotropic medication—Medication that has an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

Reliability—The ability of a test to yield consistent, repeatable results.

Validity—The ability of a test to measure accurately what it claims to measure.

Precautions

The test has been criticized on the grounds that it does not always discriminate between people with anxiety symptoms and those with depressive symptoms (people with depression also score fairly high on the HAS).

Because the HAS is an interviewer-administered and rated measure, there is some subjectivity when it comes to interpretation and scoring. Interviewer bias can impact the results. For this reason, some people prefer self-report measures where scores are completely based on the interviewee's responses.

Description

The HAS is administered by an interviewer who asks a semi-structured series of questions related to symptoms of anxiety. The interviewer then rates the individuals on a five-point scale for each of the 14 items. Seven of the items specifically address psychic anxiety and the remaining seven items address somatic anxiety. For example, the third item specifically addresses fears related to anxiety, the fifth item addresses **insomnia** and sleeping difficulties related to anxiety, and the tenth item addresses respiratory symptoms related to anxiety.

According to Hamilton, examples of psychic symptoms elicited by the HAS interview include a general anxious mood, heightened fears, feelings of tension, and difficulty concentrating. Examples of somatic symptoms include muscular pain, feelings of weakness, cardiovascular problems, and restlessness.

Results

For the 14 items, the values on the scale range from zero to four: zero means that there is no anxiety, one indicates mild anxiety, two indicates moderate anxiety, three indicates severe anxiety, and four indicates very severe or grossly disabling anxiety. The total anxiety score ranges from 0 to 56. The seven psychic anxiety items elicit a psychic anxiety score that ranges from 0 to 28. The remaining seven items yield a somatic anxiety score that also ranges from 0 to 28.

One reason that the HAS is widely used is that reliability studies have shown that it measures anxiety symptoms in a fairly consistent way. The measure's validity has also been supported by research.

Studies have shown that individuals with anxiety disorders score fairly high on the HAS. For example, persons with **generalized anxiety disorder** and **panic disorder** tend to have a total anxiety score of above 20 on the HAS. On the other hand, people with no disorder or **diagnosis** score very low on the HAS.

While there is a tendency for depressed people to also score high on the HAS, some researchers have suggested that anxiety and depression are so closely linked that people can easily score high on measures of both types of symptoms.

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Ali Fahmy, Ph.D.

Hamilton Depression Scale

Definition

The Hamilton Depression Scale (HDS or HAMD) is a test measuring the severity of depressive symptoms in individuals, often those who have already been diagnosed as having a depressive disorder. It is sometimes known as the Hamilton Rating Scale for Depression (HRSD) or the Hamilton Depression Rating Scale (HDRS).

KEY TERMS

Hypochondriasis—A mental condition in which the affected person perceives illness or symptoms of illness when none exist.

Psychomotor retardation—Slowdown in motor activity directly proceeding from mental activity.

Psychotropic medication—Medication that has an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

Reliability—The ability of a test to yield consistent, repeatable results.

Validity—The ability of a test to measure accurately what it claims to measure.

Purpose

The HDS is used to assess the severity of depressive symptoms present in both children and adults. It is often used as an outcome measure of depression in evaluations of antidepressant psychotropic medications and is a standard measure of depression used in research of the effectiveness of depression therapies and treatments. It can be administered prior to the start of medication and then again during follow-up visits, so that medication dosage can be changed in part based on the patient's test score. The HDS often used as the standard against which other measures of depression are validated.

The HDS was developed by Max Hamilton in 1960 as a measure of depressive symptoms that could be used in conjunction with clinical interviews with depressed patients. It was later revised in 1967. Hamilton also designed the Hamilton Depression Inventory (HDI), a self-report measure consistent with his theoretical formulation of depression in the HDS, and the **Hamilton Anxiety Scale** (HAS), an interviewer-rated test measuring the severity of anxiety symptoms.

Precautions

Some symptoms related to depression, such as self-esteem and self-deprecation, are not explicitly included in the HDS items. Also, because anxiety is specifically asked about on the HDS, it is not always possible to sep-

arate symptoms related to anxiety from symptoms related to depression.

Because the HDS is an interviewer-administered and rated measure, there is some subjectivity when it comes to interpretation and scoring. Interviewer bias can impact the results. For this reason, some people prefer self-report measures where scores are completely based on the interviewee's responses.

Description

Depending on the version used, there are either 17 or 21 items for which an interviewer provides ratings. Besides the interview with the depressed patient, other information can be utilized in formulating ratings, such as information gathered from family, friends, and patient records. Hamilton stressed that the interview process be easygoing and informal and that there are no specific questions that must be asked.

The 17-item version of the HDS is more commonly used than the 21-item version, which contains four additional items measuring symptoms related to depression, such as **paranoia** and **obsession**, rather than the severity of depressive symptoms themselves.

Examples of items for which interviewers must give ratings include overall depression, guilt, **suicide**, **insomnia**, problems related to work, psychomotor retardation, agitation, anxiety, gastrointestinal and other physical symptoms, loss of libido (sex drive), **hypochondriasis**, loss of insight, and loss of weight. For the overall rating of depression, for example, Hamilton believed one should look for feelings of hopelessness and gloominess, pessimism regarding the future, and a tendency to cry. For the rating of suicide, an interviewer should look for suicidal ideas and thoughts, as well as information regarding suicide attempts.

Results

In the 17-item version, nine of the items are scored on a five-point scale, ranging from zero to four. A score of zero represents an absence of the depressive symptom being measured, a score of one indicates doubt concerning the presence of the symptom, a score of two indicates mild symptoms, a score of three indicates moderate symptoms, and a score of four represents the presence of severe symptoms. The remaining eight items are scored on a three-point scale, from zero to two, with zero representing absence of symptom, one indicating doubt that the symptom is present, and two representing clear presence of symptoms.

For the 17-item version, scores can range from 0 to 54. One formulation suggests that scores between 0 and 6 indicate a normal person with regard to depression, scores between 7 and 17 indicate mild depression, scores between 18 and 24 indicate moderate depression, and scores over 24 indicate severe depression.

There has been evidence to support the reliability and validity of the HDS. The scale correlates highly with other clinician-rated and self-report measures of depression.

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Ali Fahmy, Ph.D.

Hare Psychopathy Checklist

Definition

The Hare Psychopathy Checklist-Revised (PCL-R) is a diagnostic tool used to rate a person's psychopathic or antisocial tendencies. People who are psychopathic prey ruthlessly on others using charm, deceit, violence or other methods that allow them to get with they want. The symptoms of psychopathy include: lack of a conscience or sense of guilt, lack of empathy, egocentricity, pathological lying, repeated violations of social norms, disregard for the law, shallow emotions, and a history of victimizing others.

Originally designed to assess people accused or convicted of crimes, the PCL-R consists of a 20-item symptom rating scale that allows qualified examiners to compare a subject's degree of psychopathy with that of a prototypical psychopath. It is accepted by many in the field as the best method for determining the presence and extent of psychopathy in a person.

The Hare checklist is still used to diagnose members of the original population for which it was developed—adult males in prisons, criminal psychiatric hospitals, and awaiting psychiatric evaluations or trial in other correctional and detention facilities. Recent experience suggests that the PCL-R may also be used effectively to diagnose sex offenders as well as female and adolescent offenders.

Purpose

The PCL-R is used for diagnosing psychopathy in individuals for clinical, legal or research purposes. Developed in the early 1990s, the test was originally designed to identify the degree of a person's psychopathic tendencies. Because psychopaths, however, are often repeat offenders who commit sexual assaults or other violent crimes again and again, the PCL-R is now finding use in the courtroom and in institutions as an indicator of the potential risk posed by subjects or prisoners. The results of the examination have been used in forensic settings as a factor in deciding the length and type of prison sentences and the treatment subjects should or should not receive.

Precautions

Obviously, diagnosing someone as a psychopath is a very serious step. It has important implications for a person and for his or her associates in family, clinical and forensic settings. Therefore, the test must be administered by professionals who have been specifically trained in its use and who have a wide-ranging and up-to-date familiarity with studies of psychopathy.

Professionals who administer the diagnostic examination should have advanced degrees (M.D., Ph.D., or D.Ed.) in a medical, behavioral or social science field; and registered with a reputable organization that oversees psychiatric or psychological testing and diagnostic procedures. Other recommendations include experience working with convicted or accused criminals or several years of some other related on-the-job training. Because the results are used so often in legal cases, those who administer it should be qualified to serve as expert witnesses in the courtroom. It is also a good idea, if possible, for two experts to test a subject independently with the PCL-R. The final rating would then be determined by averaging their scores.

Many studies conducted in North America and Europe attest to the value of the PCL-R for evaluating a person's degree of psychopathic traits and, in many cases, for predicting the likelihood of future violent behavior. Some critics, however, are more skeptical about its value.

Description

The Hare PCL-R contains two parts, a semi-structured interview and a review of the subject's file records and history. During the evaluation, the clinician scores 20 items that measure central elements of the psychopathic character. The items cover the nature of the subject's interpersonal relationships; his or her affective or emotional involvement; responses to other people and to situations; evidence of social deviance; and lifestyle. The material thus covers two key aspects that help define the psychopath: selfish and unfeeling victimization of other people, and an unstable and antisocial lifestyle.

The twenty traits assessed by the PCL-R score are:

- glib and superficial charm
- grandiose (exaggeratedly high) estimation of self
- need for stimulation
- pathological lying
- cunning and manipulativeness
- lack of remorse or guilt
- shallow **affect** (superficial emotional responsiveness)
- callousness and lack of empathy
- parasitic lifestyle
- poor behavioral controls

KEY TERMS

Affect—The expression of emotion displayed to others through facial expressions, hand gestures, tone of voice, etc. Types of affect include: flat (inanimate, no expression), blunted (minimally responsive), inappropriate (incongruous expressions of emotion relative to the content of a conversation), and labile (sudden and abrupt changes in type and intensity of emotion).

Egocentricity—Self-centeredness.

Forensic—Pertaining to courtroom procedure or evidence used in courts of law.

Grandiose—Having an exaggerated belief in one's importance or status. In some people, grandiosity may be so extreme as to be delusional.

Psychopath—A person who ruthlessly preys on others, using charm, deceit, violence or other methods that allows him or her to get with they want. Another word that is sometimes used for psychopath is sociopath.

Psychopathy—A psychological syndrome that includes lack of a conscience or sense of guilt, lack of empathy, egocentricity, pathological lying, repeated violations of social norms, disregard of the law, shallow emotions and a history of victimizing others.

- sexual promiscuity
- early behavior problems
- lack of realistic long-term goals
- impulsivity
- irresponsibility
- failure to accept responsibility for own actions
- many short-term marital relationships
- juvenile delinquency
- revocation of conditional release
- criminal versatility

The interview portion of the evaluation covers the subject's background, including such items as work and educational history; marital and family status; and criminal background. Because psychopaths lie frequently and easily, the information they provide must be confirmed by a review of the documents in the subject's case history.

Results

When properly completed by a qualified professional, the PCL-R provides a total score that indicates how closely the test subject matches the "perfect" score that a classic or prototypical psychopath would rate. Each of the twenty items is given a score of 0, 1, or 2 based on how well it applies to the subject being tested. A prototypical psychopath would receive a maximum score of 40, while someone with absolutely no psychopathic traits or tendencies would receive a score of zero. A score of 30 or above qualifies a person for a **diagnosis** of psychopathy. People with no criminal backgrounds normally score around 5. Many non-psychopathic criminal offenders score around 22.

See also Antisocial personality disorder; Sexual sadism

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HCR-20 *see* **Historical, Clinical, Risk Management-20**

Health maintenance organization *see* **Managed care**

Historical, Clinical, Risk Management-20

Definition

The Historical, Clinical, Risk Management-20 (HCR-20) is an assessment tool that helps mental health professionals estimate a person's probability of violence.

Purpose

The HCR-20's results help mental health professionals determine best treatment and management strategies for potentially violent, mentally disordered individuals, including parolees, forensic mental health patients, and others. For example, if an individual is standing trial for a violent offense, a judge might order that assessments (such as the HCR-20, as well as others) be performed. The results of the evaluation could be used to determine the person's future potential for violence, how the court should proceed, and which kind of facility the person might require.

Precautions

A professional trained in conducting individual assessments and in the study of violence should administer the HCR-20. The test administrator should have a background in using assessment tests or should consult a mental health professional. The HCR-20 is not intended to be a stand-alone measure, and it does not cover all risk factors. When possible, the test administrator should use supplemental test measures and investigate any unique patterns of violence and its triggers in the person's history. The HCR-20 is not meant to be administered just once; the nature of risk assessment requires ongoing reassessment as circumstances change. Final interpretation of HCR-20 results should be in the context of several fac-

tors, including the reason for the person's test referral, base rates of violence in populations with similar characteristics, and assessment of future risks in the person's environment.

Description

The HCR-20 is an assessment tool. It consists of a list of 20 probing questions about the person being evaluated for violence. The clinician gathers qualitative information about the person being assessed, guided by the HCR-20, and the results are used to make treatment decisions.

The HCR-20 provides significantly improved valid predictions over previous testing methods. Earlier testing methods tended to be more subjective, less well-focused, and based on the loosely supported judgment of test administrators, or on comparing characteristics of the person being tested with base rates of violent behavior in populations with similar characteristics. The HCR-20 extends the methods of earlier tests and supplements them with a review of dynamic variables, such as **stress** and lack of personal support— both factors important to the person's future adjustment. This review adds to the accuracy of the HCR-20, and increases its practicality.

The HCR-20 consists of three main areas: historical, clinical, and risk management. The HCR-20 domains are coded with a rating of 0 (not present), 1 (possible/less serious), or 3 (definite/serious).

Historical area

To rate historical areas, the test administrator must do an exhaustive review of background documents, interview people who know the person being assessed, and complete the **Hare Psychopathy Checklist**, a useful instrument in its own right. The historical area is considered by many to anchor the instrument. It includes 10 domains:

- previous violence
- young age at first violent incident
- relationship instability
- employment problems
- substance use problems
- major mental illness, such as **schizophrenia** or **bipolar disorder**
- psychopathy, which can be defined as personality traits that deviate from social norms, such as manipulating and exploiting others for personal gain
- early maladjustment, or exposure to family and social disruptions during childhood that lead to coping problems (could be **abuse** or divorce, for example)

KEY TERMS

Decision-makers—In the context of this entry, the term refers to prison or court officials, treatment facility administrators, or family members.

Risk assessment—The process of gathering and interpreting data useful in estimating the probability that an individual will demonstrate violence.

Risk management plan—Using the results of a risk assessment to tailor intervention strategies intended to reduce the probability that an individual will demonstrate violence.

- personality disorder, such as paranoia
- failure to respond to clinical supervision or treatment in the past—may be related to noncompliance to treatment, such as refusing to take medications or attend therapy sessions

Clinical area

The rating of the clinical area requires a clinical interview between the person being assessed and the mental health professional. The professional will also use his or her judgment, as well. The clinical area consists of five domains:

- Lack of insight, or difficulty understanding cause and effect. For example, people with poor insight might not understand why they do what they do and why their actions matter.
- Negative attitudes.
- Active symptoms of major mental illness.
- Impulsivity.
- Unresponsiveness to treatment.

Risk management

The third area, risk management, includes five domains:

- the person's plans lack feasibility
- exposure to destabilizers, which means that family or social supports are missing, or that alcohol and drugs are available
- lack of personal support
- refusal to attend counseling sessions or take medications
- stress

Results

The HCR-20 does not allow for a definite prediction of violence. Predictions based on the HCR-20 are estimates of the likelihood of violence, and should be presented in terms of low, moderate, or high probability of violence. Probability levels should be considered conditional, given short- and long-term time frames, and should be considered in relation to relevant factors the individual may encounter. These factors include situations and states of being that may dispose a person to violence or help insulate them against it. Consideration of such factors can aid in reporting the type and extent of risk presented by a person and in selecting **intervention** strategies intended to reduce the probability that an individual will demonstrate violence. These strategies when taken as a whole are called a risk management plan.

Ultimately, HCR-20 results are intended to provide information for decision-makers, so that criminal and mental health-related decisions can be based on the best available estimates of risk of violence.

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Histrionic personality disorder

Definition

Histrionic personality disorder, often abbreviated as HPD, is a type of personality disorder in which the affected individual displays an enduring pattern of attention-seeking and excessively dramatic behaviors beginning in early adulthood and present across a broad range of situations. Individuals with HPD are highly emotional, charming, energetic, manipulative, seductive, impulsive, erratic, and demanding.

Mental health professionals use the ***Diagnostic and Statistical Manual of Mental Disorders*** (the *DSM*) to diagnose mental disorders. The 2000 edition of this manual (the fourth edition text revision, also called the *DSM-IV-TR*) classifies HPD as a personality disorder. More specifically, HPD is classified as a Cluster B (dramatic, emotional, or erratic) personality disorder. The **personality disorders** which comprise Cluster B include histrionic, antisocial, borderline, and narcissistic.

Description

HPD has a unique position among the personality disorders in that it is the only personality disorder explicitly connected to a patient's physical appearance. Researchers have found that HPD appears primarily in men and women with above-average physical appearances. Some research has suggested that the connection between HPD and physical appearance holds for women rather than for men. Both women and men with HPD express a strong need to be the center of attention. Individuals with HPD exaggerate, throw temper tantrums, and cry if they are not the center of attention. Patients with HPD are naive, gullible, have a low frustration threshold, and strong dependency needs.

Cognitive style can be defined as a way in which an individual works with and solves cognitive tasks such as reasoning, learning, thinking, understanding, making decisions, and using memory. The cognitive style of individuals with HPD is superficial and lacks detail. In their interpersonal relationships, individuals with HPD use dramatization with a goal of impressing others. The enduring pattern of their insincere and stormy relationships leads to impairment in social and occupational areas.

Causes and symptoms

Causes

There is a lack of research on the causes of HPD. Even though the causes for the disorder are not defini-

KEY TERMS

Behavioral contracts—A behavioral contract is a written agreement that defines the behaviors to be performed and the consequences of the specified behaviors.

Biosocial—A biosocial model in psychology asserts that social and biological factors contribute towards the development of personality.

Catecholamine—A group of neurotransmitters synthesized from the amino acid tyrosine and released by the hypothalamic-pituitary-adrenal system in the brain in response to acute stress. The catecholamines include dopamine, serotonin, norepinephrine, and epinephrine.

Cognitive style—A way in which an individual works with and performs cognitive tasks such as reasoning, learning, thinking, understanding, making decisions, and using memory.

Differential diagnosis—The process of distinguishing one disorder from other, similar disorders.

Disingenuous—Insincere, deceitful, dishonest.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Etiology—The cause or origin of a disease or disorder. The word is also used to refer to the study of the causes of disease.

Histrionic—Theatrical.

Identity diffusion—A character formation that is scattered or spread around rather than an identity that becomes solidified or consolidated.

Noradrenergic—Acts similarly to norepinephrine or noradrenaline.

Oral phase—The first of Freud's psychosexual stages of development in which satisfaction is focused on the mouth and lips. During this stage sucking and eating are the primary means of gratification.

Personality disorder—A personality disorder is a maladaptive pattern of behavior, affect, and/or cognitive style displayed in a broad range of settings. The pattern deviates from the accepted norms of the individual's culture and can occur over a lifetime.

Response cost—A behavioral technique that involves removing a stimulus from an individual's environment so that the response that directly precedes the removal is weakened. In a token economy system, response cost is a form of punishment involving loss of tokens due to inappropriate behavior, which consequently results in decreased ability to purchase back-up reinforcers.

Somatic—Relating to the body or to the physical.

Thematic dream material—Psychoanalysts use the technique of dream interpretation to offer patients insight into their unconscious conflicts. The dreams of patients include themes, notions, or underlying ideas about specific objects, situations, or issues. When patients begin to understand the content or themes of their dreams, they may gain insight into their unconscious motives.

tively known, it is thought that HPD may be caused by biological, developmental, cognitive, and social factors.

NEUROCHEMICAL/PHYSIOLOGICAL CAUSES. Studies show that patients with HPD have highly responsive noradrenergic systems, the mechanisms surrounding the release of a neurotransmitter called norepinephrine. **Neurotransmitters** are chemicals that communicate impulses from one nerve cell to another in the **brain**, and these impulses dictate behavior. The tendency towards an excessively emotional reaction to rejection, common among patients with HPD, may be attributed to a malfunction in a group of neurotransmitters called catecholamines. (Norepinephrine belongs to this group of neurotransmitters.)

DEVELOPMENTAL CAUSES. Psychoanalytic theory, developed by Freud, outlines a series of psychosexual stages of development through which each individual passes. These stages determine an individual's later psychological development as an adult. Early psychoanalysts proposed that the genital phase, Freud's fifth or last stage of psychosexual development, is a determinant of HPD. Later psychoanalysts considered the oral phase, Freud's first stage of psychosexual development, to be a more important determinant of HPD. Most psychoanalysts agree that a traumatic childhood contributes towards the development of HPD. Some theorists suggest that the more severe forms of HPD derive from disapproval in the early mother-child relationship.

Another component of Freud's theory is the defense mechanism. Defense mechanisms are sets of systematic, unconscious methods that people develop to cope with conflict and to reduce anxiety. According to Freud's theory, all people use defense mechanisms, but different people use different types of defense mechanisms. Individuals with HPD differ in the severity of the maladaptive defense mechanisms they use. Patients with more severe cases of HPD may utilize the defense mechanisms of repression, **denial**, and dissociation.

- Repression. Repression is the most basic defense mechanism. When patients' thoughts produce anxiety or are unacceptable to them, they use repression to bar the unacceptable thoughts or impulses from consciousness.
- Denial. Patients who use denial may say that a prior problem no longer exists, suggesting that their competence has increased; however, others may note that there is no change in the patients' behaviors.
- Dissociation. When patients with HPD use the defense mechanism of dissociation, they may display two or more personalities. These two or more personalities exist in one individual without integration. Patients with less severe cases of HPD tend to employ displacement and rationalization as defenses.
- Displacement occurs when a patient shifts an **affect** from one idea to another. For example, a man with HPD may feel angry at work because the boss did not consider him to be the center of attention. The patient may displace his anger onto his wife rather than become angry at his boss.
- Rationalization occurs when individuals explain their behaviors so that they appear to be acceptable to others.

BIOSOCIAL LEARNING CAUSES. A biosocial model in psychology asserts that social and biological factors contribute to the development of personality. Biosocial learning models of HPD suggest that individuals may acquire HPD from inconsistent interpersonal **reinforcement** offered by parents. Proponents of biosocial learning models indicate that individuals with HPD have learned to get what they want from others by drawing attention to themselves.

SOCIOCULTURAL CAUSES. Studies of specific cultures with high rates of HPD suggest social and cultural causes of HPD. For example, some researchers would expect to find this disorder more often among cultures that tend to value uninhibited displays of emotion.

PERSONAL VARIABLES. Researchers have found some connections between the age of individuals with HPD and the behavior displayed by these individuals. The symptoms of HPD are long-lasting; however, histrionic character traits that are exhibited may change with age. For example, research suggests that seductiveness may be employed more often by a young adult than by an older one. To impress others, older adults with HPD may shift their strategy from sexual seductiveness to a paternal or maternal seductiveness. Some histrionic symptoms such as attention-seeking, however, may become more apparent as an individual with HPD ages.

Symptoms

DSM-IV-TR lists eight symptoms that form the diagnostic criteria for HPD:

- Center of attention: Patients with HPD experience discomfort when they are not the center of attention.
- Sexually seductive: Patients with HPD displays inappropriate sexually seductive or provocative behaviors towards others.
- Shifting emotions: The expression of emotions of patients with HPD tends to be shallow and to shift rapidly.
- Physical appearance: Individuals with HPD consistently employ physical appearance to gain attention for themselves.
- Speech style: The speech style of patients with HPD lacks detail. Individuals with HPD tend to generalize, and when these individuals speak, they aim to please and impress.
- Dramatic behaviors: Patients with HPD display self-dramatization and exaggerate their emotions.
- Suggestibility: Other individuals or circumstances can easily influence patients with HPD.
- Overestimation of intimacy: Patients with HPD overestimate the level of intimacy in a relationship.

Demographics

General United States population

The prevalence of HPD in the general population is estimated to be approximately 2%-3%.

High-risk populations

Individuals who have experienced pervasive trauma during childhood have been shown to be at a greater risk for developing HPD as well as for developing other personality disorders.

Cross-cultural issues

HPD may be diagnosed more frequently in Hispanic and Latin-American cultures and less frequently in Asian

cultures. Further research is needed on the effects of culture upon the symptoms of HPD.

Gender issues

Clinicians tend to diagnose HPD more frequently in females; however, when structured assessments are used to diagnose HPD, clinicians report approximately equal prevalence rates for males and females. In considering the prevalence of HPD, it is important to recognize that gender role stereotypes may influence the behavioral display of HPD and that women and men may display HPD symptoms differently.

Diagnosis

The **diagnosis** of HPD is complicated because it may seem like many other disorders, and also because it commonly occurs simultaneously with other personality disorders. The 1994 version of the *DSM* introduced the criterion of suggestibility and the criterion of overestimation of intimacy in relationships to further refine the diagnostic criteria set of HPD, so that it could be more easily recognizable. Prior to assigning a diagnosis of HPD, clinicians need to evaluate whether the traits evident of HPD cause significant distress. (The *DSM* requires that the symptoms cause significant distress in order to be considered a disorder.) The diagnosis of HPD is frequently made on the basis of an individual's history and results from unstructured and semi-structured interviews.

Time of onset/symptom duration

Some psychoanalysts propose that the determinants of HPD date back as early as early childhood. The pattern of craving attention and displaying dramatic behavior for an individual with HPD begins by early adulthood. Symptoms can last a lifetime, but may decrease or change their form with age.

Individual variations in HPD

Some classification systems distinguish between different types of individuals with HPD: patients with appeasing HPD and patients with disingenuous HPD. Individuals with appeasing HPD have personalities with histrionic, dependent, and obsessive-compulsive components. Individuals with disingenuous HPD possess personality traits that are classified as histrionic and antisocial. Studies have shown that relationships exist between somatic behaviors and women with HPD and between antisocial behaviors and men with HPD.

Dual diagnoses

HPD has been associated with alcoholism and with higher rates of **somatization disorder**, **conversion disorder**, and **major depressive disorder**. Personality disorders such as borderline, narcissistic, antisocial, and dependent can occur with HPD.

Differential diagnosis

Differential diagnosis is the process of distinguishing one mental disorder from other similar disorders. For example, at times, it is difficult to distinguish between HPD and **borderline personality disorder**. **Suicide** attempts, identity diffusion, and numerous chaotic relationships occur less frequently, however, with a diagnosis of HPD. Another example of overlap can occur between HPD and **dependent personality disorder**. Patients with HPD and dependent personality disorder share high dependency needs, but only dependent personality disorder is linked to high levels of self-attributed dependency needs. Whereas patients with HPD tend to be active and seductive, individuals with dependent personality disorder tend to be subservient in their demeanor.

Psychological measures

In addition to the interviews mentioned previously, self-report inventories and projective tests can also be used to help the clinician diagnose HPD. The Minnesota Multiphasic Personality Inventory-2 (MMPI-2) and the Millon Clinical Mutiaxial Inventory-III (MCMI-III) are self-report inventories with a lot of empirical support. Results of intelligence examinations for individuals with HPD may indicate a lack of perseverance on arithmetic or on tasks that require concentration.

Treatments

Psychodynamic therapy

HPD, like other personality disorders, may require several years of therapy and may affect individuals throughout their lives. Some professionals believe that psychoanalytic therapy is a treatment of choice for HPD because it assists patients to become aware of their own feelings. Long-term psychodynamic therapy needs to target the underlying conflicts of individuals with HPD and to assist patients in decreasing their emotional reactivity. Therapists work with thematic dream material related to intimacy and recall. Individuals with HPD may have difficulty recalling because of their tendency to repress material.

Cognitive-behavioral therapy

Cognitive therapy is a treatment directed at reducing the dysfunctional thoughts of individuals with HPD. Such thoughts include themes about not being able to take care of oneself. Cognitive therapy for HPD focuses on a shift from global, suggestible thinking to a more methodical, systematic, and structured focus on problems. Cognitive-behavioral training in relaxation for an individual with HPD emphasizes challenging automatic thoughts about inferiority and not being able to handle one's life. **Cognitive-behavioral therapy** teaches individuals with HPD to identify automatic thoughts, to work on impulsive behavior, and to develop better problem-solving skills. Behavioral therapists employ **assertiveness training** to assist individuals with HPD to learn to cope using their own resources. Behavioral therapists use response cost to decrease the excessively dramatic behaviors of these individuals. Response cost is a behavioral technique that involves removing a stimulus from an individual's environment so that the response that directly precedes the removal is weakened. Behavioral therapy for HPD includes techniques such as **modeling** and behavioral rehearsal to teach patients about the effect of their theatrical behavior on others in a work setting.

Group therapy

Group therapy is suggested to assist individuals with HPD to work on interpersonal relationships. Psychodrama techniques or group role play can assist individuals with HPD to practice problems at work and to learn to decrease the display of excessively dramatic behaviors. Using role-playing, individuals with HPD can explore interpersonal relationships and outcomes to understand better the process associated with different scenarios. Group therapists need to monitor the group because individuals with HPD tend to take over and dominate others.

Family therapy

To teach assertion rather than avoidance of conflict, family therapists need to direct individuals with HPD to speak directly to other family members. **Family therapy** can support family members to meet their own needs without supporting the histrionic behavior of the individual with HPD who uses dramatic crises to keep the family closely connected. Family therapists employ behavioral contracts to support assertive behaviors rather than temper tantrums.

Medications

Pharmacotherapy is not a treatment of choice for individuals with HPD unless HPD occurs with another disorder. For example, if HPD occurs with depression, antidepressants may be prescribed. Medication needs to be monitored for abuse.

Alternative therapies

Meditation has been used to assist extroverted patients with HPD to relax and to focus on their own inner feelings. Some therapists employ hypnosis to assist individuals with HPD to relax when they experience a fast heart rate or palpitations during an expression of excessively dramatic, emotional, and excitable behavior.

Prognosis

The personality characteristics of individuals with HPD are long-lasting. Individuals with HPD utilize medical services frequently, but they usually do not stay in psychotherapeutic treatment long enough to make changes. They tend to set vague goals and to move toward something more exciting. Treatment for HPD can take a minimum of one to three years and tends to take longer than treatment for disorders that are not personality disorders, such as anxiety disorders or mood disorders.

As individuals with HPD age, they display fewer symptoms. Some research suggests that the difference between older and younger individuals may be attributed to the fact that older individuals have less energy.

Research indicates that a relationship exists between poor treatment outcomes and premature termination from treatment for individuals with Cluster B personality disorders. Some researchers suggest that studies that link HPD to continuation in treatment need to consider the connection between overestimates of intimacy and premature termination from therapy.

Prevention

Early diagnosis can assist patients and family members to recognize the pervasive pattern of reactive emotion among individuals with HPD. Educating people, particularly mental health professionals, about the enduring character traits of individuals with HPD may prevent some cases of mild histrionic behavior from developing into full-blown cases of maladaptive HPD. Further research in prevention needs to investigate the relationship between variables such as age, gender, culture, and ethnicity and HPD.

See also Minnesota Multiphasic Personality Inventory

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ORGANIZATIONS

American Psychiatric Association. 1400 K Street NW, Washington D.C. 20005. <http://www.psych.org>.

American Psychological Association. 750 First Street, NE, Washington, D.C. 20002-4242. (202) 336-5500. <http://www.apa.org>.

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HMO *see* **Managed care**

Homelessness

Definition

In the United States, definitions of homelessness help determine who is able to receive shelter and assistance from certain health and social service providers. The Stewart McKinney Homeless Assistance Act of 1987 defines a homeless person as any individual who lacks housing, including an individual whose primary residence during the night is a supervised public or private facility that provides temporary living accommodations or an individual who is a resident in transitional housing. More specifically, this means an individual who lacks fixed, regular, and adequate nighttime residence, and an individual who has a primary nighttime residence that is either (i) a supervised temporary living shelter (including transitional housing for the mentally ill), (ii) an institution that provides temporary residence for individuals intended to be institutionalized, or (iii) a place not designed for or ordinarily used as a regular sleeping accommodation for human beings.

Difficulties in estimating numbers of people who experience homelessness

Methods for estimating the size of the homeless population are evolving and sometimes contested, and are complicated by varying definitions of homelessness. The U.S. Census, while attempting to identify the number of people who are homeless and who use particular types of homeless services, has complex and service-based definitions of homelessness. It also has recognized its limited abilities to define and enumerate the homeless (it is after all a national household survey). In 2000, the Census Bureau defined the Emergency and Transitional Shelter (E&TS) population by surveying people who use a sample of homeless services. They counted homeless people in emergency shelters for adults, runaway youth shelters, shelters for abused women and their children, soup kitchens, and certain outdoor locations. Technically, however, homeless people may reside in "E&TS," in foster care, in jails and prisons, in **group homes**, in worker dorms, non-sheltered in the outdoors, doubled up with families or friends, or temporarily in Census-recognized households. According to the National Coalition for the Homeless, while counting the number of people who use services such as shelters and soup kitchens can yield important information about services, applying these numbers toward estimating numbers of homeless people can result in underestimates of homelessness.

Further complicating the issue of counting homeless people is the fact that, in many cases, homelessness is a temporary condition. Because of this fact, some researchers advocate a method of counting all the people who are homeless in a given week or, alternatively, over a given period of time. However, the numbers of people who find housing and the number of people who newly find themselves homeless fluctuates over time periods. In contrast, people with mental illness or substance abuse problems tend to be chronically without homes—it is dif-

KEY TERMS

Deinstitutionalization—The process of moving people out of mental hospitals into treatment programs or halfway houses in local communities. With this movement, the responsibility for care shifted from large (often governmental) agencies to families and community organizations.

ficult for many of these people to find permanent housing. Thus, while these two time-oriented methods of counting homeless can be useful, they too have statistical problems—they can overestimate the numbers of homeless people.

Homelessness is an acute version of residential instability, which can be compared or contrasted with definitions of poverty. Thus the term *homeless* may also be extended to include people who have nowhere to go and are at imminent risk of losing housing through eviction or institutional discharge. Some definitions of homelessness further specify the duration of time without regular and adequate residence, or the types of temporary living shelter or institutions that are not fixed residences. People who live without alternatives in overcrowded or unhealthy housing conditions may be at risk of homelessness. Worldwide, national and cultural groups may have variable and often different definitions of homelessness, different terms for the condition of being without housing, and different definitions of adequate housing. For all of these reasons related both to methods of counting and varying definitions, estimating the size of the homeless population is extremely difficult.

Demographics

Census estimates of the size and composition of the homeless population are difficult to create, for reasons described above. The Emergency and Transitional Shelter (ET&S) Population count in the U.S. in 1990 was 178,638. It remained relatively stable in 2000 at 170,706. However, this figure does not include homeless adults not using ET&S services, sampling error, or some groups of homeless people not enumerated in the ET&S count. The ET&S population in 2000 was 61% male and 74% adult. Among the 26% who were youth, 51% were male. For adults, the population was 65% male, 41% were white, 40% were African-American, 20% were Latino of any race, 2% were Asian, 2% were Native American, and 9% were one other race alone.

Another estimate of homelessness is a 1988 count of homeless people that occurred over one week, counting homeless people congregated on the street, in soup kitchens, and in shelters. That estimate was 500,000 to 600,000 people.

The large variation between these estimates illustrates that, as the National Coalition for the Homeless states, "By its very nature, homelessness is impossible to measure with 100% accuracy."

Incidence of homelessness and associated diseases and conditions

Homeless adults are poor and have high rates of unmet need for health care. This is in part because poverty is associated with higher risk and rates of illness, particularly mental illnesses including substance abuse. Homeless people experience disproportionate rates and symptoms of mental health disorders, including substance abuse disorders and dual diagnoses. For these reasons, large portions of federally funded homeless services are medical services, and homeless people are often viewed according to their present or past medical classifications.

Studies researching the incidence, distribution, and control of a disease in a population (known as epidemiological studies) find that between one-third and one-half of homeless people have mental health disorders and approximately two-thirds have either a mental health or substance use disorder. People with severe mental illness are likewise more likely to become homeless, particularly when the disorder is co-morbid (co-occurs) with substance abuse. For this reason, changes in rates of homelessness are often associated with changes in mental health care and **hospitalization** policies.

Mental illnesses compound the vulnerability and needs of homeless adults, as reported by the Surgeon General. Psychiatric disorders exacerbate many types of problems, including housing instability, morbidity (disease), and mortality (death). Psychiatric disorders and lack of stable living conditions complicate general health care for homeless adults.

History

The history of homelessness is intertwined with the history of poverty in the United States. Poverty has always been problematic for humanitarian reasons and because it conflicts with the ideal of prosperity for all. Social welfare, based on individualistic ideas of deserving and undeserving poor, has improved society but not eliminated persistent poverty or homelessness. The 1960s war on poverty was a widely shared value, but in the 1980s concern about homelessness was confounded by moral evaluations of individual behaviors. While

many in the U.S. have been poor or come from poor families, fewer have experienced homelessness. Therefore, the collective understanding of homelessness in the U.S. is limited in ways that the understanding of poverty is not.

Causes and consequences

Causes

People with mental illness are at higher risk for becoming homeless due to challenges associated with **deinstitutionalization** and transition planning, and both poverty and disability associated with mental illness.

Social research has studied the causes and consequences of homelessness, surveying homeless people, examining entrances into homelessness, exits from homelessness, and effects of homelessness on health and well-being. Promising explanations for increasing rates of homelessness in the 1980s have included mental disability and illness, lack of social support through jobs and marriage, increased use of drugs and alcohol, and the erosion of low-income housing in urban areas. These explanations mirror the processes of deinstitutionalization in mental health policy, unemployment, **addiction** and abuse, and urban decay. In other words, a direct correlation can be demonstrated between policies and trends and the rates of homelessness. As deinstitutionalization occurred, for example, the number of mentally ill people without homes increased.

Consequences

Consequences of homelessness include the exacerbation of problems which may have caused homelessness. Homeless people have less access to housing, jobs, health care, and basic needs like food and clothing. Isolation and lack of social support are well-documented aspects of homelessness, particularly for homeless people living with mental health or substance abuse disorders. Homeless women and men have been found to have significantly less family support than never-homeless women and men. Disaffiliation from family often limits opportunities for recovery and prevention.

Homeless service agencies

Services for homeless people can be divided into those providing medical care, those providing housing, and those providing other basic needs. Publicly funded agencies provide the majority of medical care, especially primary and mental health care. Public and private organizations share the responsibilities of providing shelter and housing services, through both large federal programs and smaller need and faith-based programs.

Some studies find that between one-third and one-half of homeless people have mental health disorders and approximately two-thirds have either a mental health or substance use disorder. People with mental illness or substance abuse problems tend to be chronically without homes—it is difficult for many of these people to find permanent housing. *(Archive Photos, Inc. Reproduced by permission.)*

Private agencies deliver most other daily needs to homeless people, through food pantries, soup kitchens, and other charities. Limited data exists on vocational services for homeless adults.

Title VI of the McKinney Homeless Assistance Act of 1987 created the Health Care for the Homeless (HCH) program, authorizing federal funds for primary and mental health care to homeless people. Title VI authorizes several programs to provide a HCH program, a **Community Mental Health** Services block grant program, and two demonstration programs providing mental health and alcohol and drug abuse treatment services to homeless people. HCH funds support providers who offer mental health, **case management**, and health education services, as well as substance abuse treatment. In 1987, 109 grants were made for homeless health services with $46 million; 1996: $66 million were spent for this purpose. Now there are 122 McKinney grants in 48 states. In 1992, the Act was amended to include homeless and at-risk children, creating a medical home and source of health insurance for young people. The HCH program is the largest single effort to address the medical needs of the homeless. Each year, the HCH Program serves almost 500,000 clients in the U.S. To be a HCH service agency requires cultural and linguistic competencies, compassionate community outreach, and providers who reflect the community they serve.

The federal Center for Mental Health Services oversees Projects for Assistance in Transition from

Homelessness (PATH) grant program. PATH provides state funds in support services to individuals who are homeless or at risk of becoming homeless and have serious mental illnesses. These funds amounted to $22 million allocated to 365 providers in 1998. States contract with local agencies and nonprofit organizations to provide an array of services, including outreach, support services, a limited set of housing services, and mental health treatment.

There are several obstacles or barriers in providing health care to homeless people. First, homeless or persistently poor people may be concerned about their work and sustenance, devaluing their own medical needs. Alienation and depression among the homeless can also be an obstacle to providing care. There can be mutual communication problems between providers and patients. Providers may lack cultural understanding that eases work with homeless clients. Finally, lack of preventive maintenance of medical care by the homeless may result in expensive and extensive needs for care, including hospital care, which may **stress** the capacities of certain service providers.

Homelessness in context

Homelessness is both a form of poverty and an acute condition of residential instability. Homelessness is compounded by behavioral problems, mental health policy changes, disparities in health and health care, racial inequalities, fluctuations in affordable housing, and lack of social support. Overly individualistic views and explanations of homelessness do not reflect its multiple causes and effects. Like all groups, homeless people are diverse, experiencing and exiting homelessness for a myriad of reasons. Services for homeless adults likewise reflect a variety of needs and experiences. Nonetheless, homelessness remains a national and international concern, particularly in urban areas, for the twenty-first century.

How to help the homeless mentally ill

There are many ways that Americans can support community and federal efforts to help homeless people living with mental illness. Some strategies include:

- Support collective public and private efforts to build homes and provide health care for people with unmet medical needs.
- Become educated about the challenges faced by homeless and mentally ill people in American society.
- Stop the practice of equating people in poverty and with illness with their medical conditions, instead of recognizing them as human beings. Succeeding in this step could open doors for recovery of health and housing without demeaning the humanity of people in need.

See also Community mental health; Deinstitutionalization; Hospitalization; Vocational rehabilitation

Resources

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Habitat for Humanity International. 121 Habitat St., Americus, GA 31709-3498. (800) 422-4828. <http://www.habitat.org/>.

Health Care for the Homeless Information Resource Center. 262 Delaware Avenue, Delmar, NY 12054-1123. (888) 439-3300 ext. 246. <http://www.pra.inc/hch>.

National Alliance for the Mentally Ill (NAMI). Colonial Place 3, 2107 Wilson Blvd, Suite 300, Arlington VA, 22201-3042. (703) 524-7600 or (800) 950-6264. <http://www.nami.org>.

National Coalition for the Homeless. 1012 Fourteenth Street, NW, #600, Washington, DC 20005. (202) 737-6444. <http://www.nationalhomeless.org/>.

National Coalition for Homeless Veterans. 333 1/2 Pennsylvania Ave., SE, Washington, DC 20003. (800) 838-4357. <http://www.nchv.org>.

National Health Care for the Homeless Council, P.O.Box 60427, Nashville, TN 37206-0427. (615) 226-2292. <http://www.nhchc.org/>.

National Institute of Mental Health. 6001 Executive Boulevard, Rm. 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

Substance Abuse and Mental Health Services Administration (SAMHSA). Center for Mental Health Services (CMHS), Department of Health and Human Services, 5600 Fishers Lane, Rockville MD 20857. <http://www.samhsa.org>.

U.S. Department of Housing and Urban Development. 451 7th Street S.W., Washington, DC 20410. (202) 708-1112. <http://www.hud.gov>.

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Michael Polgar, Ph.D.

Hospitalization

Definition

Hospitalization or inpatient care is the most restrictive form of treatment for a psychiatric disorder, addictive disorder, or for someone with more than one **diagnosis**. Whether it is voluntary or involuntary, the patient relinquishes the freedom to move about and, once admitted, becomes subject to the rules and schedule of a treatment environment. Hospitalization is necessary in cases where an individual is in imminent danger of harming himself or others or has made a **suicide** attempt. Crisis stabilization, **behavior modification**, supervised substance abuse **detoxification**, and medication management are compelling reasons to consider hospitalization. Ideally, hospitalization is at one end of a comprehensive continuum of services for people needing treatment for behavioral problems. It is generally viewed as a last resort after other less restrictive forms of treatment have failed.

Purpose

In order to be admitted to a hospital, a medical doctor (in the case of mental health, most often a **psychiatrist**) must "admit" the patient or approve the patient's request to be admitted. Although hospitalization may be considered a drastic treatment **intervention,** it can be essential in keeping people safe, helping monitor and adjust medications, treating medication side effects, supervising alcohol and/or drug detoxification, and stabilizing a patient after an acute psychiatric episode.

Before an individual is hospitalized, an evaluation and a diagnosis must be made by a medical professional. This is required in order for the patient to receive maximum insurance coverage and to receive the most appropriate treatment.

Precautions

In the public mental health system, less restrictive forms of treatment other than hospitalization are strongly recommended first. In the late 1960s the patients' rights movement led to reforms governing **involuntary hospitalization**. Today the criteria for admission, particularly in the case of involuntary hospitalization, are extremely narrow, reflecting a strong reluctance in this country to infringe on any person's liberty. The unintended consequences of this public policy are often observed in the numbers of people with mental illnesses who are homeless. So long as they are not posing a danger to themselves or others, they are likely to remain outside the traditional treatment system.

Hospitalization has long been negatively characterized in the media, contributing to the **stigma** of seeking inpatient treatment, even when it is voluntary. Scenes from the 1975 movie *One Flew Over the Cuckoo's Nest* have defined the worst in psychiatric hospital treatment. Such conditions cannot exist long in today's more sophisticated mental health, consumer-focused environment. A reputable facility will be accredited by the Joint Commission on Accreditation of Health Care Organizations, or by a similar governing body, which usually assures a minimum level of service. Most hospitals now have a Patient Advocate, usually an attorney who is on-site daily, or accessible by phone, and whose job is to investigate complaints and protect a patient's rights. In addition, a federal law mandates that every state have a Protection and Advocacy Agency to handle complaints of abuse in hospitals. While their effectiveness varies from state to state, they can be helpful in explaining the rights of a hospitalized patient. Some states have also implemented ombudsman programs to address patient complaints and to help people negotiate the mental health system.

Treatment facilities may be locked or unlocked. A locked unit will have tighter security to protect patient privacy and to keep patients from running away. In most cases when a patient is voluntarily admitted, he or she may leave treatment at any time, invoking the right to do so against medical advice (AMA).

In the past, patients were often not part of their own treatment planning process. The rise of the patients' rights movement has led to more active patient involvement in all phases of treatment. They have the right to refuse certain forms of treatment. Most hospitals now have a clearly posted Patient's Bill of Rights and may also have a patient's council or other body to represent their interests and recommend changes to the inpatient environment.

Confidentiality is paramount in a hospital setting, so much so that hospital staff seldom acknowledge that a specific patient has been admitted. **Group therapy** rules generally **stress** the importance of keeping members and the content of group sessions confidential.

Description

Most hospital rooms are similar to basic hotel rooms and are generally large enough for two people. In the case of public hospitals, the rooms may be larger and contain more beds. Men and women are in separate wings or on separate floors. If a treatment program is housed in a medical hospital, it may cover one or more floors.

While there is wide variation in the quality of the physical surroundings and the resources available, most inpatient facilities are highly regimented. Patients get up, go to bed, eat, and take medication (if indicated) on a regular schedule. Days are filled with scheduled activities such as individual, family, or group therapy, expressive and occupational therapies, psychoeducation, recreation, and, in the case of children or adolescents, several hours of school.

Most hospital inpatient programs are based on a therapeutic milieu, which means that all the people involved in the patient's care and all the activities are designed to have a therapeutic function for the patient. For example, direct care workers are not simply aides, but they are supportive of the patient and provide valuable feedback to the physician, **psychologist**, and social worker about the patient's conduct and progress.

Preparation

Even voluntary hospitalization can be overwhelming and anxiety-provoking. As a result, when first admitted, a patient will be closely observed by the staff. If the patient was admitted because of a suicide attempt or a violent episode, a "suicide watch" may be set up with more intensive staffing or in a room that can be monitored easily by nursing staff.

As the patient adjusts to the hospital routine, more privileges and freedom will be made available. For example, a patient may earn privileges or rewards like outings with staff, a weekend pass to go home for a visit, or some other positive consequence if he or she follows hospital rules and engages in therapeutic activities.

An interdisciplinary treatment team made up of a psychiatrist, psychologist, social worker, nurse, direct care worker (sometimes called a psychiatric technician), and an expressive therapist usually oversees a patient's care while he or she is in the hospital. Treatment goals are developed by the team with patient input, with discharge as a major objective.

Aftercare

Optimally, inpatient treatment prepares a patient to deal with the realities of life outside the hospital. Emphasis is placed on how a patient will behave differently in order to remain healthy and avoid future hospitalizations. During the discharge phase, a patient may be scheduled for outpatient therapy and informed about various medications. Often, a patient experiences anxiety at the thought of leaving the hospital, and this apprehension is addressed in therapy sessions as discharge nears.

Normal results

In the past, a patient might be admitted to a hospital for a minimum of 30 days. Today's rising health care costs and the prevalence of **managed care** have led to dramatically reduced hospital stays. An optimal outcome under these conditions is medication adjustment, monitoring, and the beginning of stabilization. Studies are underway to determine if shortened stays ultimately lead to more frequent hospitalizations later.

Resources

ORGANIZATIONS

National Alliance for the Mentally Ill. Colonial Place Three, 2107 Wilson Blvd., Suite 300, Arlington, VA 22201. <http://www.nami.org>.

National Association of Protection and Advocacy Systems. 900 2nd St. NE, Washington, DC 20001. <http://www.napas.org>.

National Mental Health Association. 1021 Prince St., Alexandria, VA 22314. <http://www.nmha.org>.

National Mental Health Consumers' Self-Help Clearinghouse. 1211 Chestnut St., Suite 1207, Philadelphia, PA 19107. <http://www.mhselfhelp.org>.

Judy Leaver, M.A.

House-tree-person test

Definition

The house-tree-person test (HTP) is a projective personality test, a type of exam in which the test taker responds to or provides ambiguous, abstract, or unstructured stimuli (often in the form of pictures or drawings). In the HTP, the test taker is asked to draw houses, trees,

and persons, and these drawings provide a measure of self-perceptions and attitudes. As with other projective tests, it has flexible and subjective administration and interpretation.

Purpose

The primary purpose of the HTP is to measure aspects of a person's personality through interpretation of drawings and responses to questions. It is also sometimes used as part of an assessment of **brain** damage or overall neurological functioning.

The HTP was developed in 1948, and updated in 1969. Tests requiring human **figure drawings** were already being utilized as projective personality tests. Buck believed that drawings of houses and trees could also provide relevant information about the functioning of an individual's personality.

Precautions

Because it is mostly subjective, scoring and interpreting the HTP is difficult. Anyone administering the HTP must be properly trained. The test publishers provide a detailed 350-page administration and scoring manual.

Description

The HTP can be given to anyone over the age of three. Because it requires test takers to draw pictures, it is often used with children and adolescents. It is also often used with individuals suspected of having brain damage or other neurological impairment. The test takes an average of 150 minutes to complete; it may take less time with normally functioning adults and much more time with neurologically impaired individuals.

During the first phase of the test, test takers are asked to use a crayon to draw pictures, respectively, of a house, a tree, and a person. Each drawing is done on a separate piece of paper and the test taker is asked to draw as accurately as possible. Upon completion of the drawings, test takers are asked questions about the drawings. There are a total of 60 questions that examiners can ask. Examiners can also create their own questions or ask unscripted follow-up questions. For example, with reference to the house, the test creator wrote questions such as, "Is it a happy house?" and "What is the house made of?" Regarding the tree, questions include, "About how old is that tree?" and "Is the tree alive?" Concerning the person, questions include, "Is that person happy?" and "How does that person feel?"

KEY TERMS

Projective test—A psychological test in which the test taker responds to or provides ambiguous, abstract, or unstructured stimuli, often in the form of pictures or drawings.

Reliability—The ability of a test to yield consistent, repeatable results.

Validity—The ability of a test to measure accurately what it claims to measure.

During the second phase of the test, test takers are asked to draw the same pictures with a pencil. The questions that follow this phase are similar to the ones in the first phase. Some examiners give only one of the two phases, choosing either a crayon, a pencil, or some other writing instrument.

One variation of test administration involves asking the individual to draw two separate persons, one of each sex. Another variation is to have test takers put all the drawings on one page.

Results

The HTP is scored in both an objective quantitative manner and a subjective qualitative manner. The quantitative scoring scheme involves analyzing the details of drawings to arrive at a general assessment of intelligence, using a scoring method devised by the test creators. Research has shown this assessment of intelligence correlates highly with other **intelligence tests** such as the **Wechsler adult intelligence scale** (WAIS).

The primary use of the HTP, however, is related to the qualitative scoring scheme in which the test administrator subjectively analyzes the drawings and the responses to questions in a way that assesses the test taker's personality. For example, a very small house might indicate rejection of one's home life. A tree that has a slender trunk but has large expansive branches might indicate a need for satisfaction. A drawing of a person that has a lot of detail in the face might indicate a need to present oneself in an acceptable social light.

Other methods of interpretation focus on the function of various parts in each of the drawings. In the house drawing, the roof might represent one's intellectual side, the walls might represent the test taker's degree of ego strength, and the doors and windows might represent the individual's relation to the outside world. In the tree drawing, the branches might indicate the test taker's rela-

tion to the outside world and the trunk might indicate inner strength.

As with other subjectively scored personality tests, there is little support for its reliability and validity. However, there is some evidence that the HTP can differentiate people with specific types of brain damage. More specifically, it has been shown to be effective when looking at the brain damage present in schizophrenic patients.

See also Figure drawings; Rorschach technique

Resources

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Ali Fahmy, Ph.D.

Hypericum *see* **St. John's wort**

Hypersomnia

Definition

Hypersomnia refers to a set of related disorders that involve excessive daytime sleepiness.

Description

There are two main categories of hypersomnia: primary hypersomnia (sometimes called idiopathic hypersomnia) and recurrent hypersomnia (sometimes called recurrent primary hypersomnia). Both are characterized by the same signs and symptoms and differ only in the frequency and regularity with which the symptoms occur.

Primary hypersomnia is characterized by excessive daytime sleepiness over a long period of time. The symptoms are present all, or nearly all, of the time. Recurring hypersomnia involves periods of excessive daytime sleepiness that can last from one to many days, and recur over the course of a year or more. The primary difference between this and primary hypersomnia is that persons experiencing recurring hypersomnia will have prolonged periods where they do not exhibit any signs of hypersomnia, whereas persons experiencing primary hypersomnia are affected by it nearly all the time. One of the best documented forms of recurrent hypersomnia is Kleine-Levin syndrome, although there are other forms as well.

KEY TERMS

Hypothalamus—A part of the forebrain that controls heartbeat, body temperature, thirst, hunger, blood pressure, blood sugar levels, and other functions.

Narcolepsy—A disorder characterized by frequent and uncontrollable attacks of deep sleep.

There are many different causes for daytime sleepiness that are not considered hypersomnia, and there are many diseases and disorders in which excessive daytime sleepiness is a primary or secondary symptom. Feelings of daytime sleepiness are often associated with the use of common substances such as caffeine, alcohol, and many medications. Other common factors that can lead to excessive daytime sleepiness that is not considered hypersomnia include shift work and **insomnia**. Shift work can disrupt the body's natural sleep rhythms. Insomnia can cause excessive daytime sleepiness because of lack of nighttime sleep, and is a separate disorder.

Causes and symptoms

People experiencing hypersomnia do not get abnormal amounts of nighttime sleep. However, they often have problems waking up in the morning and staying awake during the day. People with hypersomnia nap frequently, and upon waking from the nap, do not feel refreshed. Hypersomnia is sometimes misdiagnosed as **narcolepsy**. In many ways the two are similar. One significant difference is that people with narcolepsy experience a sudden onset of sleepiness, while people with hypersomnia experience increasing sleepiness over time. Also, people with narcolepsy find daytime sleep refreshing, while people with hypersomnia do not.

People with Kleine-Levin syndrome have symptoms that differ from the symptoms of other forms of hypersomnia. These people may sleep for 18 or more hours a day. In addition, they are often irritable, uninhibited, and make indiscriminate sexual advances. People with Kleine-Levin syndrome often eat uncontrollably and rapidly gain weight, unlike people with other forms of hypersomnia. This form of recurrent hypersomnia is very rare.

The causes of hypersomnia remain unclear. There is some speculation that in many cases it can be attributed

to problems involving the hypothalamus, but there is little evidence to support that claim.

Demographics

Hypersomnia is an uncommon disorder. In general, 5% or fewer of adults complain of excessive sleepiness during the daytime. That does not mean all those who complain of excessive sleepiness have hypersomnia. There are many other possible causes of daytime sleepiness. Of all the people who visit sleep clinics because they feel they are too sleepy during the day, only about 5–10% are diagnosed with primary hypersomnia. Kleine-Levin syndrome is present in about three times more males than females, but it is a very rare syndrome.

Hypersomnia generally appears when the patient is between 15 and 30 years old. It does not begin suddenly, but becomes apparent slowly, sometimes over years.

Diagnosis

Hypersomnia is characterized by excessive daytime sleepiness, and daytime naps that do not result in a more refreshed or alert feeling. Hypersomnia does not include lack of nighttime sleep. People experiencing problems with nighttime sleep may have insomnia, a separate sleep disorder. In people with insomnia, excessive daytime sleepiness may be a side effect.

The ***Diagnostic and Statistical Manual of Mental Disorders***, which presents the guidelines used by the American Psychiatric Association for **diagnosis** of disorders, states that symptoms must be present for at least a month, and must interfere with a person's normal activities. Also, the symptoms cannot be attributed to failure to get enough sleep at night or to another sleep disorder. The symptoms cannot be caused by another significant psychological disorder, nor can they be a side effect of a medicinal or illicit drug or a side effect of a general medical condition. For a diagnosis of recurrent hypersomnia, the symptoms must occur for at least three days at a time, and the symptoms have to be present for at least two years.

Treatments

There have been some attempts at using drugs to treat hypersomnia. No substantial body of evidence supports the effectiveness of these treatments. Stimulants are not generally recommended to treat hypersomnia as they treat the symptoms but not the base problem. Some researchers believe that treatment of the hypothalamus may be a possible treatment for hypersomnia.

Prognosis

Kleine-Levin syndrome has been reported to resolve occasionally by itself around middle age. Except for that syndrome, hypersomnia is considered both a lifelong disorder and one that can be significantly disabling. There is no body of evidence that concludes there is a way to treat the majority of hypersomnia cases successfully.

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ORGANIZATIONS

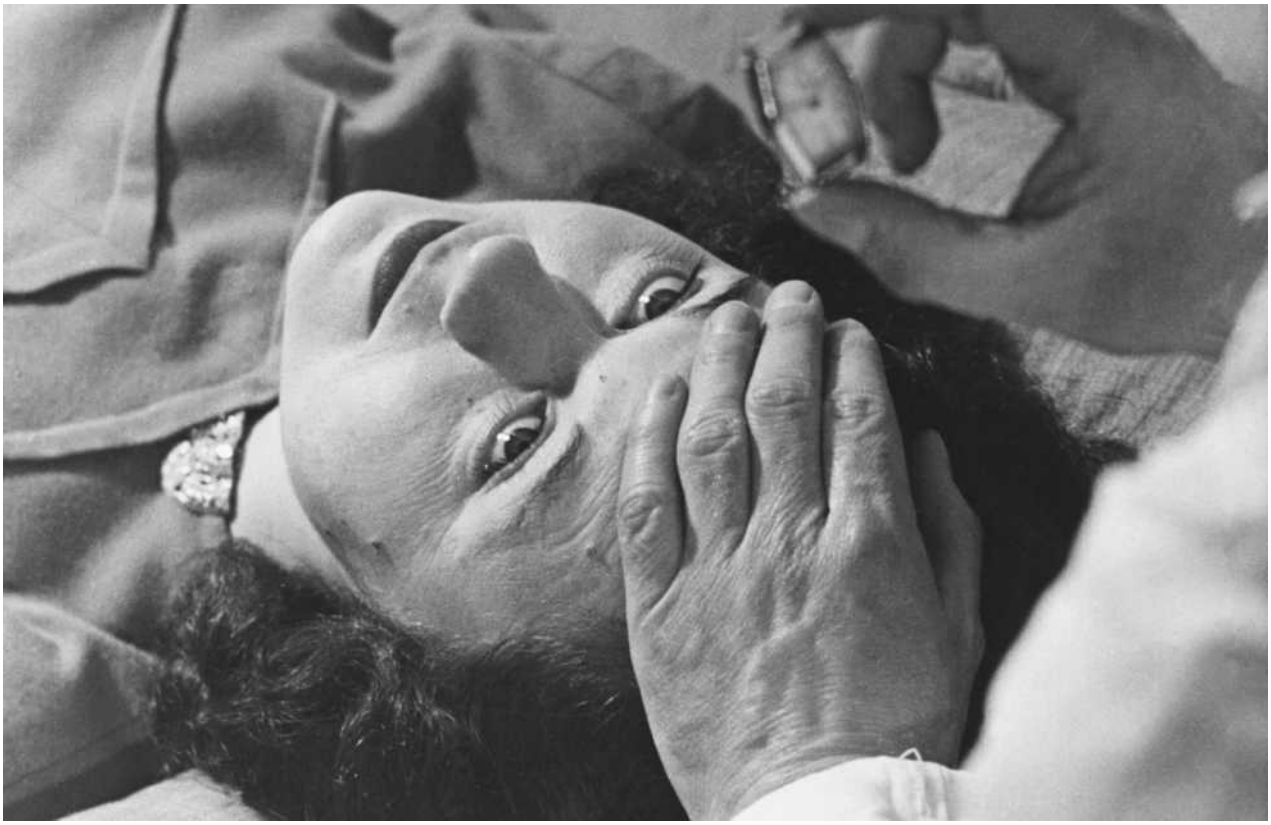
American Academy of Sleep Medicine. 6301 Bandel Road NW, Suite 101, Rochester, MN 55901. (507) 287-6006. <www.asda.org>.

Tish Davidson, A.M.

Hypnotherapy

Definition

Hypnotherapy is a combination of hypnosis and therapeutic **intervention**. The therapist leads the patient to positive change while the patient is deeply relaxed in a state of heightened suggestibility called trance.



Hypnosis can be a highly effective form of treatment for many mental, psychosomatic, and physical disorders. Hypnosis is a trance state in which the hypnotized person is in a heightened, more receptive state of mind. During hypnosis, the patient is not unconscious, does not lose control of his or her faculties, and does not do things under hypnosis that he or she would be unwilling to do otherwise. *(Hulton-Deutsch Collection/ CORBIS. Photo reproduced by permission.)*

Purpose

Hypnosis, when using proven therapeutic procedures, can be a highly effective form of treatment for many mental, psychosomatic, and physical disorders. For example, through the use of regressive techniques, an adult patient may mentally voyage back to a point in youth that was particularly troublesome, allowing the healing of old emotional wounds. Another patient can be led to understand that emotional pain has been converted to physical pain, and that the pain can be eliminated once the source has been addressed. Or, a person suffering from chronic pain can be taught to control the pain without use of medications. There are a number of techniques for correcting dysfunctional behaviors such as self-destructive habits, anxiety disorders, and even managing side effects of various medical treatments and procedures.

Hypnotherapy has been used to stop self-destructive and addictive habits like smoking. It has also been used to curb the urge to eat for overeaters, to stem the disruptive actions of tics, cure **insomnia**, stop bed-wetting, and minimize anxiety. Excessive **stress** can be generated from any number of sources and can be the springboard for anxiety. Some of the more prominent sources of anxiety and stress for which people seek hypnotherapy are: public speaking, test taking, and job stress. Hypnotherapy also works well for other anxiety disorders such as phobias and has proven to be an effective treatment for mild to moderate depression. In one study, hypnotherapy was used in conjunction with traditional cognitive therapy, to assist persons who had severe aversion to needles. The treatment was necessary, because it was essential that each participant receive periodic medical injections. However, the participants would have become non-compliant without the adjunct intervention of hypnotherapy. In another case, involving care for terminally ill cancer patients, it was concluded that hypnotherapy was more effective at enhancing quality of life and relieving anxiety and depressive symptoms, when compared to others who received traditional care.

Precautions

Confusion can occur when one seeks a hypnotherapist, as a result of the various titles, certifications, and

licenses in the field. Many states do not regulate the title "hypnotist" or "hypnotherapist," so care must be exercised when selecting someone to see. As a rule, it is best to consult a professional in the field of mental health or medicine, although alternative sources for hypnosis are available. Care must be taken also by the therapist to ensure adequate training and sufficient experience for rendering this specialized service. The therapist must be well grounded in a psychotherapeutic approach before undertaking the use of hypnotherapy. Professionals should not attempt hypnotherapy with any disorder for which they would not use traditional therapeutic approaches. The patient seeking hypnotherapy is reminded that unskilled or amateur hypnotists can cause harm and should not be consulted for the purpose of implementing positive change in an individual's life. The detrimental effects of being subjected to amateur or inadequately trained persons can be severe and long lasting. (See abnormal results below.)

Description

In order to understand hypnotherapy, it is necessary to understand the underlying concepts of hypnosis.

History of hypnosis

It appears that hypnosis, under other names, has been used since the beginning of time. In fact, it has been insinuated that the earliest description of hypnosis may be portrayed in the Old Testament and in the Talmud. There is also evidence of hypnosis in ancient Egypt, some 3,000 years ago. However, the man credited with the development of what has become modern hypnosis is Friedrich Anton Mesmer, an Austrian physician. One day, Mesmer watched a magician on a street in Paris demonstrate that he could have spectators do his bidding by touching them with magnets. Fascinated by the demonstration, Mesmer believed the magnets had power of their own and from this belief developed his theory of "animal magnetism." He also believed that good health depended on having correct magnetic flow and that the direction of one's magnetic flow could be reversed easily. He further believed that he could direct this magnetic flow into inanimate objects, that could then be used for the good health of others. The term "mesmerism" came to be applied to his mystical workings. He experienced much success in helping the people of Paris as well as visitors who came from other countries, upon hearing of his powers. Later he was completely discredited by a special commission of the French Academy appointed by the King of France, causing him to leave the country. Two of the more famous members of the French Academy at the time were chairman of the commission Benjamin Franklin, American ambassador to France, and Dr. Guillotine, the inventor of the execution device.

Later, around 1840, a patient in the office of Scottish physician James Braid, accidentally entered a state of trance while waiting for an eye examination. Braid, aware of the disfavor of mesmerism and animal magnetism coined the term "hypnosis," and thus began the serious study of this altered state of awareness.

What is hypnosis?

It is far easier to describe what hypnosis is not rather than to describe what it is. For example, it is not one person controlling the mind of another. The patient is not unconscious and does not lose control of his or her faculties. People will not do things under hypnosis that they would be unwilling to do otherwise. The person being hypnotized is always in control. The hypnotized person decides how deep the trance will be, what suggestions will be accepted, and when to awaken. Therefore, a hypnotyized person cannot be forever "lost" if the therapist should fall dead during an induction or while the patient is deep in trance.

Hypnosis is first and foremost a self-accepted journey away from the reality of the moment. Although the trance state is often referred to as if the patient is asleep, nothing could be further from the truth. The patient is fully awake at all times. The hypnotic subject is simply in a heightened, more receptive state of mind. This fact is proven with inductions called open-eye techniques, where the patient keeps his/her eyes open during the hypnotherapy. Full and deep trance is still achievable.

Trance is commonplace. People fall into traces many times without even being aware that it happened. Examples of this are: reaching the destination of a morning commute, but not recalling the passing of familiar landmarks; daydreaming while sitting in a college classroom; or that anxiety-free state achieved just before going to sleep. The difference between these altered states and clinically used hypnotherapy is that a professionally trained person is involved in helping the patient achieve the trance, which can be done in many ways.

A typical hypnotherapy session has the patient seated comfortably with their feet on the floor and palms on their lap. Of course, the patient could choose to lie down if that option is available and if that will meet the patient's expectation of hypnosis. The therapist can even set the stage for a favorable outcome by asking questions like, "Would you prefer to undergo hypnosis in this chair or on the sofa?" Once patients make the choice, they are in effect agreeing to undergo hypnosis. Depending on

the approach used by the therapist, the next events can vary, but generally will involve some form of relaxing the patient. Suggestions will lead the patient to an increasingly relaxed state. The therapist may wish to confirm the depth of trance by performing tests with the patient. For example, the therapist may suggest that when the eyes close that they will become locked and cannot be opened. The therapist then checks for this by having patients try to open their eyes. Following a successful trial showing the patient's inability to open the eyes, the therapist might then further relax them by using deepening techniques. Deepening techniques will vary for each patient and depend largely on whether the patient represents information through auditory, visual, or kinesthetic means. If the patient is more affected by auditory suggestions, the therapist would use comments such as "You hear the gentle patter of rain on the roof;" or, "The sound of the ocean waves allow you to relax more and more." For the visual person, the therapist might use statements such as, "You see the beautiful placid lake, with trees bending slightly with the breeze." Finally, with the kinesthetic person phrases such as, "You feel the warm sun and gentle breeze on your skin," could be used. It is important for the therapist to know if the patient has difficulty with the idea of floating or descending because these are sometimes used to enhance the experience for the patient. However, if the patient has a fear of heights or develops a feeling of oppression with the thought of traveling downward and going deeper and deeper, suggestions implying the unwanted or feared phenomenon will not be taken and can thwart the attempt.

Modern techniques

In order for a hypnotherapist to convey positive suggestions for change, the patient must be in a receptive state. The state is called trance and the method of achieving a trance is through induction. Induction techniques are many and varied and involve the therapist offering suggestions that the patient follows. The formerly common "your eyes are getting heavy" suggestion may still exist, but other more reliable and acceptable (by the patient) forms of induction have come to the forefront. The artful hypnotherapist is always aware of the present condition of the patient and uses this information to lead him/her down the path of induction. In its lighter stages, trance can be noted by the relaxation of muscles. At this point, hands can levitate when given the suggestion, and paresthesia, a feeling of numbness, can be induced. In a medium trance, a patient can be led to experience partial or complete **amnesia**, or failure to recall events of the induction after the fact. A deep trance opens the patient to powerful auditory, visual, or kinesthetic experiences. The phenomenon of time distortion is experienced most profoundly at this level. Patients may believe they have been away briefly, and may react with disbelief when told they were away much longer. Although some work can be done in lighter states of trance, the best circumstance for implementing change is when the patient reaches a deep trance state. At this level, the patient is focused inwardly and is more receptive to positive suggestions for change. This is also the point at which the therapist can invoke posthypnotic suggestions, or instructions given to the patient so he/she will perform some act or experience some particular sensation following awakening from the trance. For example, these suggestions, if accepted by the patient, can be formed to make foods taste bad, cigarettes taste bad, delay impulses, curb hunger, or eliminate pain. However, it should be noted that posthypnotic suggestions given to a person, which run counter to the person's value system or are not something they are likely to do under ordinary circumstances, will not be accepted and therefore not implemented.

Neuro-Linguistic Programming (NLP) is the name given to a series of models and techniques used to enhance the therapist's ability to do hypnotherapy. NLP consists of a number of models, with a series of techniques based on those models. Sensory acuity and physiology is one model whose premise is that a person's thought processes change their physiological state. People recognize such a physiological change when startled. The body receives a great dose of adrenaline, the heart beats faster, the scare may be verbalized by shouting, and the startled person may sweat. Sensory acuity, (i.e., being attuned to changes occurring in another person) will strengthen communication to a person in ways over and above simple verbal cues, therefore making the therapist more effective. A second model of NLP deals with representational systems. The idea behind this model is that different people represent knowledge in different sensory styles. In other words, an individual's language reveals that person's mode of representation. There are three basic modes of representation. These are: Auditory, Visual, and Kinesthetic. The same information will be expressed differently by each. For example, the auditory person might say, "That sounds good to me;" the visual person might convey, "I see it the same way;" and the kinesthetic person would offer, "I'm comfortable with it too."

Preparation

Before people subject themselves to hypnotherapy they are advised to learn as much about the process and about the chosen therapist as is necessary to feel comfortable. Rapport and trust are two key ingredients in

making a potential hypnotherapy patient comfortable. Therapists should be open and willing to answer all questions regarding qualifications, expertise, and methods used. A well-qualified professional will not undertake the use of hypnosis without interviewing the patient to ascertain their level of understanding of the process. This is very important for two reasons. First, it allows the patient the opportunity to have questions answered and to develop some rapport with the therapist. Second, it is important for the therapist to know the patient's expectations since meeting these expectations will enhance the likelihood of success.

Aftercare

Depending on the purpose of the hypnotherapy (i.e., smoking cessation, weight loss, improvement in public speaking, or addressing some deep emotional turmoil), follow-up may be advisable. When trying to eradicate unwanted habits, it is good practice to revisit the therapist, based upon a date prearranged between the therapist and the patient, to report progress and, if necessary, to obtain secondary hypnotherapy to reinforce progress made.

Risks

One obvious risk to patients is the insufficiently trained therapist. The inadequately trained therapist can cause harm and distort the normally pleasant experience of hypnotherapy. A second risk for patients is the unscrupulous practitioner who may be both inadequately trained and may have some hidden agenda. These rare individuals are capable of causing great harm to the patient and to the profession. As mentioned above, the patient should carefully scrutinize their chosen therapist before submitting themselves to this dynamic form of therapy.

Normal results

The result of hypnotherapy is overwhelmingly positive and effective. Countless success stories exist attesting to the benefits of this technique. Many people have stopped smoking, lost weight, managed pain, remembered forgotten information, stopped other addictions, or improved their health and well-being through its use.

Abnormal results

Abnormal results can occur in instances where amateurs, who know the fundamentals of hypnosis, entice friends to become their experimental subjects. Their lack of full understanding can lead to immediate consequences, which can linger for some time after the event. If, for example, the amateur plants the suggestion that the subject is being bitten by mosquitoes, the subject would naturally scratch where the bites were perceived. When awakened from the trance, if the amateur forgets to remove the suggestion, the subject will continue the behavior. Left unchecked, the behavior could land the subject in a physician's office in an attempt to stop the itching and scratching cycle. If the physician is astute enough to question the genesis of the behavior and hypnosis is used to remove the suggestion, the subject may experience long-term negative emotional distress and anger upon understanding exactly what happened. The lack of full understanding, complete training, and supervised experience on the part of the amateur places the subject at risk.

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ORGANIZATIONS

American Society of Clinical Hypnosis. 2250 East Devon Avenue, Suite 336, Des Plaines, IL 60018.

Society for Clinical and Experimental Hypnosis. 129-A Kings Park Drive, Liverpool, NY 13088.

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Hypoactive sexual desire disorder

Definition

Hypoactive sexual desire disorder (HSDD) is defined as the persistent or recurrent extreme aversion to, absence of, and avoidance of all, or almost all, genital sexual contact with a sexual partner. Synonyms for HSDD include sexual aversion, inhibited sexual desire, sexual **apathy**, and sexual anorexia. HSDD is not rare, occurring in both sexes. It is the most common of all female sexual disorders, occurring in at least 20% of women in the United States.

Description

The affected person has a low level of sexual interest and desire that is manifested by a failure to initiate or be responsive to a partner's initiation of sexual activity. HSD becomes a diagnosable disorder when it causes marked distress or interpersonal instability, according to the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (also known as the *DSM-IV-TR*), the handbook used by mental health professionals to diagnose mental disorders. HSDD may be either situational (solely oriented against one partner), or it may be general, in which case there is a lack of sexual interest in anyone. In the extreme form of HSDD, the patient not only lacks sexual desire, but may also find sex to be repulsive, revolting, and distasteful. Phobic or panic responses may be present in extreme cases of HSDD. HSDD may be the result of either physical or emotional factors.

Causes and symptoms

Causes

PRIMARY HSDD. HSDD may be a primary condition in which the patient has never felt much sexual desire or interest, or it may have occurred secondarily when the patient formerly had sexual desire, but no longer has interest. If lifelong or primary, HSDD may be the consequence of sexual trauma such as incest, sexual **abuse**, or rape. In the absence of sexual trauma, there is often a repressive family attitude concerning sex that is sometimes enhanced by rigid religious training. A third possibility is that initial attempts at sexual intercourse resulted in pain or sexual failure. Rarely, HSDD in both males and females may result from insufficient levels of the male sex hormone, testosterone.

ACQUIRED HSDD. Acquired, situational HSDD in the adult is commonly associated with boredom in the relationship with the sexual partner. Depression, the use of psychoactive or antihypertensive medications, and hormonal deficiencies may contribute to the problem. HSDD may also result from impairment of sexual function, particularly **erectile dysfunction** on the part of the male, or **vaginismus** on the part of the female. Vaginismus is defined as a conditioned voluntary contraction or spasm of the lower vaginal muscles resulting from an unconscious desire to prevent vaginal penetration. An incompatibility in sexual interest between the sexual partners may result in relative HSDD in the less sexually active member. This usually occurs in the presence of a sexually demanding partner.

PAINFUL INTERCOURSE. Painful intercourse (**dyspareunia**) is more common in women than in men, but may be a deterrent to genital sexual activity in both sexes. The causes are usually physical in nature and related to an infection of the prostate gland, urethra, or testes. Occasionally, an allergic reaction to a spermicidal preparation or condom may interfere with sexual intercourse. Painful erections may be a consequence of Peyronie's disease, which is characterized by fibrotic changes in the shaft of the penis that prevent attainment of a normal erection. In the female, dyspareunia may be caused by vaginismus or local urogenital trauma or inflammatory conditions such as hymenal tears, labial lacerations, urethral bruising, or inflammatory conditions of the labial or vaginal glands.

PRIAPISM. Priapism is the occurrence of any persistent erection of more than four hours duration occurring in the absence of sexual stimulation. It is not associated with sexual excitement and the erection does not subside after ejaculation. Priapism can occur at any age, but clusters of occurrence are common between the ages of five and 10 years and between the ages of 20 and 50. In children, priapism is commonly associated with leukemia and sickle cell disease, or occurs secondary to trauma. The most common cause in adults is the intrapenile injection of agents to correct erectile dysfunction. Priapism may also occur secondary to the use of psychotropic

drugs, such as **chlorpromazine** and prazosin. The pain accompanying priapism may be a cause of HSDD.

PROLACTINOMA. A rare but important cause of HSDD is a functioning prolactin-secreting tumor of the pituitary gland, a prolactinoma. Men with this condition typically state that they can achieve an erection, but that they have no interest in sexual relations. In the female, prolactinomas are associated with galactorrhea (lactation in the absence of pregnancy), amenorrhea, symptoms of estrogen deficiency, and dyspareunia. Although prolactinomas are benign tumors, they can cause visual disturbances by enlarging and causing pressure on the optic nerves within the confines of the *sella turcica,* the location of the pituitary gland at the base of the **brain**. Headaches and enlargement of the male breasts are fairly common in this condition. The **diagnosis** is confirmed by the finding of high levels of circulating prolactin in the blood. Enlargement of the pituitary gland area may be detected by the use of **magnetic resonance imaging** (MRI) or computerized axial tomography (CAT) scanning, also called **computed tomography**.

DELAYED SEXUAL MATURATION. Delayed sexual maturation is a potential cause of HSDD. It is present in boys if there is no testicular enlargement by age 13-and-a-half or if there are more than five years between the initial and complete growth of the genitalia. In girls, delayed sexual maturation is characterized by a lack of breast enlargement by age 13, or by a period greater than five years between the beginning of breast growth and the onset of menstruation. Delayed puberty may be the result of familial constitutional disorders, genetic defects such as Turner's syndrome in females and Klinefelter's syndrome in males, central nervous system disorders such as pituitary conditions that interfere with the secretion of gonadotropic hormones, and chronic illnesses such as diabetes mellitus, chronic renal failure, and cystic fibrosis.

SEXUAL ANHEDONIA. Sexual anhedonia is a rare variant of HSDD seen in the male, in which the patient experiences erection and ejaculation, but no pleasure from orgasm. The cause is attributed to penile anesthesia, due to psychological or emotional factors in a hysterical or obsessive person. Psychiatric referral is indicated unless there is evidence of spinal cord injury or peripheral neuropathy. Loss of tactile sensation of the penis is unlikely to be organic in cause unless there is associated anesthetic areas in the vicinity of the anus or scrotum.

Symptoms

The HSDD patient complains of a lack of interest in sex even under circumstances that are ordinarily erotic in nature, such as pornography. Sexual activity is infrequent and eventually is absent, often resulting in serious marital discord. HSDD may be selective and focused against a specific sexual partner. When boredom with the usual sexual partner is the cause and frequency of sex with the usual partner decreases, real or fantasized sexual desire toward others may be normal or even increased.

If the cause of HSDD falls into a detectable category such as abnormalities of the genitalia, or is due to a related condition such as a prolactinoma, chronic renal disease, diabetes mellitus, genetic disorder, or is familial in nature, the patient will manifest the signs and symptoms of the comorbid (co-occurring) condition. It is important to identify such causes, as their presence will usually dictate appropriate therapy.

KEY TERMS

Comorbid—Having another disorder or condition simultaneously.

Dyspareunia—Painful sexual intercourse.

Galactorrhea—Lactation occurring in the absence of pregnancy

Hypogonadism—Abnormally decreased gonad function with retardation of sexual development.

Priapism—Painful involuntary penile erection persisting in excess of four hours.

Prolactin—A hormone that stimulates milk production and breast development.

Vaginismus—An involuntary tightening of the vaginal muscles that makes sexual intercourse painful, difficult, or impossible.

Treatments

Currently, there is no approved drug or pharmacological treatment for HSDD and **psychotherapy** has proved to be only minimally effective. A primary goal of therapy is aimed at removal of the underlying cause of HSDD. The choice of medical therapy or behavioral or dynamic psychotherapy depends on the cause. If the cause is related to a medical condition, therapy is directed toward the cure or amelioration of that condition. Examples include cure or amelioration of underlying comorbid conditions such as genitourinary infections, improvement in diabetic control, avoidance of substance abuse and of medications that may be potentially responsible.

Therapy should also be directed towards other accompanying sexual disorders such as erectile dysfunction, which may be contributory. In cases where insuffi-

cient testosterone is suspected as a possible cause, serum androgen levels should be tested. A testosterone level less than 300 ng/dl in males and less than 10 ng/dl in females indicates a need for supplemental replacement therapy. If the cause is deemed to be of an interpersonal nature, **couples therapy** may be beneficial, in which case the support and understanding of the sexual partner is essential. Tricyclic antidepressants (TCAs) or monoamine oxidase inhibitors (MAOIs) may help in the treatment of accompanying depression or panic symptoms.

A recent study has reported that almost a third of nondepressed women with HSDD responded favorably to therapy with sustained release tablets of **bupropion** hydrochloride. The responders noted significant increases in the number of sexual arousals, sexual fantasies, and in the desire to engage in sexual activities. Bupropion hydrochloride (sold as Wellbutrin by Glaxo Wellcome) is currently approved by the FDA for the treatment of depression. Its favorable action on HSDD may be attributable to its enhancement of certain **neurotransmitters** that affect sexual desire, principally norepinephrine and dopamine.

Prognosis

The prognosis for HSDD depends primarily on the underlying cause or causes. In certain medical conditions, the prognosis for development, or recovery of sexual interest, is good. Examples include therapy of hypogonadism with testosterone, or the appropriate treatment of a prolactin-secreting pituitary tumor. On the other hand, in certain genetic defects such as Turner's syndrome and Klinefelter's syndrome, attainment of sexual function is impossible. By far, however, the vast majority of HSDD cases are situational in nature, usually relating to dissatisfaction or loss of interest in the sexual partner. In cases of marital discord, significant assistance may be obtained by counseling given by a health professional trained in the field. Cases of dissatisfaction by both partners often do not respond to such therapy, and frequently culminate in separation, finding a new sexual partner, and divorce.

Prevention

Unfortunately, it is difficult or impossible to predict the occurrence of HSDD in situational cases that comprise the majority of patients. The patience, understanding and support of the sexual partner is essential in those cases of HSDD in which the cause is temporary or transient. Some therapists recommend a period of abstinence from genital sex and have emphasized the value of a period of concentration on non-genital sex in the treatment of HSDD.

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Ralph Myerson, M.D.

Hypochondriasis

Definition

The primary feature of hypochondriasis is excessive fear of having a serious disease. These fears are not relieved when a medical examination finds no evidence of disease. People with hypochondriasis are often able to acknowledge that their fears are unrealistic, but this intellectual realization is not enough to reduce their anxiety. In order to qualify for a **diagnosis** of hypochondriasis, preoccupation with fear of disease must cause a great deal of distress or interfere with a person's ability to perform important activities, such as work, school activities, or family and social responsibilities. Hypochondriasis is included in the category of somatoform disorders in the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*), which is the reference handbook that clinicians use to guide the diagnosis of mental disorders. Some experts, however, have argued that hypochondriasis shares many features with **obsessive-compulsive disorder** or **panic disorder** and would be more appropriately classified with the anxiety disorders.

Description

The fears of a person with hypochondriasis may be focused on the possibility of a single illness, but more often they include a number of possible conditions. The focus of the fears may shift over time as a person notices

a new symptom or learns about an unfamiliar disease. The fears appear to develop in response to minor physical abnormalities, like **fatigue**, aching muscles, a mild cough or a small sore. People with hypochondriasis may also interpret normal sensations as signs of disease. For instance, an occasional change in heart rate or a feeling of dizziness upon standing up will lead a person with hypochondriasis to fears of heart disease or **stroke**. Sometimes hypochondriacal fears develop after the death of a friend or family member, or in response to reading an article or seeing a television program about a disease. Fear of illness can also increase in response to **stress**. Individuals with hypochondriasis visit physicians frequently; and when told there is nothing physically wrong, they are likely to seek a second opinion since their fears are not soothed. Their apparent distrust of their physicians' opinions can cause tensions in doctor-patient relationships, leading to the patient's further dissatisfaction with health care providers. Physicians who regularly see a patient with hypochondriasis may become skeptical about any reported symptom, increasing the danger that a real illness may be overlooked. People with hypochondriasis also run the risk of undergoing unnecessary medical tests or receiving unneeded medications. Although they are usually not physically disabled, they may take frequent sick days from work, or annoy friends and family with constant conversation or complaints about illness, reducing their ability to function effectively in some aspects of life.

KEY TERMS

Comorbid psychopathology—The presence of other mental disorders in a patient together with the disorder that is the immediate focus of therapy.

Somatoform disorders—A group of psychiatric disorders in the *DSM-IV-TR* classification that are characterized by the patient's concern with external physical symptoms or complaints. Hypochondriasis is classified as a somatoform disorder.

Causes and symptoms

Causes

AMPLIFICATION OF SENSORY EXPERIENCE. One theory suggests that people with hypochondriasis are highly sensitive to physical sensations. They are more likely than most people to pay close attention to sensations within their bodies (heart rate, minor noises in the digestive tract, the amount or taste of saliva in the mouth, etc.), which magnifies their experience of these feelings. While many people fail to notice minor discomfort as they go about their regular activities, the individual with hypochondriasis pays constant attention to inner sensations and becomes alarmed when these sensations vary in any way. This heightened scrutiny may actually increase the intensity of the sensations, and the intensity of the experience fuels fears that the sensations signal an underlying illness. Once the fears are aroused, preoccupation with the symptom increases, further enhancing the intensity of sensations. The tendency to amplify may be either temporary or chronic; it may also be influenced by situational factors, which helps to explain why hypochondriacal fears are made worse by stress or by events that appear to justify concerns about illness. Some researchers have observed that heightened sensitivity to internal sensations is also a feature of panic disorder, and have suggested that there may be an overlap between the two disorders.

DISTORTED INTERPRETATION OF SYMPTOMS. Another theory points to the centrality of dysfunctional thinking in hypochondriasis. According to this theory, the internal physical sensations of the person with hypochondriasis are not necessarily more intense than those of most people. Instead, people with hypochondriasis are prone to make catastrophic misinterpretations of their physical symptoms. They are pessimistic about the state of their physical health, and overestimate their chances of falling ill. Hypochondriasis thus represents a cognitive bias; whereas most people assume they are healthy unless there is clear evidence of disease, the person with hypochondriasis assumes he or she is sick unless given a clean bill of health. Interestingly, research suggests that people with hypochondriasis make more realistic estimations of their risk of disease than most people, and in fact underestimate their risk of illness. Most people simply underestimate their risk even more. Some studies indicate that people with hypochondriasis are more likely to have suffered frequent or serious illnesses as children, which may explain the development of a negative cognitive bias in interpreting physical sensations or symptoms.

Symptoms

The primary symptom of hypochondriasis is preoccupation with fears of serious physical illness or injury. The fears of persons with hypochondriasis have an obsessive quality; they find thoughts about illness intrusive and difficult to dismiss, even when they recognize that their fears are unrealistic. In order to relieve the anxiety that arises from their thoughts, people with hypochondriasis may act on their fears by talking about their symptoms; by seeking information about feared dis-

eases in books or on the Internet; or by "doctor-shopping," going from one specialist to another for a consultation. Others may deal with their fears through avoidance, staying away from anything that might remind them of illness or death. Persons with hypochondriasis vary in their insight into their disorder. Some recognize themselves as "hypochondriacs," but suffer anxiety in spite of their recognition. Others are unable to see that their concerns are unreasonable or exaggerated.

Demographics

According to *DSM-IV-TR*, hypochondriasis affects 1%–5% of the general population in the United States. The rates of the disorder are higher among clinical outpatients, between 2% and 7%. One recent study suggests that full-blown hypochondriasis is fairly rare, although lesser degrees of worry about illness are more common, affecting as many as 6% of people in a community sample.

Hypochondriasis can appear at any age, although it frequently begins in early adulthood. Men and women appear to suffer equally from the disorder. *DSM-IV-TR* notes that people from some cultures may appear to have fears of illness that resemble hypochondriasis, but are in fact influenced by beliefs that are traditional in their culture.

Diagnosis

Hypochondriasis is most likely to be diagnosed when one of the doctors consulted by the patient considers the patient's preoccupation with physical symptoms and concerns excessive or problematic. After giving the patient a thorough physical examination to rule out a general medical condition, the doctor will usually give him or her a psychological test that screens for anxiety or depression as well as hypochondriasis. If the results suggest a diagnosis of hypochondriasis, the patient should be referred for **psychotherapy**. It is important to note, however, that patients with hypochondriasis usually resist the notion that their core problem is psychological. A successful referral to psychotherapy is much more likely if the patient's medical practitioner has been able to relate well to the patient and work gradually toward the notion that psychological problems might be related to fears of physical illness.

Specific approaches that have been found useful by primary care doctors in bringing psychological issues to the patient's attention in nonthreatening ways include the following:

- Whenever possible, the doctor should draw connections between the patient's current physical symptoms and recent setbacks or upsetting incidents in the patient's life. For example, the patient may come in with health worries within a few days of having a problem in other areas of life, such as their car needing repairs, a quarrel with a family member, an overdue bill, etc.
- The doctor may consider asking the patient to keep a careful diary of his or her symptoms and other occurrences. This diary may be useful in guiding the patient to see patterns in his or her worries about health.
- The doctor may want to schedule the patient for regular but short appointments. It is also better to see the patient briefly than to prescribe medications in place of an appointment, because many patients with hypochondriasis abuse medications.
- Another approach is to conduct routine screening tests during a yearly physical for patients with hypochondriasis, while discouraging them from scheduling extra appointments each time they notice a minor physical problem.
- The doctor should maintain a realistic but optimistic tone in his or her conversation with the patient. He or she may wish to talk to the patient about health-related fears and clarify the differences between normal internal body sensations and serious symptoms.

In order to receive a *DSM-IV-TR* diagnosis of hypochondriasis, a person must meet all six of the following criteria:

- The person must be preoccupied with the notion or fear of having a serious disease. This preoccupation is based on misinterpretation of physical symptoms or sensations.
- Appropriate medical evaluation and reassurance that there is no illness present do not eliminate the preoccupation.
- The belief or fear of illness must not be of delusional intensity. Delusional health fears are more likely to be bizarre in nature— for instance, the belief that one's skin emits a foul odor or that food is rotting in one's intestines. The preoccupations must not be limited to a concern about appearance; excessive concerns that focus solely on defects in appearance would receive a diagnosis of body dysmorphic disorder.
- The preoccupation must have lasted for at least six months.
- The person's preoccupation with illness must not simply be part of the presentation of another disorder, including **generalized anxiety disorder**, obsessive-compulsive disorder, panic disorder, separation anxiety, major depressive episode, or another somatoform disorder.

DSM-IV-TR also differentiates between hypochondriasis with and without poor insight. Poor insight is specified when the patient does not recognize that his or her concerns are excessive or unreasonable.

Treatments

Traditionally, hypochondriasis has been considered difficult to treat. In the last few years, however, cognitive and behavioral treatments have demonstrated effectiveness in reducing the symptoms of the disorder.

Cognitive therapy

The goal of cognitive therapy for hypochondriasis is to guide patients to the recognition that their chief problem is fear of illness, rather than vulnerability to illness. Patients are asked to monitor worries and to evaluate how realistic and reasonable they are. Therapists encourage patients to consider alternative explanations for the physical signs they normally interpret as disease symptoms. Behavioral experiments are also employed in an effort to change the patient's habitual thoughts. For instance, a patient may be told to focus intently on a specific physical sensation and monitor increases in anxiety. Another behavioral assignment might ask the patient to suppress urges to talk about health-related worries with family members, then observe their anxiety level. Most people with hypochondriasis believe that their anxiety will escalate until they release it by seeking reassurance from others. In fact, anxiety usually crests and subsides in a matter of minutes. Cognitive therapy effectively reduces many symptoms of the disorder, and many improvements persist up to a year after treatment ends.

BEHAVIORAL STRESS MANAGEMENT. One study by Clark and colleagues compared cognitive therapy to behavioral stress management. This second form of therapy focuses on the notion that stress contributes to excessive worry about health. Patients were asked to identify stressors in their lives and taught stress management techniques to help them cope with these stressors. The researchers taught the patients relaxation techniques and problem-solving skills, and the patients practiced these techniques in and out of sessions. Although this treatment did not focus directly on hypochondriacal worries, it was helpful in reducing symptoms. At the end of the study, behavioral stress management appeared to be less effective than cognitive therapy in treating hypochondriasis, but a follow-up a year later found that the results of two therapies were comparable.

EXPOSURE AND RESPONSE PREVENTION. This therapy begins by asking patients to make a list of their hypochondriacal behaviors, such as checking body sensations, seeking reassurance from physicians or friends, and avoiding reminders of illness. Behavioral assignments are then developed. Patients who frequently monitor their physical sensations or seek reassurance are asked not to do so, and to allow themselves to experience the anxiety that accompanies suppression of these behaviors. Patients practice exposing themselves to anxiety until it becomes manageable, gradually reducing hypochondriacal behaviors in the process. In a study comparing exposure and response prevention to cognitive therapy, both therapies produced clinically significant results. Although cognitive therapy focuses more on thoughts and exposure therapy more on behaviors, both appear to be effective in reducing both dysfunctional thoughts and behaviors.

Prognosis

Untreated hypochondriasis tends to be a chronic disorder, although the intensity of the patient's symptoms may vary over time. *DSM-IV-TR* notes that the following factors are associated with a better prognosis: the symptoms develop quickly; are relatively mild; are associated with an actual medical condition; and are not associated with comorbid psychopathology or benefits derived from being ill.

Prevention

Hypochondriasis may be difficult to prevent in a health-conscious society, in which people are constantly exposed to messages reminding them to seek regular medical screenings for a variety of illnesses, and telling them in detail about the illnesses of celebrities and high-ranking political figures. Trendy new diagnostic techniques like full-body MRIs may encourage people with hypochondriasis to seek unnecessary and expensive medical consultations. Referring patients with suspected hypochondriasis to psychotherapy may also help to reduce their overuse of medical services.

See also Exposure treatment; Cognitive-behavioral therapy

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Danielle Barry, M.S.

Hypomanic episode *see* **Manic episode**

I

Imaginal desensitization *see* **Exposure treatment**

Imaginal exposure *see* **Exposure treatment**

Imaging studies

Definition

Imaging studies are tests performed with a variety of techniques that produce pictures of the inside of a patient's body.

Description

Imaging tests are performed using sound waves, radioactive particles, magnetic fields, or x rays that are detected and converted into images after passing through body tissues. Dyes are sometimes used as contrasting agents with x-ray tests so that organs or tissues not seen with conventional x rays can be enhanced. The operating principle of the various techniques is based on the fact that rays and particles interact differently with various types of tissues, especially when abnormalities are present. In this way, the interior of the body can be visualized and pictures are provided of normal structure and function as well as of abnormalities. In the fields pertaining to mental health including psychology and psychiatry, imaging is often used to help rule out other health problems that could be causing symptoms (such as **brain** tumors), and imaging studies are often used in research. Once a person's **diagnosis** has been established, various imaging techniques may help to confirm the diagnosis, and also serve as a way to study the disorder. The imaging techniques may shed new light on the way the disorder affects the brain, so that new treatment methods can be discovered.

Major imaging techniques in mental health

Computed tomography scan (CT scan)

Computed tomography, or computed axial tomography (CAT), scans show a cross-section of a part of the body, such as the brain. In this technique, a thin x-ray beam is used to produce a series of exposures detected at different angles. The exposures are fed into a computer which overlaps them, yielding a single image analogous to a slice of the organ or body part being scanned. A dye is often injected into the patient so as to improve contrast and obtain images that are clearer than images obtained with regular x rays.

Magnetic resonance imaging (MRI)

Magnetic resonance imaging also produces cross-sectional images of the body, but MRI uses powerful magnetic fields instead of radiation. MRI uses a cylinder housing a magnet that will induce the required magnetic field. The patient lies on a platform inside the scanner. The magnetic field aligns the hydrogen atoms present in the tissue being scanned in a given direction. Following a burst of radio-frequency radiation, the atoms flip back to their original orientation while emitting signals that are fed into a computer for conversion into a two- or three-dimensional image. Dyes can also be injected into patients to produce clearer images.

Positron emission tomography (PET)

Positron emission tomography uses a form of sugar that contains a radioactive atom which emits particles called positrons. The positrons are absorbed to a different extent by cells varying in their metabolic rate. PET scans are especially useful for brain imaging studies and are used to illustrate the differences between brains of people without mental disorders and brains of people with mental disorders. For example, because PET scans can detect brain activity, PET scans of the brains of depressed and non-depressed persons can show

KEY TERMS

CT scan—An imaging technique that uses a computer to combine multiple x-ray images into a two-dimensional cross-sectional image.

MRI—Magnetic resonance imaging. A special imaging technique used to image internal parts of the body, especially soft tissues.

PET—Abbreviation for positron emission tomography, a highly specialized imaging technique using radioactive substances to identify active tumors, as well as nuerological disease progression.

SPECT—Abbreviation for single photon emission computerized tomography, a highly specialized imaging technique using radioactive substances used in research, and to identify neurological disorder/disease progression.

researchers where brain activity is decreased in depressed patients. Similar scans have been taken of brains affected by **schizophrenia** or **Alzheimer's disease**. Such research can help scientists discover new ways to treat these disorders.

Single photon emission computerized tomography (SPECT)

Single photon emission computerized tomography is used in research, and in diagnosing brain disorders such as Alzheimer's and Parkinson's diseases. As of 2002, research for Parkinson's disease at Harvard, for example, in the Division of Neurochemistry is focused on the diagnosis of the disease before motor control is compromised signalling the advancing degeneration. It uses a radio-labeled compound that targets key proteins responsible for regulating brain dopamine levels to determine neural changes before problems with motor symptoms begin to occur. This research is also being used to improve PET imaging in the diagnosis and consequent treatment of these neurological disorders.

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Imaging techniques *see* **Imaging studies**

Imipramine

Definition

Imipramine is a tricyclic antidepressant. It is sold under the brand name Tofranil in the United States.

Purpose

Imipramine is used to relieve symptoms of depression.

Imipramine is also used in the treatment of **enuresis** (bed-wetting) in persons between the ages of six and 25.

Description

Imipramine hydrochloride was the first tricyclic antidepressant to be discovered. Tricyclic antidepressants act to change the balance of naturally occurring chemicals in the **brain** called **neurotransmitters** that regulate the transmission of nerve impulses between cells. Mental well-being is partially dependent on maintaining the correct balance between these brain chemicals. Imipramine is thought to act primarily by increasing the concentration of norepinephrine and serotonin (both chemicals that stimulate nerve cells) and, to a lesser extent, by blocking the action of another brain chemical, acetylcholine. Imipramine shares most of the properties of other tricyclic antidepressants, such as **amitriptyline**, **amoxapine**, **clomipramine**, **desipramine**, **nortriptyline**, **protriptyline**, and **trimipramine**.

The therapeutic effects of imipramine, like other antidepressants, appear slowly. Maximum benefit is often not evident for two to three weeks after starting the drug. People taking imipramine should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Recommended dosage

Imipramine is usually started with a total dosage of up to 100 mg per day divided into several smaller doses. This is generally increased to a total of 200 mg per day divided into several doses. Total dosages for patients who are not hospitalized should be no more than 200 mg per day. The recommended maximum dosage for the drug for all patients is 250 to 300 mg per day. Before dosages greater than 200 mg per day are taken, an electrocardiogram (EKG) should be done. This should be repeated at regular intervals until a steady state dosage is reached. Lower dosages are recommended for adolescents and for people over age 60. The lowest dosage that controls symptoms of depression should be used.

Imipramine should be withdrawn gradually, rather than abruptly discontinued. This will help reduce the possibility of a relapse into depression.

Precautions

Like all tricyclic antidepressants, imipramine should be used cautiously and with close physician supervision in people, especially the elderly, who have benign prostatic hypertrophy (enlarged prostate), urinary retention, and glaucoma, especially angle-closure glaucoma (the most severe form). Before starting treatment, people with these conditions should discuss the relative risks and benefits of treatment with their doctors to help determine if imipramine is the right antidepressant for them.

A common problem with tricyclic antidepressants is sedation (drowsiness, lack of physical and mental alertness). This side effect is especially noticeable early in therapy. In most patients, sedation decreases or disappears entirely with time, but until then patients taking imipramine should not perform hazardous activities requiring mental alertness or coordination. The sedative effect is increased when imipramine is taken with other central nervous system depressants, such as alcoholic beverages, sleeping medications, other sedatives, or antihistamines. It may be dangerous to take imipramine in combination with these substances.

Imipramine may increase heart rate and **stress** on the heart. It may be dangerous for people with cardiovascular disease, especially those who have recently had a heart attack, to take this drug or other antidepressants in the same pharmacological class. Older people and persons with a history of heart disease may develop heart arrhythmias (irregular heartbeat), heart conduction abnormalities, congestive heart failure, heart attack, abnormally rapid heart rates and strokes.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Anticonvulsant—A medication used to control abnormal electrical activity in the brain that causes seizures.

Electrocardiogram—(EKG) A test that measures the electrical activity of the heart as it beats. An abnormal EKG can indicate possible cardiac disease.

Enuresis—The inability to control urination; bedwetting.

Hypertension—High blood pressure, often brought on by smoking, obesity, or other causes; one of the major causes of strokes.

Manic—Referring to mania, a state characterized by excessive activity, unwarranted euphoria, excitement or emotion.

Methylphenidate—A mild central nervous system stimulant that is used to treat hyperactivity.

Orthostatic hypotension—A sudden decrease in blood pressure due to a change in body position, as when moving from a sitting to standing position.

Tachycardia—A pulse rate above 100 beats per minute.

Until a therapeutic dosage has been determined, people starting imipramine should be closely watched for signs of **suicide**. The risk of suicide is increased when imipramine is taken in overdose or combined with alcohol.

Manic episodes and the emergence of symptoms of pre-existing psychotic states have been reported when imipramine therapy is started.

Side effects

Imipramine shares side effects common to all tricyclic antidepressants. The most frequent of these are dry mouth, constipation, urinary retention, increased heart rate, sedation, irritability, dizziness, and decreased coordination. As with most side effects associated with tricyclic antidepressants, the intensity is highest at the beginning of therapy and tends to decrease with continued use.

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Patients may also chew sugarless gum or suck on sugarless candy in order to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Imipramine usage has been linked to both increases and decreases in blood pressure and heart rate. Heart attacks, congestive heart failure, and strokes have been reported.

Confusion, disorientation, **delusions**, **insomnia**, and anxiety have also been reported as side effects in a small percentage of people taking imipramine. Problems associated with the skin (loss of sensation, numbness and tingling, rashes, spots, itching and puffiness), **seizures**, and ringing in the ears have also been reported. Nausea, vomiting, loss of appetite, diarrhea, and abdominal cramping are all side effects associated with imipramine usage in a small number of people.

Interactions

Methylphenidate may increase the effects of imipramine. This is usually avoided by reducing the dosage of imipramine.

Imipramine may increase the depressant action of alcohol. For this reason, persons taking imipramine should not drink alcoholic beverages.

Dangerously high blood pressure has resulted from the combination of tricyclic antidepressants, such as imipramine, and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Because of this, imipramine should never be taken in combination with MAO inhibitors. Patients taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting imipramine or any other tricyclic antidepressant. The same holds true when discontinuing imipramine and starting an MAO inhibitor.

The sedative effects of imipramine are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or medications used for other mental disorders such as **schizophrenia**. The anticholinergic (drying out) effects of imipramine are additive with other anticholinergic drugs such as **benztropine**, **biperiden**, **trihexyphenidyl**, and antihistamines.

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax: (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

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Impulse-control disorders

Definition

Impulse-control disorders are psychological disorders characterized by the repeated inability to refrain from performing a particular action that is harmful either to oneself or others.

Description

Impulse-control disorders are thought to have both neurological and environmental causes and are known to be exacerbated by **stress**. Some mental health professionals regard several of these disorders, such as compulsive gambling or shopping, as addictions. In impulse-control disorder, the impulse action is typically preceded by feelings of tension and excitement and followed by a sense of relief and gratification, often—but not always—accompanied by guilt or remorse.

The Fourth Edition Text Revision of the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*, a handbook that mental health professionals use to diagnose mental disorders) describes several impulse-control disorders:

- Pyromania. This disorder is diagnosed when a person has deliberately started fires out of an attraction to and curiosity about fire. In order to meet the criteria for this **diagnosis**, the firestarter cannot seek monetary gain or be trying to destroy evidence of criminal activity, or be trying to make a political statement or improve one's standard of living.
- Trichotillomania. This disorder is characterized by compulsive hair-pulling.
- Intermittent explosive disorder. This diagnosis is indicated when a person cannot resist aggressive impulses that lead to serious acts of assault or property destruction.
- Kleptomania. The recurrent failure to resist the urge to steal, even though the items stolen are not needed for personal use or for their monetary value, is required for diagnosis of this disorder.
- Pathological gambling. This form of persistent gambling disrupts the affected individual's relationships or career.
- Impulse-control disorders not otherwise specified. This category is reserved for clinicians' use when the clinician has established that a patient's disorder is caused by lack of impulse control, but does not meet the criteria for the disorders listed above or the criteria for any other disorder listed in the *DSM-IV-TR*.

A condition not listed in the *DSM-IV-TR* that some experts consider an impulse-control disorder is repetitive self-mutilation, in which people intentionally harm themselves by cutting, burning, or scratching their bodies. Other forms of repetitive self-mutilation include sticking oneself with needles, punching or slapping the face, and swallowing harmful substances. Self-mutilation tends to occur in persons who have suffered traumas early in life, such as sexual **abuse** or the death of a parent, and often has its onset at times of unusual stress. In many cases, the triggering event is a perceived rejection by a parent or romantic interest. Characteristics commonly seen in persons with this disorder include perfectionism, dissatisfaction with one's physical appearance, and difficulty controlling and expressing emotions. It is often seen in conjunction with **schizophrenia**, post-traumatic stress syndrome, and various **personality disorders**. Usual onset is late childhood or early adolescence; it is more frequent in females than in males.

Those who consider self-mutilation an impulse-control disorder do so because, like the other conditions that fall into this category, it is a habitual, harmful activity. Victims often claim that the behavior is accompanied by feelings of excitement and that it reduces or relieves negative feelings such as tension, anger, anxiety, depression, and loneliness. They also describe it as addictive. Self-mutilating behavior may occur in episodes, with periods of remission, or may be continuous over a number of years. Repetitive self-mutilation often worsens over time, resulting in increasingly serious forms of injury that may culminate in **suicide**.

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Inderal *see* **Propranolol**

Informed consent

Definition

Informed consent is a legal document in all 50 states, prepared as an agreement for treatment, non-treatment, or for an invasive procedure that requires physicians to

disclose the benefits, risks, and alternatives to the treatment, non-treatment, or procedure. It is the method by which a fully informed, rational patient may be involved in the choices about his or her health. Informed consent applies to mental health practitioners (psychiatrists, psychologists, etc.) in their treatment with their clients in generally the same way as physicians with their patients.

Description

Informed consent stems from the legal and ethical right the patient has to decide what is done to his or her body, and from the mental health provider's ethical duty to ensure that the patient is involved in decisions about his or her own health care. The process of ensuring informed consent for treatment involves three phases, all of which involve information exchange between doctor and patient and are a part of patient education. First, in words the patient can understand, the therapist must convey the details of a treatment or procedure, its potential benefits and serious risks, and any feasible alternatives. The patient should be presented with information on the most likely outcomes of treatment. Second the practitioner must evaluate whether or not the person has understood what has been said, must ascertain that the risks have been accepted, and that the patient is giving consent to proceed with the treatment with full knowledge and forethought. Finally, the patient must sign the consent form, which documents in generic format the major points of consideration. The only exception to this is securing informed consent during extreme emergencies. It is critical that the patient receive enough information on which to base informed consent, and that the consent is wholly voluntary and has not been forced in any way.

According to the Ethical Principles of Psychologists and Code of Conduct designed by the American Psychological Association, informed consent also applies when conducting research involving human subjects prior to their participation. Participants in the study should be informed in understandable language to three main points. First, the participant should be informed about the nature of the research. Secondly, participants should be informed that their participation is completely voluntary and that they are free to withdraw from or not participate in the study at any time. Consent must be made without pressure being put on the participant to engage in the study. Finally, the potential consequences of participating or withdrawing should be presented to the participant. This includes risks, discomfort, and limitations of confidentiality.

With regard to either therapy treatment or research participation, another member of the health care/research team may obtain the signed informed consent with the assurance that the provider has satisfied the requirements of informed consent.

The actual informed consent form is to document the process and protect the provider and the hospital. Legally, it is proof that things have been covered and the patient agrees to the procedure, risks, benefits, options, etc. The informed consent process is in place for the protection of the patient. The process is in place to ensure that everything is discussed with the patient: all of the options, all of the common risks, the worst case scenario, and other similar situations.

Viewpoints

There is a theory that the practice of acquiring informed consent is rooted in the post-World War II Nuremberg Trials. Following the war crimes tribunal in 1949, as a result of the Kaarl Brandt case, 10 standards were put forth regarding physician's requirements for experimentation on human subjects. This established a new standard of ethical medical behavior for the post-WW II human rights age, and the concept of voluntary informed consent was established. A number of rules accompanied voluntary informed consent within the realms of research. It could only be requested for experimentation for the gain of society, for the potential acquisition of knowledge of the pathology, and for studies performed that avoided physical and mental suffering to the fullest extent possible..

A crucial component of informed consent is that the person signing it is competent or able to make a rational decision and meaningfully give consent. This situation gets more complicated when working with people who are unable to understand what has been explained or are unable to make a reasonable decision about their health care. According to the Code of Conduct for Psychologists designed by the American Psychological Association, if this is the case, informed permission from a "legally authorized person" should then be sought, if that is a legal alternative. The ethical guidelines are more stringent than legal guidelines in many states, where the informed consent of the parent or guardian is all that is required, whether or not the professional has attempted to explain the procedure to the client.

Although it is necessary to present the procedure or treatment formally to the patient, there is concern that this process could hurt the therapeutic relationship between the client and therapist. For example, if an informed consent is too detailed, it could frighten a new client who may be hesitant about therapy to begin with. In addition, informing patients about the risks of treatment might scare them into refusing it when the risks of non-treatment are even greater. There are however,

advantages to the informed consent process. First, it can be empowering to the patient to understand that he/she plays an important role in their own treatment. They are encouraged to be active participants in the treatment process and know their options well enough to make the best treatment decisions for themselves. This also shifts the responsibility to patients to work with the therapist towards their mental health goals, possibly increasing self-confidence and autonomy, and decreasing dependence on the therapist.

Professional implications

There are undoubtedly many issues regarding Informed Consent. As modern society continues to be litigious, the courts and/or government may take on a more active role in deciding the extent to which patients must be informed of treatments, procedures, and clinical trials in which they voluntarily become enrolled. Therefore, health care providers must become more educated as to what needs to be conveyed to patients, and to what extent.

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Inhalants and related disorders

Definition

The inhalants are a class of drugs that include a broad range of chemicals found in hundreds of different products, many of which are readily available to the general population. These chemicals include volatile solvents (liquids that vaporize at room temperature) and aerosols (sprays that contain solvents and propellants). Examples include glue, gasoline, paint thinner, hair spray, lighter fluid, spray paint, nail polish remover, correction fluid, rubber cement, felt-tip marker fluids, vegetable sprays, and certain cleaners. The inhalants share a common route of administration— that is, they are all drawn into the body by breathing. They are usually taken either by breathing in the vapors directly from a container (known as "sniffing"); by inhaling fumes from substances placed in a bag (known as "bagging"); or by inhaling the substance from a cloth soaked in it (known as "huffing"). Inhalants take effect very quickly because they get into the bloodstream rapidly via the lungs. The "high" from inhalants is usually brief, so that users often take inhalants repeatedly over several hours. This pattern of use can be dangerous, leading to unconsciousness or even death.

The latest revision of the manual that is used by mental health professionals to diagnose mental disorders is the ***Diagnostic and Statistical Manual of Mental Disorders*** published in 2000 (also known as *DSM-IV-TR*). It lists inhalant dependence and inhalant abuse as substance use disorders. In addition, the inhalant-induced disorder of inhalant intoxication is listed in the substance-related disorders section as well. Inhalant withdrawal is not listed in the *DSM-IV-TR* because it is not clear that there is a "clinically significant" withdrawal syndrome. In addition, withdrawal is not included as a symptom of inhalant dependence, whereas withdrawal is a symptom of dependence for all other substances. Withdrawal symptoms are symptoms that occur when a person who is dependent on a substance stops using the substance.

KEY TERMS

Aerosol—A liquid substance sealed in a metal container under pressure with an inert gas that propels the liquid as a spray or foam through a nozzle.

Euphoria—A feeling or state of well-being or elation.

Gateway drug—A mood-altering drug or substance, typically used by younger or new drug users, that may lead to the use of more dangerous drugs.

Nystagmus—A persistent involuntary movement of the eyes from side to side. It is one of the symptoms of inhalant intoxication syndrome.

Sudden sniffing death—Death resulting from heart failure caused by heavy use of inhalants in a single session.

Syndrome—A group of symptoms that together characterize a disease or disorder.

Volatile solvent—A solvent (substance that will dissolve another substance) that evaporates at room temperature.

Anesthetic gases (such as nitrous oxide, chloroform, or ether) and nitrites (including amyl or butyl nitrite) are not included under inhalant-related disorders in the *DSM-IV-TR* because they have slightly different intoxication syndromes. Problems with the use of these substances are to be diagnosed under "Other Substance-Related Disorders." There is, however, a significant degree of overlap between the symptoms of disorders related to inhalants and these "other" substances.

Inhalant dependence

Inhalant dependence, or **addiction**, is essentially a syndrome in which a person continues to use inhalants in spite of significant problems caused by or made worse by the use of these substances. People who use inhalants heavily may develop tolerance to the drug, which indicates that they are physically dependent on it. Tolerance is the reduced response to a drug that develops with repeated use.

Inhalant abuse

Inhalant abuse is a less serious condition than inhalant dependence; in most cases, it does not involve physical dependence on the drug. Inhalant abuse refers essentially to significant negative consequences from the recurrent use of inhalants.

Inhalant intoxication

When a person uses enough of an inhalant, they will get "high" from it. The symptoms of intoxication differ slightly depending on the type of inhalant, the amount used, and other factors. There is, however, a predictable set of symptoms of inhalant intoxication. When too much of the substance is taken, an individual can overdose.

Description

Inhalant dependence

Dependence on inhalants involves problems related to the use of inhalants. It is often difficult for a person to stop using the inhalants despite these problems. Individuals dependent on inhalants may use these chemicals several times per week or every day. They may have problems with unemployment, with family relationships, and/or such physical problems as kidney or liver damage caused by the use of inhalants.

Inhalant abuse

People who abuse inhalants typically use them less frequently than those who are dependent on them. Despite less frequent use, however, a person with inhalant abuse suffers negative consequences. For example, the use of inhalants may contribute to poor grades or school truancy.

Inhalant intoxication

Intoxication from inhalants occurs rapidly (usually within five minutes) and lasts for a short period of time (from five to 30 minutes). Inhalants typically have a depressant effect on the central nervous system, similar to the effects of alcohol; and produce feelings of euphoria (feeling good), excitement, dizziness, and slurred speech. In addition, persons intoxicated by inhalants may feel as if they are floating, or feel a sense of increased power. Severe intoxication from inhalants can cause coma or even death.

Causes and symptoms

Causes

Because inhalants are readily available and inexpensive, they are often used by children (ages six to 16) and the poor. Factors that are associated with inhalant use include poverty; a history of childhood abuse; poor grades; and dropping out of school. The latter two factors may simply be a result of inhalant use, however, rather than its cause.

The use of inhalants is highly likely to be influenced by peers. Inhalants are often used in group settings. The solitary consumption of inhalants is associated with heavy, prolonged use; it may indicate that the person has a more serious problem with these substances.

Symptoms

INHALANT DEPENDENCE. The *DSM-IV-TR* specifies that three or more of the following symptoms must occur at any time during a 12-month period (and cause significant impairment or distress) in order to meet diagnostic criteria for inhalant dependence:

- Tolerance. The individual either has to use increasingly higher amounts of the drug over time in order to achieve the same effect, or finds that the same amount of the drug has much less of an effect over time than before. After using inhalants regularly for a while, people may find that they need to use at least 50% more than the amount they started with in order to get the same effect.
- Loss of control. The person either repeatedly uses a larger quantity of inhalant than planned, or uses the inhalant over a longer period of time than planned. For instance, someone may begin using inhalants on school days, after initially limiting their use to weekends.
- Inability to stop using. The person has either unsuccessfully attempted to cut down or stop using the inhalants, or has a persistent desire to stop using. Users may find that despite efforts to stop using inhalants on school days, they cannot stop.
- Time. The affected person spends large amounts of time obtaining inhalants, using them, being under the influence of inhalants, and recovering from their effects. Obtaining the inhalants might not take up much time because they are readily available for little money, but the person may use them repeatedly for hours each day.
- Interference with activities. The affected person either gives up or reduces the amount of time involved in recreational activities, social activities, and/or occupational activities because of the use of inhalants. The person may use inhalants instead of playing sports, spending time with friends, or going to work.
- Harm to self. The person continues to use inhalants in spite of developing either a physical (liver damage or heart problems, for example) or psychological problem (such as depression or memory problems) that is caused by or made worse by the use of inhalants.

INHALANT ABUSE. The *DSM-IV-TR* specifies that one or more of the following symptoms must occur at any time during a 12-month period (and cause significant impairment or distress) in order to meet diagnostic criteria for inhalant abuse:

- Interference with role fulfillment. The person's use of inhalants frequently interferes with his or her ability to fulfill obligations at work, home, or school. People may find they are unable to do chores or pay attention in school because they are under the influence of inhalants.
- Danger to self. The person repeatedly uses inhalants in situations in which their influence may be physically hazardous (while driving a car, for example).
- Legal problems. The person has recurrent legal problems related to using inhalants (such as arrests for assaults while under the influence of inhalants).
- Social problems. The person continues to use inhalants despite repeated interpersonal or relationship problems caused by or made worse by the use of inhalants. For example, the affected person may get into arguments related to inhalant use.

INHALANT INTOXICATION. The *DSM-IV-TR* specifies that the following symptoms must be present in order to meet diagnostic criteria for inhalant intoxication:

- Use. The person recently intentionally used an inhalant.
- Personality changes. The person experiences significant behavioral or psychological changes during or shortly after use of an inhalant. These changes may include spoiling for a fight; assaultiveness; poor judgment; **apathy** ("don't care" attitude); or impaired functioning socially or at work or school.
- Inhalant-specific intoxication syndrome. Two or more of the following symptoms occur during or shortly after inhalant use or exposure: dizziness; involuntary side-to-side eye movements (nystagmus); loss of coordination; slurred speech; unsteady gait (difficulty walking); lethargy (**fatigue**); slowed reflexes; psychomotor retardation (moving slowly); tremor (shaking); generalized muscle weakness; blurred vision or double vision; stupor or coma; and euphoria (a giddy sensation of happiness or well-being).

Demographics

Inhalants are one of the few substances more commonly used by younger children rather than older ones. It has been estimated that 10%–20% of youths aged 12–17 have tried inhalants. About 6% of the United States population admits to having tried inhalants prior to fourth grade. The peak time for inhalant use appears to be between the seventh and ninth grades. Inhalants are sometimes referred to as "gateway" drugs, which means that they are one of the first drugs that people try before moving on to such other substances as alcohol, marijuana, and cocaine. Only a small proportion of those who have used inhalants would meet diagnostic criteria for dependence or abuse.

Samples of some hazardous substances that might be abused as inhalants, including nail polish remover, aerosols, paint thinner, and glue. *(Custom Medical Stock Photo, Inc. Reproduced by permission.)*

Males generally use inhalants more frequently than females. However, a National Household Survey on Drug Abuse has shown no gender differences in rates of inhalant use in youths between the ages of 12 and 17. Children younger than 12 and adults who use inhalants, however, are more likely to be male.

Diagnosis

People rarely seek treatment on their own for inhalant dependence or abuse. In some cases, the child or adolescent is brought to a doctor by a parent or other relative who is concerned about personality changes, a chemical odor on the child's breath, or other signs of inhalant abuse. The parent may also have discovered empty containers of the inhaled substance in the child's room or elsewhere in the house. In other cases, the child or adolescent's use of inhalants is diagnosed during a medical interview, when he or she is brought to a hospital emergency room after overdosing on the inhalant or being injured in an accident related to inhalant use. Although inhalants can be detected in blood or urine samples, laboratory tests may not always confirm the **diagnosis** because the inhalants do not remain in the system very long.

Inhalant dependence

Other substance use disorders are commonly seen among people diagnosed with inhalant dependence. The use of inhalants is usually secondary to the use of other substances, however; only occasionally are inhalants a person's primary drug of choice.

Inhalant abuse

The use of other substances is not uncommon among people who abuse inhalants.

Inhalant intoxication

Intoxication from the use of such other substances as alcohol, sedatives, hypnotics (medications to induce sleep), and anxiolytics (tranquilizers) can resemble intoxication caused by inhalants. Furthermore, people under the influence of inhalants may experience **hallucinations** (typically auditory, visual, or tactile); other perceptual disturbances (such as illusions); or **delusions** (believing they can fly, for example).

Treatments

Inhalant dependence and abuse

Chronic inhalant users are difficult to treat because they often have many serious personal and social problems. They also have difficulty staying away from inhalants; relapse rates are high. Treatment usually takes a long time and involves enlisting the support of the person's family; changing the friendship network if the individual uses with others; teaching coping skills; and increasing self-esteem.

Inhalant intoxication

Inhalant intoxication is often treated in a hospital emergency room when the affected person begins to suffer serious psychological (such as hallucinations or delusions) or medical consequences (difficulty breathing, headache, nausea, vomiting) from inhalant use. The most serious medical risk from inhalant use is "sudden sniffing death." A person using inhalants, especially if they are using the substance repeatedly in a single, prolonged session, may start to have a rapid and irregular heartbeat or severe difficulty breathing, followed by heart failure and death. Sudden sniffing death can occur within minutes. In addition, inhalant use can cause permanent damage to the **brain**, lung, kidney, muscle, and heart. The vapors themselves cause damage, but there are also dangerously high levels of copper, zinc, and heavy metals in many inhalants.

People who use inhalants may also be treated for injuries sustained while under the influence of inhalants or while using inhalants. For example, individuals intoxicated by inhalants may fall and injure themselves, or they may drive while intoxicated and have an accident. People who use inhalants may also die from or require treatment for burns because many inhalants are highly flammable. They may also need emergency treatment for suffocation from inhaling with a plastic bag over the head, or for choking on inhaled vomit.

Prognosis

Inhalant dependence and abuse

The course of inhalant abuse and dependence differs somewhat depending on the affected person's age. Younger children who are dependent on or abuse inhalants use them regularly, especially on weekends and after school. As children get older, they often stop using inhalants. They may stop substance use altogether or they may move on to other substances. Adults who abuse or are dependent on inhalants may use inhalants regularly for years. They may also frequently "binge" on inhalants (i.e., using them much more frequently for shorter periods of time). This pattern of use can go on for years.

The use of inhalants and subsequent dependence on the substance occurs among people who do not have access to other drugs or are otherwise isolated (such as prison inmates). Also, as with other substance use disorders, people who have greater access to inhalants are more likely to develop dependence on them. This group of people may include workers in industrial settings with ready access to inhalants.

Prevention

Comprehensive prevention programs that involve families, schools, communities, and the media (such as television) can be effective in reducing substance abuse. The recurring theme in these programs is to stay away from drugs in the first place, which is the primary method of ensuring that one does not develop a substance use disorder.

Parents can help prevent the misuse of inhalants by educating their children about the negative effects of inhalant use. Both teachers and parents can help prevent inhalant abuse and dependence by recognizing the signs of inhalant use, which include chemical odors on the child's breath or clothes; slurred speech; a drunken or disoriented appearance; nausea or lack of appetite; and inattentiveness and lack of coordination.

See also Polysubstance abuse

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American Psychological Association. 750 First Street, NE, Washington, DC 20002-4242. (800) 374-2721. <http://www.apa.org>.

National Clearinghouse for Alcohol and Drug Information. (800) 729-6686. <http://www.health.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

National Institute on Drug Abuse (NIDA). 5600 Fishers Lane, Room 10-05, Rockville, MD 20857. Nationwide Helpline: (800) 662-HELP. <http://www.nida.nih.gov>.

National Library of Medicine. 8600 Rockville Pike, Bethesda, MD 20894. <http://www.nlm.nih.gov/medlineplus/drugabuse.html>.

Jennifer Hahn, Ph.D.

Insomnia

Definition

Insomnia is a condition that occurs when a person in unable to get long enough or refreshing enough sleep at night. Insomnia can result from an inability to fall asleep, an inability to stay asleep, or waking too early before having gotten enough sleep.

Description

Insomnia is a disorder in which people are unable to get enough, or enough restorative, sleep because of one or more factors. People with insomnia often have daytime symptoms related to a lack of sleep, such as daytime sleepiness, **fatigue**, and decreased mental clarity.

There are two main types of insomnia. One is acute insomnia (sometimes called transient insomnia). This type occurs when insomnia symptoms exist over a reasonably short period of time. The other type is chronic insomnia, which is diagnosed when the symptoms manifest themselves over a longer period (generally more than one month). Insomnia can also be classified as either primary or secondary. Primary insomnia is a disorder that cannot be attributed to another condition or disorder. Secondary insomnia can be traced back to a source, which may be a medical condition; the use of medications, alcohol, or other substances; or a mental disorder such as severe depression.

KEY TERMS

Hypnotic—A type of medication that induces sleep.

Not all disruptions in the normal pattern of sleeping and waking are considered insomnia. Such factors as jet lag, unusually high levels of **stress**, changing work shifts, or other drastic changes in the person's routine can all lead to sleep problems. Unless the problems are ongoing and severe enough that they are causing distress for the person in important areas of life, he or she is not considered to have insomnia.

Causes and symptoms

The symptoms of insomnia can vary greatly from person to person. Some people find that they have trouble falling asleep at night and can lie in bed for hours without being able to drift off. Others find that they fall asleep easily but wake many times during the night. Other people awaken too early in the morning and are then unable to get back to sleep. Some people even get enough hours of sleep but find that they do not feel rested, often because their sleep is too light.

Not all people experiencing insomnia have symptoms that occur during the daytime, but many do. Some people experience such symptoms as reduced ability to concentrate or pay attention, decreased alertness, and mental sluggishness. Some people have trouble staying awake. More people think that they have this symptom than actually do. Upon clinical examination many people who think that they are excessively sleepy during the day actually are not.

Many different things are thought to cause or contribute to insomnia. Such stressors as starting a new job or changes in routine, such as beginning to work a different shift, can lead to temporary sleep problems. Sleep problems can become aggravated, and persist after the worry or change causing the sleep problem has been resolved. This persistence is thought to be related to the anxiety created by attempting to go to sleep and not expecting to fall asleep. Anxiety about sleep loss can lead to a vicious circle in which the person has more and more concern about being able to fall asleep, making it ever increasingly difficult to do so. Some people even report that they are better able to fall asleep when they are not in their beds. This relative success is thought to occur because the new environment is not associated with the fear and anxiety of not being able to sleep, therefore making it easier to fall asleep.

Many other factors are thought to lead to or perpetuate insomnia. These include drinking tea or coffee, eating a large meal, taking certain medications or drugs of abuse (cocaine, **amphetamines**) that have a stimulating effect, or exercising heavily in the hours before attempting to sleep. Also, attempting to sleep in a room with too much light or noise can make it harder for some people to sleep. Doing activities in bed that are not associated with sleep, such as reading or watching television, can make it more difficult for some people to fall asleep when they finally want to. Sleep may be even more difficult if the television show or book was frightening or upsetting.

Demographics

There are many different opinions about how much of the general American population experiences insomnia. Estimates suggest that around 5–20% of the adult population suffers from some form of insomnia or long-term sleeping problems. Nearly half report at least occasional sleeping problems. Accurate data is difficult to gather, as many people misperceive how much sleep they actually get and how many times they normally wake up during the night. It is generally agreed, however, that women are more likely than men to suffer from insomnia. As people get older, they are also are more likely to experience insomnia. People who are nervous or tense are more likely to have insomnia than those who are not. Lastly, people who live near airports or other sources of nighttime as well as daytime noise have higher rates of insomnia than the general population.

Diagnosis

According to the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*), which presents the guidelines used by the American Psychiatric Association for **diagnosis** of disorders, in order to be diagnosed with primary insomnia, a person must experience the symptoms for at least a month, and the symptoms must cause them distress or reduce their ability to function successfully. The symptoms cannot be caused by a different sleep disorder, a medical condition, or be a side effect of medications or substance abuse.

Insomnia may also be comorbid with (occur together with) other psychiatric disorders, including mania, depression, and the anxiety disorders.

Insomnia is a disorder that is usually self-reported; that is, patients usually bring up the subject of sleep

problems with their doctors rather than the doctor suggesting the diagnosis. There are no laboratory tests for insomnia, but the doctor may suggest keeping a sleep diary, in which the patient notes the time they went to bed, the time(s) at which they got up during the night, their activities before bed, etc. Sleep diaries can be helpful in uncovering specific factors related to the insomnia.

Treatments

Many treatments have been explored for treating insomnia, in a number of different settings. The patient may wish to consider consulting a sleep clinic or a doctor who specializes in the treatment of **sleep disorders** as well as their family doctor.

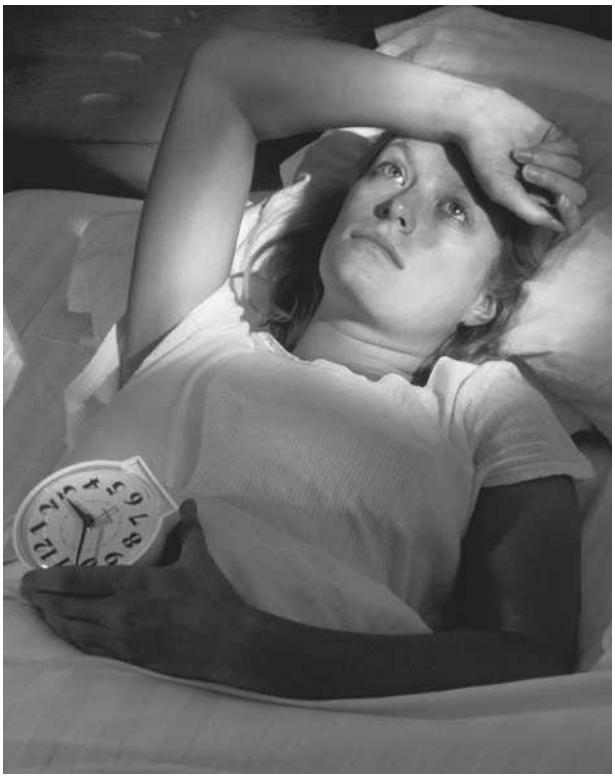
Behavioral and educational therapies are usually tried first, because they do not have side effects and cannot create a chemical dependence the way some sleep medications can. Many different approaches have been designed to help patients whose insomnia is linked to particular factors.

Behavioral treatments

One common behavioral therapy involves changing any pre-bedtime activities or behaviors that might interfere with sleep. Avoiding large meals, alcohol or caffeinated beverages, or intensive exercise in the hours before bedtime may help the patient to fall asleep.

Another non-medicinal treatment for insomnia involves controlling the patient's mental associations with the bedroom. The patient is trained to associate the bed only with sleep, not with the frustration of trying to fall asleep or with such waking activities as reading or watching television. As part of this training, if the patient cannot sleep after a certain amount of time, he or she is instructed to get out of bed and spend time somewhere else in the house doing an activity that they find relaxing. The patient lies down again only when sleepy. This technique helps to prevent frustration from trying to sleep.

Another common technique that does not involve medication is sleep restriction therapy. During this therapy, the amount of time that patients are allowed to spend in bed is limited to only slightly more time than they believe that they already sleep at night. Gradually the amount of time patients are allowed to spend in bed is increased until they are getting a full night's sleep. Unfortunately, many people find this treatment difficult to stick with, because they often become mildly sleep-deprived. The resultant fatigue can be useful, however, as it may help them fall asleep more easily and to stay asleep longer at night.



People with insomnia do not get enough restorative sleep, and often have daytime symptoms such as daytime sleepiness, fatigue, and decreased mental clarity. *(Photo by David Pollack. Corbis Stock Market. Reproduced by permission.)*

Teaching relaxation and the ability to concentrate on relaxing thoughts or images can also help patients experiencing insomnia. Most of these therapies also include setting times for waking and having the patient stick to them even if he or she has not gotten a full night of sleep. The elimination of all daytime napping can help to facilitate sleep at night. These treatments are effective by themselves but may also be combined with other approaches. The course of treatment depends on the patient's specific symptoms.

Treatment with medications

Many different medicines, which are called hypnotics, are used to treat insomnia. These are usually not recommended for use for longer than a week because they may cause dependence. In addition, there is always the risk of side effects. There are many different types of hypnotics, and choosing one for a patient depends on the patient's symptoms, other drugs that he or she may be taking, any medical or psychological conditions, and

other health factors. Medication treatment is best used in coordination with a behavioral therapy program.

Alternative remedies

Alternative remedies for insomnia, particularly herbal preparations, should be mentioned because they are among the most popular nonprescription treatments for sleep problems. According to *Prevention* magazine, insomnia is the sixth most common condition treated with herbal formulas in the United States; it accounts for 18% of all use of herbal preparations. Some herbs used for insomnia are safer than others. Persons who are using alternative remedies, whether to treat insomnia or other conditions, *should always tell their doctor what they are taking, how much, and how often*. This warning is important because some herbal preparations that are safe in themselves can interact with prescription medications.

Prognosis

Untreated insomnia has potentially serious consequences, including an increased risk of motor vehicle accidents, impaired school or job performance, and a high rate of absenteeism from work. Fortunately, insomnia can be treated very effectively in most patients. Treatment using a combination of approaches is usually most effective. Patients who have had insomnia once are at an increased risk for recurrent insomnia.

See also Caffeine and related disorders; Chamomile; Passionflower; Valerian

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Tish Davidson, A.M.

Intake evaluation *see* **Assessment and diagnosis**

Intelligence tests

Definition

Intelligence tests are psychological tests that are designed to measure a variety of mental functions, such as reasoning, comprehension, and judgment.

Purpose

The goal of intelligence tests is to obtain an idea of the person's intellectual potential. The tests center around a set of stimuli designed to yield a score based on the test maker's model of what makes up intelligence. Intelligence tests are often given as a part of a battery of tests.

Precautions

There are many different types of intelligence tests and they all do not measure the same abilities. Although the tests often have aspects that are related with each other, one should not expect that scores from one intelligence test, that measures a single factor, will be similar to scores on another intelligence test, that measures a variety of factors. Also, when determining whether or not to use an intelligence test, a person should make sure that the test has been adequately developed and has solid research to show its reliability and validity. Additionally, psychometric testing requires a clinically trained examiner. Therefore, the test should only be administered and interpreted by a trained professional.

A central criticism of intelligence tests is that psychologists and educators use these tests to distribute the limited resources of our society. These test results are used to provide rewards such as special classes for gifted students, admission to college, and employment. Those who do not qualify for these resources based on intelligence test scores may feel angry and as if the tests are denying them opportunities for success. Unfortunately, intelligence test scores have not only become associated with a person's ability to perform certain tasks, but with self-worth.

Many people are under the false assumption that intelligence tests measure a person's inborn or biological intelligence. Intelligence tests are based on an individual's interaction with the environment and never exclusively measure inborn intelligence. Intelligence tests have been associated with categorizing and stereotyping people. Additionally, knowledge of one's performance on an intelligence test may affect a person's aspirations and motivation to obtain goals. Intelligence tests can be culturally biased against certain groups.

Description

When taking an intelligence test, a person can expect to do a variety of tasks. These tasks may include having to answer questions that are asked verbally, doing mathematical problems, and doing a variety of tasks that require eye-hand coordination. Some tasks may be timed and require the person to work as quickly as possible. Typically, most questions and tasks start out easy and progressively get more difficult. It is unusual for anyone to know the answer to all of the questions or be able to complete all of the tasks. If a person is unsure of an answer, guessing is usually allowed.

The four most commonly used intelligence tests are:

- **Stanford-Binet Intelligence Scales**
- **Wechsler-Adult Intelligence Scale**
- **Wechsler Intelligence Scale for Children**
- Wechsler Primary & Preschool Scale of Intelligence

Advantages

In general, intelligence tests measure a wide variety of human behaviors better than any other measure that has been developed. They allow professionals to have a uniform way of comparing a person's performance with that of other people who are similar in age. These tests also provide information on cultural and biological differences among people.

Intelligence tests are excellent predictors of academic achievement and provide an outline of a person's mental strengths and weaknesses. Many times the scores have revealed talents in many people, which have led to an improvement in their educational opportunities. Teachers, parents, and psychologists are able to devise individual curricula that matches a person's level of development and expectations.

Disadvantages

Some researchers argue that intelligence tests have serious shortcomings. For example, many intelligence tests produce a single intelligence score. This single score is often inadequate in explaining the multidimensional aspects of intelligence. Another problem with a single score is the fact that individuals with similar intelligence test scores can vary greatly in their expression of these talents. It is important to know the person's performance on the various subtests that make up the overall intelligence test score. Knowing the performance on these various scales can influence the understanding of a person's abilities and how these abilities are expressed. For example, two people have identical scores on intelligence tests. Although both people have the same test score, one person may have obtained the score because of strong verbal skills while the other may have obtained the score because of strong skills in perceiving and organizing various tasks.

Intelligence tests are psychological tests that are designed to measure a variety of mental functions, such as reasoning, comprehension, and judgment. Intelligence tests are often given as part of a battery of tests. *(Lew Merrim/Science Source. Photo Researchers, Inc. Reproduced by permission.)*

Furthermore, intelligence tests only measure a sample of behaviors or situations in which intelligent behavior is revealed. For instance, some intelligence tests do not measure a person's everyday functioning, social knowledge, mechanical skills, and/or creativity. Along with this, the formats of many intelligence tests do not capture the complexity and immediacy of real-life situations. Therefore, intelligence tests have been criticized for their limited ability to predict non-test or nonacademic intellectual abilities. Since intelligence test scores can be influenced by a variety of different experiences and behaviors, they should not be considered a perfect indicator of a person's intellectual potential.

Results

The person's raw scores on an intelligence test are typically converted to standard scores. The standard

scores allow the examiner to compare the individual's score to other people who have taken the test. Additionally, by converting raw scores to standard scores the examiner has uniform scores and can more easily compare an individual's performance on one test with the individual's performance on another test. Depending on the intelligence test that is used, a variety of scores can be obtained. Most intelligence tests generate an overall intelligence quotient or IQ. As previously noted, it is valuable to know how a person performs on the various tasks that make up the test. This can influence the interpretation of the test and what the IQ means. The average of score for most intelligence tests is 100.

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Keith Beard, Psy.D.

Intermittent explosive disorder

Definition

Intermittent explosive disorder (IED) is a disorder characterized by impulsive acts of aggression, as contrasted with planned violent or aggressive acts. The aggressive episodes may take the form of "spells" or "attacks," with symptoms beginning minutes to hours before the actual acting-out. The ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (also known as *DSM-IV-TR*) is the basic reference work consulted by mental health professionals in determining the **diagnosis** of a mental disorder. *DSM-IV-TR* classifies IED under the general heading of "Impulse-Control Disorders Not Elsewhere Classified." Other names for IED include rage attacks, anger attacks, and episodic dyscontrol.

Description

Intermittent explosive disorder was originally described by the eminent French **psychiatrist** Esquirol as a "partial insanity" related to senseless impulsive acts. Esquirol termed this disorder *monomanies instinctives*, or *instinctual monomanias*. These apparently unmotivated acts were thought to result from instinctual or involuntary impulses, or from impulses related to ideological obsessions.

People with intermittent explosive disorder have a problem with controlling their temper. In addition, their violent behavior is out of proportion to the incident or event that triggered the outburst. Impulsive acts of aggression, however, are not unique to intermittent explosive disorder. Impulsive aggression can be present in many psychological and nonpsychological disorders. The diagnosis of intermittent explosive disorder (IED) is essentially a diagnosis of exclusion, which means that it is given only after other disorders have been ruled out as causes of impulsive aggression.

Patients diagnosed with IED usually feel a sense of arousal or tension before an outburst, and relief of tension after the aggressive act. Patients with IED believe that their aggressive behaviors are justified; however, they feel genuinely upset, regretful, remorseful, bewildered or embarrassed by their impulsive and aggressive behavior.

Causes and symptoms

Causes

Recent findings suggest that IED may result from abnormalities in the areas of the **brain** that regulate behavioral arousal and inhibition. Research indicates that impulsive aggression is related to abnormal brain mechanisms in a system that inhibits motor (muscular movement) activity, called the serotoninergic system. This system is directed by a neurotransmitter called serotonin, which regulates behavioral inhibition (control of behavior). Some studies have correlated IED with abnormalities on both sides of the front portion of the brain. These localized areas in the front of the brain appear to be involved in information processing and controlling movement, both of which are unbalanced in persons diagnosed with IED. Studies using **positron emission tomography** (PET) scanning have found lower levels of brain glucose (sugar) metabolism in patients who act in impulsively aggressive ways.

Another study based on data from electroencephalograms (EEGs) of 326 children and adolescents treated in a psychiatric clinic found that 46% of the youths who manifested explosive behavior had unusual high-amplitude brain wave forms. The researchers concluded that a significant subgroup of people with IED may be predisposed to explosive behavior by an inborn characteristic of their central nervous system. In sum, there is a substantial amount of convincing evidence that IED has biological causes, at least in some people diagnosed with the disorder.

Other clinicians attribute IED to cognitive distortions. According to cognitive therapists, persons with IED have a set of strongly negative beliefs about other people, often resulting from harsh punishments inflicted by the parents. The child grows up believing that others "have it in for him" and that violence is the best way to restore damaged self-esteem. He or she may also have observed one or both parents, older siblings, or other relatives acting out in explosively violent ways. In short, people who develop IED have learned, usually in their family of origin, to believe that certain acts or attitudes on the part of other people "justify" aggressive attacks on them.

Although gender roles are not a "cause" of IED to the same extent as biological and familial factors, they are regarded by some researchers as helping to explain why most people diagnosed with IED are males. According to this theory, men have greater permission from society to act violently and impulsively than women do. They therefore have less reason to control their aggressive impulses. Women who act explosively, on the other hand, would be considered unfeminine as well as unfriendly or dangerous.

KEY TERMS

Assaultive—An act with intent of causing harm.

Electroencephalograph—(EEG) An instrument that measures the normal and abnormal electrical activity in the brain.

Episodic dyscontrol—Another term for intermittent explosive disorder.

Malingering—Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

Monomania—A nineteenth-century term for a pathological obsession with one idea or one social cause. Nineteenth-century psychiatrists often associated explosive behavior with monomania. The word is no longer used as a technical term.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Toxicology screen—A blood or urine test that detects the presence of toxic chemicals, alcohol, or drugs in body fluids.

Symptoms

IED is characterized by violent behaviors that are impulsive as well as assaultive. One example involved a man who felt insulted by another customer in a neighborhood bar during a conversation that had lasted for several minutes. Instead of finding out whether the other customer intended his remark to be insulting, or answering the "insult" verbally, the man impulsively punched the other customer in the mouth. Within a few minutes, however, he felt ashamed of his violent act. As this example indicates, the urge to commit the impulsive aggressive act may occur from minutes to hours before the "acting out" and is characterized by the buildup of tension. After the outburst, the IED patient experiences a sense of relief from the tension. While many patients with IED blame someone else for causing their violent outbursts, they also express remorse and guilt for their actions.

Demographics

IED is apparently a rare disorder. Most studies, however, indicate that it occurs more frequently in males. The most common age of onset is the period from late childhood through the early 20s. The onset of the disorder is frequently abrupt, with no warning period. Patients with IED are often diagnosed with at least one other disorder—particularly **personality disorders**, substance abuse (especially alcohol abuse) disorders, and neurological disorders.

Diagnosis

As mentioned, IED is essentially a diagnosis of exclusion. Patients who are eventually diagnosed with IED may come to the attention of a psychiatrist or other

mental health professional by several different routes. Some patients with IED, often adult males who have assaulted their wives and are trying to save their marriages, are aware that their outbursts are not normal and seek treatment to control them. Younger males with IED are more likely to be referred for diagnosis and treatment by school authorities or the juvenile justice system, or brought to the doctor by concerned parents.

A psychiatrist who is evaluating a patient for IED would first take a complete medical and psychiatric history. Depending on the contents of the patient's history, the doctor would give the patient a physical examination to rule out head trauma, epilepsy, and other general medical conditions that may cause violent behavior. If the patient appears to be intoxicated by a drug of abuse or suffering symptoms of withdrawal, the doctor may order a toxicology screen of the patient's blood or urine. Specific substances that are known to be associated with violent outbursts include phencyclidine (PCP or "angel dust"), alcohol, and cocaine. The doctor will also give the patient a mental status examination and a test to screen for neurological damage. If necessary, a neurologist may be consulted and **imaging studies** performed of the patient's brain.

If the physical findings and laboratory test results are normal, the doctor may evaluate the patient for personality disorders, usually by administering diagnostic questionnaires. The patient may be given a diagnosis of antisocial or **borderline personality disorder** in addition to a diagnosis of IED.

In some cases the doctor may need to rule out **malingering**, particularly if the patient has been referred for evaluation by a court order and is trying to evade legal responsibility for his behavior.

Treatments

Some adult patients with IED appear to benefit from cognitive therapy. A team of researchers at the University of Pennsylvania found that cognitive approaches that challenged the patients' negative views of the world and of other people was effective in reducing the intensity as well as the frequency of violent episodes. With regard to gender roles, many of the men reported that they were helped by rethinking "manliness" in terms of self-control rather than as something to be "proved" by hitting someone else or damaging property.

Several medications have been used for treating IED. These include **carbamazepine** (Tegretol), an antiseizure medication; **propranolol** (Inderal), a heart medication that controls blood pressure and irregular heart rhythms; and lithium, a drug used to treat bipolar type II manic-depression disorder. The success of treatment with lithium and other mood-stabilizing medications is consistent with findings that patients with IED have a high lifetime rate of **bipolar disorder**.

Prognosis

Little research has been done on patients who meet *DSM-IV-TR* criteria for IED, although one study did find that such patients have a high lifetime rate of comorbid (co-occurring) bipolar disorder. In some people, IED decreases in severity or resolves completely as the person grows older. In others, the disorder appears to be chronic.

Prevention

As of 2002, preventive strategies include educating young people in parenting skills, and teaching children skills related to self-control. Recent studies summarized by an article in a professional journal of psychiatry indicate that self-control can be practiced like many other skills, and that people can improve their present level of self-control with appropriate coaching and practice.

See also Gender issues in mental health; Self-control strategies

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Internet addiction disorder

Definition

Internet **addiction** disorder refers to the problematic use of the Internet, including the various aspects of its technology, such as electronic mail (e-mail) and the World Wide Web. The reader should note that Internet addiction disorder is not listed in the mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (2000), which is also called the *DSM*. Internet addiction has, however, been formally recognized as a disorder by the American Psychological Association.

Description

In some respects, addictive use of the Internet resembles other so-called "process" addictions, in which a person is addicted to an activity or behavior (including gambling, shopping, or certain sexual behaviors) rather than a substance (mood-altering drugs, tobacco, food, etc.). People who develop problems with their Internet use may start off using the Internet on a casual basis and then progress to using the technology in dysfunctional ways. Many people believe that spending large amounts of time on the Internet is a core feature of the disorder. The amount of time by itself, however, is not as important a factor as the ways in which the person's Internet use is interfering with their daily functioning. Use of the Internet may interfere with the person's social life, school work, or job-related tasks at work. In addition, cases have been reported of persons entering Internet chat rooms for people with serious illnesses or disorders, and pretending to be a patient with that disorder in order to get attention or sympathy. Treatment options often mirror those for other addictions. Although only a limited amount of research has been done on this disorder, the treatments that have been used appear to be effective.

Causes and symptoms

Causes

No one knows what causes a person to be addicted to the Internet, but there are several factors that have been proposed as contributing to Internet addiction. One theory concerns the mood-altering potential of behaviors related to process addictions. Just as a person addicted to shopping may feel a "rush" or pleasurable change in mood from the series of actions related to a spending spree— checking one's credit cards, driving to the mall, going into one's favorite store, etc.— the person with an Internet addiction may feel a similar "rush" from booting up their computer and going to their favorite web sites. In other words, some researchers think that there are chemical changes that occur in the body when someone is engaging in an addictive behavior. Furthermore, from a biological standpoint, there may be a combination of genes that make a person more susceptible to addictive behaviors, just as researchers have located genes that affect a person's susceptibility to alcohol.

In addition to having features of a process addiction, Internet use might be reinforced by pleasurable thoughts and feelings that occur while the person is using the Internet. Although researchers in the field of addiction studies question the concept of an "addictive personality" as such, it is possible that someone who has one addiction may be prone to become addicted to other substances or activities, including Internet use. People with such other mental disorders or symptoms as depression, feelings of isolation, **stress**, or anxiety, may "self-medicate" by using the Internet in the same way that some people use alcohol or drugs of abuse to self-medicate the symptoms of their mental disorder.

From a social or interpersonal standpoint, there may be familial factors prompting use of the Internet. For

KEY TERMS

Carpal tunnel syndrome—A disorder of the hand and wrist characterized by pain, weakness, or numbness in the thumb and other fingers. It is caused by pressure on a nerve in the wrist. Carpal tunnel syndrome is frequently associated with heavy use of a computer, typewriter, or musical keyboard.

Denial—A psychological defense mechanism that reduces anxiety by excluding recognition of an addiction or similar problem from the conscious mind.

Process addiction—An addiction to a mood-altering behavior or series of behaviors rather than a substance.

Reinforcement—A term that refers to the ability of a drug, substance, or behavior to produce effects that will make the user want to take the substance or perform the behavior again.

Rush—The initial intensely pleasurable sensation experienced from injecting a narcotic or stimulant drug. The term has also been applied to the feeling of excitement experienced from the behaviors involved in process addictions.

People who develop problems with their Internet use may start off using the Internet on a casual basis and then progress to using the technology in dysfunctional ways. Use of the Internet may interfere with the person's social life, school work, or job-related tasks at work. Many of the treatments that have been used for Internet addiction have been modeled after other addiction treatment programs and support groups. *(Oleg Nikishin/Getty Images. Reproduced by permission.)*

example, a person might "surf the Web" to escape family conflict. Another possibility is that social or peer dynamics might prompt excessive Internet use. Some affected persons may lack the social skills that would enable them to meet people in person rather than online. Peer behavior might also encourage Internet use if one's friends are using it. **Modeling** may play a role—users can witness and experience how others engage in Internet use and then replicate that behavior. The interactive aspects of the Internet, such as chat rooms, e-mail, and interactive games like Multi-User Dungeons and Dragons (MUDS), seem to be more likely to lead to Internet addiction than purely solitary web surfing.

One question that has not yet been answered concerning Internet addiction is whether it is a distinctive type of addiction or simply an instance of a new technology being used to support other addictions. For example, there are gambling casinos on the Internet that could reinforce a person's pre-existing gambling addiction. Similarly, someone addicted to shopping could transfer their addiction from the local mall to online stores. Persons addicted to certain forms of sexual behavior can visit pornography sites on the Internet or use chat rooms as a way to meet others who might be willing to participate in those forms of behavior. Researchers may need to determine whether there is such a disorder as "pure" Internet addiction.

Symptoms

One symptom of Internet addiction is excessive time devoted to Internet use. A person might have difficulty cutting down on his or her online time even when they are threatened with poor grades or loss of a job. There have been cases reported of college students failing courses because they would not take time off from Internet use to attend classes. Other symptoms of addiction may include lack of sleep, **fatigue**, declining grades or poor job performance, **apathy**, and racing thoughts. There may also be a decreased investment in social relationships and activities. A person may lie about how much time was spent online or deny that they have a problem. They may be irritable when offline, or angry toward anyone who questions their time on the Internet.

Demographics

In the past, people reported to have an Internet addiction disorder were stereotyped as young, introverted, socially awkward, computer-oriented males. While this stereotype may have been true in the past, the availability of computers and the increased ease of access to the Internet are quickly challenging this notion. As a result, problematic Internet use can be found in any age group, social class, racial or ethnic group, level of education and income, and gender.

Diagnosis

As previously noted, Internet addiction disorder has not yet been added as an official **diagnosis** to the *DSM*. The following, however, is a set of criteria for Internet addiction that has been proposed by addiction researchers. The criteria are based on the diagnostic standards for pathological gambling.

The patient must meet all of the following criteria:

- He or she is preoccupied with the Internet (thinks about previous online activity or is anticipating the next online session).
- He or she needs to spend longer and longer periods of time online in order to feel satisfied.
- He or she has made unsuccessful efforts to control, cut back, or stop Internet use.

- He or she is restless, moody, depressed, or irritable when attempting to cut down or stop Internet use.
- He or she repeatedly stays online longer than he or she originally intended.

The person must meet at least one of the following criteria:

- He or she has jeopardized or risked the loss of a significant relationship, job, educational or career opportunity because of Internet use.
- He or she has lied to family members, a therapist, or others to conceal the extent of involvement with the Internet.
- He or she uses the Internet as a way of escaping from problems or of relieving an unpleasant mood (such as feelings of helplessness, guilt, anxiety, or depression).

Treatments

Since Internet addiction disorder is a relatively new phenomenon, there is little research on the effectiveness of treatment procedures. Some professionals advocate abstinence from the Internet. Others argue that it may be unrealistic to have a person completely end all Internet use. As society becomes more and more dependent on computers for business transactions, educational programs, entertainment, and access to information as well as interpersonal communication, it will be difficult for a computer-literate person to avoid using the Internet. Learning how to use the Internet in moderation is often the main objective in therapy, in a way analogous to the way that people with eating disorders need to come to terms with food. Many of the procedures that have been used to treat Internet addiction have been modeled after other addiction treatment programs and **support groups**.

If a person's Internet addiction disorder has a biological dimension, then such medication as an antidepressant or anti-anxiety drug may help them with these aspects of the addiction. Psychological interventions may include such approaches as changing the environment to alter associations that have been made with Internet use, or decrease the **reinforcement** received from excessive Internet use. Psychological interventions may also help the person identify thoughts and feelings that trigger their use of the Internet. Interpersonal interventions may include such approaches as **social skills training** or coaching in communication skills. Family and couple therapy may be indicated if the user is turning to the Internet to escape from problems in these areas of life.

Relapsing into an addictive behavior is common for anyone dealing with addiction disorders. Recognizing and preparing for relapse is often a part of the treatment process. Identifying situations that would trigger excessive Internet use and generating ways to deal with these situations can greatly reduce the possibility of total relapse.

Prognosis

Although extensive studies have not yet been done, treatment appears to be effective in maintaining and changing the behavior of people drawn to excessive use of the Internet. If the disorder is left untreated, the person may experience an increased amount of conflict in his or her relationships. Excessive Internet use may jeopardize a person's employment or academic standing. In addition, such physical problems may develop as fatigue, carpal tunnel syndrome, back pain, and eyestrain.

Prevention

If a person knows that he or she has difficulty with other forms of addictive behavior, they should be cautious in exploring the types of application that are used on the Internet. In addition, it is important for people to engage in social activities outside the Internet. Finally, mental health workers should investigate ways in which to participate in the implementation of new technology rather than waiting for its aftereffects.

See also Factitious disorder; Pathological gambling disorder; Relapse and relapse prevention

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Interpersonal psychotherapy *see* **Interpersonal therapy**

Interpersonal therapy

Definition

Interpersonal therapy (IPT) is a short-term supportive **psychotherapy** that focuses on the connection between interactions between people and the development of a person's psychiatric symptoms.

Purpose

Interpersonal therapy was initially developed to treat adult depression. It has since been applied to the treatment of depression in adolescents, the elderly, and people with Human Immunodeficiency Virus (HIV) infection. There is an IPT conjoint (couple) therapy for people whose marital disputes contribute to depressive episodes. IPT has also been modified for the treatment of a number of disorders, including substance abuse; bulimia and **anorexia nervosa**; **bipolar disorder**; and dysthymia. Research is underway to determine the efficacy of IPT in the treatment of patients with **panic disorder** or **borderline personality disorder**; depressed caregivers of patients with traumatic **brain** injuries; depressed pregnant women; and people suffering from protracted bereavement.

Interpersonal therapy is a descendant of psychodynamic therapy, itself derived from **psychoanalysis**, with its emphasis on the unconscious and childhood experiences. Symptoms and personal difficulties are regarded as arising from deep, unresolved personality or character problems. **Psychodynamic psychotherapy** is a long-term method of treatment, with in-depth exploration of past family relationships as they were perceived during the client's infancy, childhood, and adolescence.

There are seven types of interventions that are commonly used in IPT, many of which reflect the influence of psychodynamic psychotherapy: a focus on clients' emotions; an exploration of clients' resistance to treatment; discussion of patterns in clients' relationships and experiences; taking a detailed past history; an emphasis on clients' current interpersonal experiences; exploration of the therapist/client relationship; and the identification of clients' wishes and fantasies. IPT is, however, distinctive for its brevity and its treatment focus. IPT emphasizes the ways in which a person's current relationships and social context cause or maintain symptoms rather than exploring the deep-seated sources of the symptoms. Its goals are rapid symptom reduction and improved social adjustment. A frequent byproduct of IPT treatment is more satisfying relationships in the present.

IPT has the following goals in the treatment of depression: to diagnose depression explicitly; to educate the client about depression, its causes, and the various treatments available for it; to identify the interpersonal context of depression as it relates to symptom development; and to develop strategies for the client to follow in coping with the depression. Because interpersonal therapy is a short-term approach, the therapist addresses only one or two problem areas in the client's current functioning. In the early sessions, the therapist and client determine which areas would be most helpful in reducing the client's symptoms. The remaining sessions are then organized toward resolving these agreed-upon problem areas. This time-limited framework distinguishes IPT from therapies that are open-ended in their exploration. The targeted approach of IPT has demonstrated rapid improvement for patients with problems ranging from mild situational depression to severe depression with a recent history of **suicide** attempts.

Interpersonal therapy has been outlined in a manual by Klerman and Weissman, which ensures some standardization in the training of interpersonal therapists and their practice. Because of this standardized training format, IPT is not usually combined with other talk therapies. Treatment with IPT, however, is often combined with drug therapy, particularly when the client suffers from such mood disorders as depression, dysthymia, or bipolar disorder.

Precautions

Training programs in interpersonal therapy are still not widely available, so that many practicing therapists base their work on the manual alone without additional supervision. It is unclear whether reading the manual alone is sufficient to provide an acceptable standard of care.

While interpersonal therapy has been adapted for use with substance abusers, it has not demonstrated its effectiveness with this group of patients. Researchers studying patients addicted to opiates or cocaine found little benefit to incorporating IPT into the standard recovery programs. These findings suggest that another treatment method that offers greater structure and direction would be more successful with these patients.

Description

Since the interpersonal therapy model was developed for the treatment of depression and then modified for use with other populations and mental disorders, an understanding of IPT's approach to depression is crucial. Interpersonal therapists focus on the functional role of depression rather than on its etiology or cause, and they look at the ways in which problematic interactions develop when a person becomes depressed. The IPT

framework considers clinical depression as having three components: the development of symptoms, which arise from biological, genetic and/or psychodynamic processes; social interactions with other people, which are learned over the course of one's life; and personality, made up of the more enduring traits and behaviors that may predispose a person to depressive symptoms. IPT intervenes at the levels of symptom formation and social functioning, and does not attempt to alter aspects of the client's personality.

Subtypes of IPT

Interpersonal therapy offers two possible treatment plans for persons with depressive disorders. The first plan treats the acute episode of depression by eliminating the current depressive symptoms. This approach requires intervening while the person is in the midst of a depression. The acute phase of treatment typically lasts two to four months with weekly sessions. Many clients terminate treatment at that point, after their symptoms have subsided. Maintenance treatment (IPT-M) is the second treatment plan and is much less commonly utilized than acute treatment. IPT-M is a longer-term therapy based on the principles of interpersonal therapy but with the aim of preventing or reducing the frequency of further depressive episodes. Some clients choose IPT-M after the acute treatment phase. IPT-M can extend over a period of two to three years, with therapy sessions once a month.

Psychoeducation in IPT

Treatment with IPT is based on the premise that depression occurs in a social and interpersonal context that must be understood for improvement to occur. In the first session, the psychiatric history includes a review of the client's current social functioning and current close relationships, their patterns and their mutual expectations. Changes in relationships prior to the onset of symptoms are clarified, such as the death of a loved one, a child leaving home, or worsening marital conflict.

IPT is psychoeducational in nature to some degree. It involves teaching the client about the nature of depression and the ways that it manifests in his or her life and relationships. In the initial sessions, depressive symptoms are reviewed in detail, and the accurate naming of the problem is essential. The therapist then explains depression and its treatment and may explain to the client that he or she has adopted the "sick role." The concept of the "sick role" is derived from the work of a sociologist named Talcott Parsons, and is based on the notion that illness is not merely a condition but a social role that affects the attitudes and behaviors of the client and those around him or her. Over time, the client comes to see that the

KEY TERMS

Bereavement—The emotional experience of loss after the death of a friend or relative.

Bingeing—An excessive amount of food consumed in a short period of time. Usually, while a person binge eats, he or she feels disconnected from reality, and feels unable to stop. The bingeing may temporarily relieve depression or anxiety, but after the binge, the person usually feels guilty and depressed.

Dysthymia—Depression of low intensity.

Dysthymic disorder—A mood disorder that is less severe than depression but usually more chronic.

Etiology—The cause or origin of a disease or disorder. The word is also used to refer to the study of the causes of disease.

Psychosocial—A term that refers to the emotional and social aspects of psychological disorders.

Purging—Inappropriate actions taken to prevent weight gain, often after bingeing, including self-induced vomiting or the misuse of laxatives, diuretics, enemas, or other medications.

Remission—In the course of an illness or disorder, a period of time when symptoms are absent.

Role—The set of customary or expected behavior patterns associated with a particular position or function in society. For example, a person's role as mother is associated with one set of expected behaviors, and her role as a worker with a very different set.

Role transition—Life changes that require an alteration in one's social or occupational status or self-image.

Stigma—A mark or characteristic trait of a disease or defect; by extension, a cause for reproach or a stain on one's reputation. Such sexually transmitted diseases as HIV infection carry a severe social stigma.

Supportive—An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or exploratory approaches to treatment.

sick role has increasingly come to govern his or her social interactions.

Identification of problem areas

The techniques of IPT were developed to manage four basic interpersonal problem areas: unresolved **grief**; role transitions; interpersonal role disputes (often marital disputes); and interpersonal deficits (deficiencies). In the early sessions, the interpersonal therapist and the client attempt to determine which of these four problems is most closely associated with the onset of the current depressive episode. Therapy is then organized to help the client deal with the interpersonal difficulties in the primary problem area. The coping strategies that the client is encouraged to discover and employ in daily life are tailored to his or her individual situation.

UNRESOLVED GRIEF. In normal bereavement, a person experiences symptoms such as sadness, disturbed sleep, and difficulty functioning but these usually resolve in two to four months. Unresolved grief in depressed people is usually either delayed grief, which has been postponed and then experienced long after the loss; or distorted grief, in which there is no felt emotion of sadness but there may be nonemotional symptoms, often physical. If unresolved grief is identified as the primary issue, the goals of treatment are to facilitate the mourning process. Successful therapy will help the client re-establish interests and relationships that can begin to fill the void of what has been lost.

ROLE DISPUTES. Interpersonal role disputes occur when the client and at least one other significant person have differing expectations of their relationship. The IPT therapist focuses on these disputes if they seem stalled or repetitious, or offer little hope of improvement. The treatment goals include helping the client identify the nature of the dispute; decide on a plan of action; and begin to modify unsatisfying patterns, reassess expectations of the relationship, or both. The therapist does not direct the client to one particular resolution of difficulties and should not attempt to preserve unworkable relationships.

ROLE TRANSITIONS. Depression associated with role transitions occurs when a person has difficulty coping with life changes that require new roles. These may be such transitions as retirement, a career change, moving, or leaving home. People who are clinically depressed are most likely to experience role changes as losses rather than opportunities. The loss may be obvious, as when a marriage ends, or more subtle, as the loss of freedom people experience after the birth of a child. Therapy is terminated when a client has given up the old role; expressed the accompanying feelings of guilt, anger, and loss; acquired new skills; and developed a new social network around the new role.

INTERPERSONAL DEFICITS. Interpersonal deficits are the focus of treatment when the client has a history of inadequate or unsupportive interpersonal relationships. The client may never have established lasting or intimate relationships as an adult, and may experience a sense of inadequacy, lack of self-assertion, and guilt about expressing anger. Generally, clients with a history of extreme social isolation come to therapy with more severe emotional disturbances. The goal of treatment is to reduce the client's social isolation. Instead of focusing on current relationships, IPT therapy in this area focuses on the client's past relationships; the present relationship with the therapist; and ways to form new relationships.

IPT in special populations

ELDERLY CLIENTS. In translating the IPT model of depression to work with different populations, the core principles and problem areas remain essentially the same, with some modifications. In working with the elderly, IPT sessions may be shorter to allow for decreased energy levels, and dependency issues may be more prominent. In addition, the therapist may work with an elderly client toward tolerating rather than eliminating long-standing role disputes.

CLIENTS WITH HIV INFECTION. In IPT with HIV-positive clients, particular attention is paid to the clients' unique set of psychosocial stressors: the **stigma** of the disease; the effects of being gay (if applicable); dealing with family members who may isolate themselves; and coping with the medical consequences of the disease.

ADOLESCENTS. In IPT with adolescents, the therapist addresses such common developmental issues as separation from parents; the client's authority in relationship to parents; the development of new interpersonal relationships; first experiences of the death of a relative or friend; peer pressure; and single-parent families. Adolescents are seen weekly for 12 weeks with once-weekly additional phone contact between therapist and client for the first four weeks of treatment. The parents are interviewed in the initial session to get a comprehensive history of the adolescent's symptoms, and to educate the parents as well as the young person about depression and possible treatments, including a discussion of the need for medication. The therapist refrains from giving advice when working with adolescents, and will primarily use supportive listening, while assessing the client for evidence of suicidal thoughts or problems with school attendance. So far, research does not support the efficacy of antidepressant medication in treating adolescents, though most

clinicians will give some younger clients a trial of medication if it appears to offer relief.

CLIENTS WITH SUBSTANCE ABUSE DISORDERS. While IPT has not yet demonstrated its efficacy in the field of substance abuse recovery, a version of IPT has been developed for use with substance abusers. The two goals are to help the client stop or cut down on drug use; and to help the client develop better strategies for dealing with the social and interpersonal consequences of drug use. To meet these goals, the client must accept the need to stop; take steps to manage impulsiveness; and recognize the social contexts of drug purchase and use. Relapse is viewed as the rule rather than the exception in treating substance abuse disorders, and the therapist avoids treating the client in a punitive or disapproving manner when it occurs. Instead, the therapist reminds the client of the fact that staying away from drugs is the client's decision.

CLIENTS WITH EATING DISORDERS. IPT has been extended to the treatment of eating disorders. The IPT therapist does not focus directly on the symptoms of the disorder, but rather, allows for identification of problem areas that have contributed to the emergence of the disorder over time. IPT appears to be useful in treating clients with bulimia whose symptoms are maintained by interpersonal issues, including social anxiety; sensitivity to conflict and rejection; and difficulty managing negative emotions. IPT is helpful in bringing the problems underlying the bingeing and purging to the surface, such as conflict avoidance; difficulties with role expectations; confusion regarding needs for closeness and distance; and deficiencies in solving social problems. IPT also helps people with bulimia to regulate the emotional states that maintain the bulimic behavior.

Anorexia nervosa also appears to be responsive to treatment with IPT. Research indicates that there is a connection between interpersonal and family dysfunction and the development of anorexia nervosa. Therapists disagree as to whether interpersonal dysfunction causes or is caused by anorexia. IPT has been helpful because it is not concerned with the origin but rather seeks to improve the client's interpersonal functioning and thereby decreasing symptoms. IPT's four categories of grief, interpersonal disputes, interpersonal deficits, and role transitions correspond to the core issues of clients with anorexia. **Social phobia** is another disorder that responds well to IPT therapy.

Aftercare

Interpersonal therapy as a maintenance approach (IPT-M) could be viewed as aftercare for clients suffering from depression. It is designed as a preventive measure by focusing on the period after the acute depression has passed. Typically, once the client is in remission and is symptom-free, he or she takes on more responsibilities and has increased social contact. These changes can lead to increased **stress** and greater vulnerability to another episode of depression. IPT-M enables clients to reduce the stresses associated with remission and thereby lower the risk of recurrence. The goal of maintenance therapy is to keep the client at his or her current level of functioning. Research has shown that for clients with a history of recurrent depression, total prevention is unlikely, but that maintenance therapy may delay a recurrence.

In general, long-term maintenance psychotherapy by itself is not recommended unless there are such reasons as pregnancy or severe side effects that prevent the client from being treated with medication. IPT-M does, however, seem to be particularly helpful with certain groups of patients, either alone or in combination with medication. Women appear to benefit, due to the importance of social environment and social relations in female gender roles; the effects of the menstrual cycle on symptoms; and complications related to victimization by rape, incest, or battering. IPT is also useful for elderly clients who can't take antidepressants due to intolerable side effects or such medical conditions as autoimmune disorders, cardiovascular disorders, diabetes, or other general medical conditions.

Normal results

The expected outcomes of interpersonal therapy are a reduction or the elimination of symptoms and improved interpersonal functioning. There will also be a greater understanding of the presenting symptoms and ways to prevent their recurrence. For example, in the case of depression, a person will have been educated about the nature of depression; what it looks like for him or her; and the interpersonal triggers of a depressive episode. A person will also leave therapy with strategies for minimizing triggers and for resolving future depressive episodes more effectively. While interpersonal therapy focuses on the present, it can also improve the client's future through increased awareness of preventive measures and strengthened coping skills.

Abnormal results

Research has shown that IPT requires clients' commitment to therapy prior to starting the treatment. If clients are resistant to an educational approach, the results of IPT are generally poor. It has been found that when people do not accept IPT's methods and approach at the outset, they are unlikely to be convinced over the course of therapy and they receive little benefit from

treatment. IPT clients appear to do better in therapy if they have confidence in their therapist; therefore, if the initial fit between therapist and client is not good, therapy will often be unsuccessful. A client should listen to his or her instincts early in treatment, and either seek out another interpersonal therapist or find a therapist who uses a different approach— such as **cognitive-behavioral therapy**, which was also developed specifically for the treatment of depression.

See also Bulimia nervosa; Gender issues in mental health; Grief; Major depressive disorder

Resources

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International Society for Interpersonal Psychotherapy. c/o Myrna M. Weissman, Columbia University, 1051 Riverside Drive, Unit 24, New York, NY 10032. <http://interpersonalpsychotherapy.org>.

Holly Scherstuhl, M.Ed.

Intervention

Definition

A standard dictionary defines intervention as an influencing force or act that occurs in order to modify a given state of affairs. In the context of behavioral health, an intervention may be any outside process that has the effect of modifying an individual's behavior, cognition, or emotional state. For example, a person experiencing **stress** symptoms may find a variety of interventions effective in bringing relief. Deep breathing, vigorous exercise, talking with a therapist or counselor, taking an anti-anxiety medication, or a combination of these activities are all interventions designed to modify the symptoms and potentially the causes of stress-related discomfort.

The term is also used to describe a specific process designed to break through **denial** on the part of persons with serious addictive disorders. Interventions in this sense of the word involve carefully orchestrated confrontations in which friends, family members, and (in many cases) employers confront the person with the negative impact and consequences of his or her **addiction**. The goal of an intervention is to bring the addicted person to acknowledge that he or she suffers from a disorder and agree to treatment. This goal, however, is not always realized.

Description

According to the *Report of the Surgeon General on Mental Health* published in 1999, one in five Americans in a given year will experience behavioral health difficulties of sufficient magnitude and discomfort as to benefit from some form of therapeutic intervention. Unfortunately, only a small number of these persons seek help. The report goes on to state that the efficacy of mental health treatments is now well documented and a range of treatment interventions exist for even the most serious mental disorders.

There is no one-size-fits-all-intervention for behavioral health disorders. Recent research advances and greater understanding of behavioral health problems have provided an expanded range of treatments that promise better outcomes than those available in the past. For people who overcome the barriers of **stigma**, discrimination,

and limited access, there is a broad variety of helpful interventions from which to choose. Both personal preference and the severity of discomfort may influence the choice of "talk therapy," the use of medications, participation in self-help or **support groups**, or even inpatient treatment. In most cases, a combination of different interventions has proven to be most effective. As a result, many therapists tend to be eclectic in their practice and use a combination of approaches to in order to be as effective as possible with a wide variety of people.

Psychotherapy or "talk therapy" involves face-to-face meetings with a therapist who may specialize in a certain approach to treatment.

- Psychoanalysis is the oldest form of "talk therapy." It is a long-term form of treatment intended to uncover a person's unconscious motivations and early patterns in order to resolve present issues.
- Behavioral therapy is designed to change thinking patterns and behavior. Exposure therapy is a subtype of behavioral therapy that is useful in treating **obsessive-compulsive disorder** and **post-traumatic stress disorder**. The client is deliberately exposed to stimuli that trigger the painful thoughts or feelings under carefully controlled conditions that include support from the therapist. The individual is then taught techniques to avoid performing the compulsive behaviors or to work through the traumatic event.
- Cognitive therapy seeks to identify and correct dysfunctional thinking patterns that lead to troublesome feelings or behavior.
- **Family therapy** includes discussion and problem-solving sessions that include all members of the family.
- Group therapy takes place in a small group with the guidance of a therapist. The focus is on individual issues; group members assist each other in problem solving.
- Movement, art, and music therapists use these forms of creative expression to help people deal with strong emotions that are less easily handled in a "talk therapy" format.

Drug therapy involves the use of prescribed medications to treat the symptoms of certain mental or emotional disorders. It is important for patients to be aware of possible side effects of the medications; to inform the doctor of all other medications and alternative remedies that they are taking; and to have their blood, blood pressure, or other vital signs monitored regularly by the prescribing physician.

Electroconvulsive therapy (ECT) is used to treat depression and a few other specific conditions that have not responded to other interventions. It involves a controlled series of electric shocks to certain areas of the **brain**. It has been proven effective for some people despite the fact that it continues to be controversial. Patients should be fully aware of the side effects of ECT and assure themselves that the professional has been properly trained to administer ECT.

Psychosocial treatments may include **talk therapy** and medication in combination with social and vocational training to assist people recovering from severe mental illnesses. Psychosocial interventions may also include education about the illness itself, techniques for managing its symptoms, and ways in which friends and family members can help.

Psychoeducation is a word used to describe the process of teaching people about their illness, its treatment, and early warning signs of relapse, so that they can seek treatment before the illness worsens. Psychoeducation may also include learning about coping strategies, problem solving, and preparation of a crisis plan in the event of a relapse or future episode.

Self-help groups and support groups are another form of intervention that has become increasingly common in recent years. They exist for almost all disorders and are often based on the basic principles and values of the Alcoholics Anonymous movement founded in the 1930s. Although they are not led by professionals, these groups may be therapeutic because members give one another ongoing support and assistance. Group members share frustrations and successes, recommendations about specialists and community resources, and helpful tips about recovery. They also share friendship and hope for themselves, their loved ones, and others in the group. Unqualified acceptance by other people can be a powerful intervention for people recovering from a mental illness or addictive disorder.

Preparation

A common question about interventions concerns sources of help or further information. Many communities have a local hotline number that provides referrals and resources, or a mental health association that can direct callers to appropriate clinics, agencies, or groups. Helping resources may include the following:

- A **community mental health** center, usually a part of the state's department of mental health.
- Local mental health organizations with which the reader may be familiar.
- Family physicians.
- Clergy or spiritual counselors.

- Family service agencies, including charities and family or social services sponsored by various churches, synagogues, or other religious groups.
- High school or college guidance counselors.
- Marriage and family counselors.
- Child guidance counselors.
- Accredited psychiatric hospitals.
- Hotlines, crisis centers, and emergency rooms.

There are several categories of mental health professionals who have been specially trained to provide a range of interventions to relieve suffering, treat specific symptoms, or improve overall mental health. Competent professionals are licensed or certified by a particular specialty board or state licensing body. Their credentials imply a certain level of education, training, experience, and subscription to a code of ethics. Mental health professionals include:

- **Psychiatrists**. Psychiatrists are medical doctors with specialized training in the **diagnosis** and treatment of behavioral and emotional illnesses. They are qualified to prescribe medications. They may also specialize in certain fields within psychiatry, such as child/adolescent or geriatric psychiatry.
- **Psychologists**. These professionals are counselors with a doctoral degree (Ph.D. or Psy.D.) and two or more years of supervised work experience. They are trained to make diagnoses, administer and interpret psychological tests, and provide individual, family and group therapy.
- Clinical **social workers**. Clinical social workers have completed a master's degree in social work from an accredited graduate program. They are trained to make diagnoses and provide individual, family and group therapy.
- Licensed professional counselors and mental health counselors also hold a master's degree with supervised work experience and are trained to make diagnoses and provide individual, family and group therapy.
- Certified alcohol and drug abuse counselors. These professionals have specialized training in the treatment of alcohol and drug abuse. They are able to diagnose and provide counseling to individuals, families and groups.
- Nurse psychotherapists. Nurse psychotherapists are registered nurses (RNs) with specialized training in psychiatric and mental health nursing. They can diagnose disorders and provide counseling to individuals, families and groups.
- Marital and family therapists. These counselors have completed a master's or doctor's degree with specialized training in marital and family therapy. They are also trained to diagnose and provide individual, family and group counseling.
- Pastoral counselors. These counselors are ordained clergy with advanced training and certification in Level II clinical pastoral education as well as the master's degree in theology (M. Div.) required by most American denominations for ordination. In addition to offering psychological counseling to individuals, families and groups, pastoral counselors have been trained to offer spiritual and sacramental ministry to those who request it.

Resources

ORGANIZATIONS

American Psychological Association. 750 First Street, NE, Washington, DC 20002. (800) 374-2721. <www.apa.org>.

National Alliance for the Mentally Ill (NAMI). Colonial Place Three, 2107 Wilson Blvd., Suite 300, Arlington, VA 22201. <http://www.nami.org>.

National Mental Health Association. 1021 Prince St., Alexandria, VA 22314. <http://www.nmha.org>.

National Mental Health Consumers' Self-Help Clearinghouse. 1211 Chestnut St, Suite 1207, Philadelphia, PA 19107. <www.mhselfhelp.org>.

Judy Leaver, M.A.

Involuntary hospitalization

Definition

Involuntary **hospitalization** is a legal procedure used to compel an individual to receive inpatient treatment for a mental health disorder against his or her will. The legal justifications vary somewhat from state to state, but are generally based on a determination that a person is imminently dangerous to self or others; is gravely disabled; or clearly needs immediate care and treatment. Involuntary hospitalization is synonymous with involuntary commitment or involuntary treatment, and is an extremely controversial course of action. It is generally a last resort used in dealing with a person who is so ill that he/she is unable to use proper judgment or insight in deciding to refuse treatment.

Purpose

Civil commitment laws in the United States have been justified on the historical foundation of two funda-

mental powers and responsibilities of government. First, governments are responsible for protecting each citizen from injury by another. This power of protection is commonly called police powers. The second power, known as *parens patriae* (Latin for "parent of the nation") is based on the government's responsibility to care for a disabled citizen as a loyal parent would care for a child. A person with a significant mental illness may be civilly committed, or involuntarily hospitalized, under either of these powers. It is understood that the purpose of civil commitment is protecting the safety of the public or of the ill person.

Thirty-four states currently permit some type of involuntary commitment procedure. Most require proof of dangerousness, which can be interpreted in ambiguous ways but generally means the danger is imminent or provable. The legal process usually requires a court hearing within 24–72 hours after the emergency commitment procedure to assure due process.

Beyond safety issues, mental health professionals have thought that proper psychiatric treatment, even when administered against a person's wishes, is preferable to the continued worsening of a serious mental illness. There is some question currently about the effectiveness of forced treatment in the legal and mental health communities. Indeed "involuntary treatment" is considered by many patients' rights advocates and mental health consumers to be an oxymoron (a figure of speech that uses seeming contradictions). It may, in fact, protect public safety at the expense of the liberty, dignity and health of the person with a mental illness.

Precautions

The use of involuntary hospitalization or any other form of forced treatment is perhaps the most controversial issue in the wider mental health community, pitting family members, citizen advocacy groups, professionals and consumers against one another on the subject. In addition, legal advocates and the courts take very seriously the denial of a person's liberty. Involuntary hospitalization is one of the most extreme examples of denial of liberty in a democratic society.

Most people involved in the debate would agree that forced treatment is indicative of a failed treatment system. There is some evidence that forced treatment is generally harmful and counterproductive. Yet, many people with an intensely personal stake in such a decision may see the necessity of forced treatment to prevent harm to the person with an illness or to others. Outspoken advocates may believe that in the case of involuntary **intervention**, only custodial care should be provided. There is great concern, often based on experience, that a person who has been civilly committed to a treatment facility, will also receive such forced treatment as strong antipsychotic medications or **electroconvulsive therapy** (ECT). The issue of a person's ability to exercise **informed consent** about his/her treatment is clouded when the legal process of civil commitment has been initiated. In addition, there is concern that inpatient treatment will add to the **stigma** of being diagnosed with a mental illness. One research study found that persons who had been hospitalized (voluntarily or involuntarily) for treatment of a mental illness were even more likely to suffer discrimination in the job market than those who had received only outpatient treatment.

On the other hand, there are many mental health consumers who claim that an incident of involuntary hospitalization in their own treatment history may not only have saved their lives, but enabled them to receive treatment at a time when they were not capable of making a decision to do so. Family members sometimes consider involuntary hospitalization their only recourse to prevent the downward spiral of a loved one into a severe and debilitating mental illness, contact with the criminal justice system, or the devastation and dangers of **homelessness**.

KEY TERMS

Deinstitutionalization—The process of moving people out of mental hospitals into treatment programs or halfway houses in local communities. With this movement, the responsibility for care shifted from large (often governmental) agencies to families and community organizations.

Due process—A term referring to the regular administration of a system of laws that conform to fundamental legal principles and are applied without favor or prejudice to all citizens. In the context of involuntary commitment, due process means that people diagnosed with a mental illness cannot be deprived of equal protection under the laws of the United States on the basis of their diagnosis.

Oxymoron—A figure of speech that involves a seeming contradiction, as in the phrase "making haste slowly."

Description

As of 2002, involuntary hospitalization is a complex process because of the legal requirements that have been put in place to protect citizens from being hospitalized

because of a family quarrel or similar interpersonal issue. In the nineteenth century, for example, it was commonplace for husbands who wanted to end a marriage to have wives hospitalized against their will, or for parents to commit "disobedient" children. At present, however, most states require the person who thinks someone else should be hospitalized to call 911 or their local police department. A general summary of the events that may follow the call to 911 follows, but it should be noted that procedures vary from state to state and that the following is a general synopsis. In many cases, the department will send a patrol team rather than only one officer. If the person who has made the call is in the same house (or other location) as the person needing treatment, one officer will usually talk to the caller in one room while the other talks to the affected person in a different room (if circumstances permit). The officers may also interview other family members, neighbors, bystanders, or others who may know the affected person or have witnessed their behavior. Then the two police officers will compare their evaluations of the situation. In most jurisdictions the police officers can make one of three decisions: they can decide that the person who made the call has misjudged the situation (for example, the other person may simply be intoxicated); they can decide that the affected person is mentally ill but not necessarily dangerous; or they can take the affected person to the nearest hospital emergency room. They may ask the person who called them to accompany them to the hospital. In some states, however, the officers themselves must witness the affected person attempting to harm him- or herself or someone else before they can take him or her to the emergency room.

In the emergency room, the **psychiatrist** on duty will evaluate the affected person for dangerousness as well as the presence of mental illness. He or she will interview the police officers and anyone who accompanied them as well as the affected person. If the affected person has been receiving treatment for a mental disorder, the psychiatrist will usually contact the therapist. In some cases the person will need a medical evaluation, including assessment for substance abuse or withdrawal, before the doctor can proceed with a psychiatric assessment. The psychiatric assessment will be thorough, and documented as completely as possible; laboratory tests will be ordered if necessary. When the assessment is complete, *the doctor is legally required to decide in favor of the least restrictive environment to which the patient can be safely discharged for continued care*.

If the doctor decides that the person is dangerous but not mentally ill, he or she will turn the person over to law enforcement. If the person has threatened to kill themselves, but the psychiatrist does not consider the threat to be lethal, he or she may allow the patient to leave the emergency room after assessment. A decision to hospitalize the person involuntarily is based on three considerations: loss of emotional control; clear evidence of a psychotic disorder; evidence of impulsivity with serious thoughts, threats, or plans to kill self or others. In most cases, the affected person will be reassessed the next day. Most states stipulate that the affected person is entitled to a hearing before a judge who specializes in mental health law within 72 hours of hospitalization. The judge can order the person released if he or she thinks the person is not dangerous.

Readers who are concerned about the mental health of a family member, roommate, or friend are advised to gather information about the legal requirements for involuntary hospitalization in their state ahead of time, because it is not easy to think clearly when someone is acting in a bizarre or frightening manner. It is also a good idea to write down the name and telephone number of the person's therapist (if they have one), and the names of any medications that the person is taking.

Risks

A number of factors in the early 1980s led to a trend toward declining use of involuntary hospitalization for people with significant mental illnesses. The development and effectiveness of a range of new medications meant that treatment in general was more successful. The continued move toward **deinstitutionalization**, or moving people out of hospitals and into their communities, contributed as well. Treating people in hospitals is inherently expensive and was being viewed as less effective, compared to more innovative and less costly forms of treatment in smaller community-based programs. Finally, a continuing concern about civil liberties led to closer court scrutiny, the right to a hearing and legal counsel, and laws establishing a person's rights to the least restrictive form of treatment.

Recently, however, after a number of tragic and highly publicized violent incidents involving people with severe untreated mental illness, there appears to be a trend toward modification of the criteria required for involuntary hospitalization, court-ordered treatment, and outpatient commitment. Those who advocate liberalizing the process would like a person's previous mental health history to be included in the court's consideration and the standard of dangerousness to be broadened.

Most persons involved in the mental health community believe that an adequately funded, community-based continuum of care and treatment would drastically reduce the number of cases in which involuntary treatment of any kind is necessary. The use of psychiatric **advance directives** may have an effect on the use of

involuntary treatment as well. A psychiatric advance directive is a clearly written statement of an individual's psychiatric treatment preferences or instructions, somewhat like a living will for medical conditions. Psychiatric advance directives have not yet been tested in the court system but are widely endorsed throughout the mental health community as an alternative to involuntary treatment.

See also Advance directives; Schizophrenia; Suicide

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Judge David L. Bazelon Center for Mental Health Law. 1101 15th St. NW, Suite 1212, Washington, DC 20005. <http://www.bazelon.org>.

National Alliance for the Mentally Ill (NAMI). Colonial Place Three, 2107 Wilson Blvd, Suite 300, Arlington, VA 22201. <http://www.nami.org>.

National Mental Health Association. 1021 Prince St. Alexandria, VA 22314. <http://www.nmha.org >.

National Mental Health Consumers' Self-Help Clearinghouse. 1211 Chestnut Street, Suite 1207, Philadelphia, PA 19107. <http://www.mhselfhelp.org>.

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J

Jin Shin Jyutsu *see* **Bodywork therapies**
Journal therapy *see* **Creative therapies**

K

K-ABC *see* **Kaufman Assessment Battery for Children**

KAIT *see* **Kaufman Adolescent and Adult Intelligence Test**

Kaufman Adolescent and Adult Intelligence Test

Definition

The Kaufman Adolescent and Adult Intelligence Test (KAIT) is an individually administered general intelligence test appropriate for adolescents and adults, aged 11 to over 85 years.

Purpose

The KAIT is intended to measure both fluid and crystallized intelligence. Fluid intelligence refers to abilities such as problem solving and reasoning, and generally thought not to be influenced by one's cultural experience or education. Crystallized intelligence refers to acquired knowledge and is thought to be influenced by one's cultural experience and education.

The KAIT was developed by Alan S. Kaufman and Nadeen L. Kaufman as a method of measuring intelligence assuming broader definitions of fluid and crystallized abilities than assumed by other measures. Also, they wanted a test based on theories that accounted for developmental changes in intelligence. Although the Kaufmans had earlier designed a test for younger children, the **Kaufman Assessment Battery for Children** (K-ABC), they did not consider the KAIT to be an extension of this test. They believed that the developmental and neuropsychological changes specific to adults and adolescents warranted a different testing approach than did the changes relevant to younger children. Thus, a different approach was used when developing the KAIT, although the K-ABC was also based somewhat on the split between fluid and crystallized intelligence.

Theoretically, the KAIT is most influenced by Horn and Cattell's formulation of the distinction between fluid and crystallized intelligence, sometimes referred to as Gf-Gc theory. Gf refers to general fluid abilities and Gc refers to general crystallized abilities. The KAIT is also influenced by Piaget's theory of cognitive development, specifically the formal operations stage experienced in adolescence. During this stage, adolescents begin to perform more complex mental operations and are better able to transform and manipulate information. Another theoretical influence of the KAIT is Luria's theory of planning ability. This theory attempted to explain developmental changes occurring in early adolescence that influence decision making and problem solving.

Precautions

There are very specific rules governing administration of the test that must be adhered to for scoring to be accurate. Thus, administrators must be properly trained to administer the KAIT. Specifically, for all subtests there is a discontinue rule, instructing administrators when to stop administering test items.

The KAIT is not appropriate for children younger than 11. A test more appropriate for younger children, such as the K-ABC, should be given instead. The K-ABC is appropriate for children up to the age of 12 years and six months, so there is some overlap between the two tests, specifically for children between 11 and 12 years and six months old.

Description

The KAIT includes two components, a core battery and an expanded battery. The core battery consists of a fluid scale, a crystallized scale, and six subtests, and takes about 65 minutes to complete. The expanded battery

KEY TERMS

Factor analysis—A statistical method for summarizing relationships between variables.

Mean—The mathematical average of all scores in a set of scores. The means are based on a comparison to others in the same age group. Standardizing the means within age groups allow for comparisons across age groups.

Standard deviation—A measure of variability in a set of scores. The standard deviations are based on a comparison to others in the same age group. Standardizing in this way then allows scores to be comparable across age groups.

Standardization—The administration of a test to a sample group of people for the purpose of establishing scoring norms.

includes the core battery elements, as well as four additional subtests, and takes about 90 minutes to complete.

The following core battery subtests are related to fluid intelligence: logical steps, a test of sequential reasoning; mystery codes, a test measuring induction; and Rebus learning, a test of long-term memory. The following core battery subtests are related to crystallized intelligence: definitions, a test of word knowledge and language development; double meanings, a measure of language comprehension; and auditory comprehension, a test of listening ability.

The expanded battery also includes memory for block designs, a measure of visual processing related to fluid intelligence; famous faces, a test of cultural knowledge related to crystallized intelligence; auditory delayed recall; and Rebus delayed recall. The two delayed recall subtests provide a general measure of delayed memory.

There is also an optional supplemental mental status exam included in the KAIT battery. This subtest is only given to examinees with suspected mental impairment.

One strength of the KAIT is that most of the subtests are presented in both visual and auditory formats. This gives test takers more variety and allows for measurement of intelligence in different contexts. Also, the test was designed in a way to keep test takers active and engaged.

In contrast to other adult-specific or adolescent-specific **intelligence tests**, the KAIT is appropriate for a wider age range. This allows for more accurate tracking of intelligence changes between adolescence and adulthood.

Results

The KAIT yields several different kinds of scores, including raw scores, scaled scores, and intelligent quotient (IQ) scores. Raw scores and scaled scores are calculated for each subtest (six for the core battery; 10 for the expanded battery). Raw scores are calculated first, and simply refer to the number of points achieved by the examinee on a particular subtest. Raw scores are converted to scaled scores to ease comparison between subtests and between examinees. The subtest scaled scores are standardized to have a mean of 10 and a standard deviation of three.

Three IQ scores are obtained: composite intelligence, fluid intelligence, and crystallized intelligence. The IQ scores have a mean of 100 and a standard deviation of 15. The fluid intelligence IQ score is based on the sum of the three fluid intelligence subtests (logical steps, mystery codes, and Rebus learning). The crystallized intelligence IQ score is based on the sum of the three crystallized intelligence subtests (definitions, double meanings, and auditory comprehension). The composite intelligence IQ score is based on all six core subtests. The expanded battery subtests are not utilized when computing the three IQ scores.

Overall, the KAIT has high reliability and validity. Studies have indicated that in relation to other general intelligence tests, the crystallized, fluid, and composite IQ scores are accurately and consistently measured. Data looking at trends related to age show that average subtest and IQ scores are fairly consistent across the age range in which the KAIT is administered.

The KAIT yields IQ scores in a relatively wide range, from much lower-than-average intelligence to much higher-than-average intelligence. Because of this, the KAIT is often used as an assessment of individuals with exceptional abilities, such as gifted children.

There have been factor analysis studies comparing the KAIT to the widely used Wechsler scales of intelligence, (the **Wechsler intelligence scale for children** and the **Wechsler adult intelligence scale**). The KAIT crystallized IQ has been shown to measure abilities similar to those measured by the Wechsler scales' verbal intelligence factor. However, the KAIT Fluid IQ has been shown to measure abilities considerably different from those measured by the Wechsler performance factor, which is thought to be a measure of fluid intelligence.

See also Stanford-Binet intelligence scales

Resources

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Ali Fahmy, Ph.D.

Kaufman Assessment Battery for Children

Definition

The Kaufman Assessment Battery for Children (K-ABC) is a standardized test that assesses intelligence and achievement in children aged two years, six months to 12 years, six months. The edition published in 1983 by Kaufman and Kaufman was in the process of being revised in 2002 to expand its age range (to cover children ages three to eighteen) and enhance its usefulness. In addition, new subtests were being added and existing subtests updated.

Purpose

The K-ABC was developed to evaluate preschoolers, minority groups, and children with learning disabilities. It is used to provide educational planning and placement, neurological assessment, and research. The assessment is to be administered in a school or clinical setting and is intended for use with English speaking, bilingual, or nonverbal children. There is also a Spanish edition that is to be used with children whose primary language is Spanish.

Precautions

The K-ABC is especially useful in providing information about nonverbal intellectual abilities. However, it has been criticized for not focusing on measures of verbal intelligence in the Mental Processing Composite score, which measures intelligence. Additionally, the separation of intelligence and achievement scores has been questioned by researchers who claim the two terms are misleading. For example, many subtests in the achievement composite are in fact measures of intelligence rather than achievement (knowledge acquired through school and/or home environment). The K-ABC should be used with caution as the primary instrument for identifying the intellectual abilities of children.

Administration and interpretation of results (as with all psychometric testing) requires a competent examiner who is trained in psychology and individual intellectual assessment—preferably a **psychologist**.

KEY TERMS

Psychometric testing—Pertaining to testing and measurement of mental or psychological abilities. Psychometric tests convert an individual's psychological traits and attributes into a numerical estimation or evaluation.

Description

Administration of the K-ABC takes between 35 and 85 minutes. The older the child, the longer the test generally takes to administer. It is comprised of four global test scores that include:

- sequential processing scales
- simultaneous processing scales
- achievement scales
- mental processing composite

There is an additional nonverbal scale that allows applicable subtests to be administered through gestures to hearing impaired, speech/language impaired, or children who do not speak English.

The test consists of 16 subtests—10 mental processing subtests and six achievement subtests. Not all subtests are administered to each age group, and only three subtests are administered to all age groups. Children ages two years, six months are given seven subtests, and the number of subtests given increase with the child's age. For any one child, a maximum of 13 subtests are administered. Children from age seven years to 12 years, six months are given 13 subtests.

The sequential processing scale primarily measures short-term memory and consists of subtests that measure problem-solving skills where the emphasis is on following a sequence or order. The child solves tasks by arrang-

ing items in serial or sequential order including reproducing hand taps on a table, and recalling numbers that were presented. It also contains a subtest that measures a child's ability to recall objects in correct order as presented by the examiner.

The simultaneous processing scale examines problem-solving skills that involve several processes at once. The seven subtests comprising this scale are facial recognition, identification of objects or scenes in a partially completed picture, reproduction of a presented design by using rubber triangles, selecting a picture that completes or is similar to another picture, memory for location of pictures presented on a page, and arrangement of pictures in meaningful order.

The achievement scales measure achievement and focus on applied skills and facts that were learned through the school or home environment. The subtests are expressive vocabulary; ability to name fictional characters, famous persons, and well-known places; mathematical skills; ability to solve riddles; reading and decoding skills; and reading and comprehension skills.

The sequential and simultaneous processing scales are combined to comprise the mental processing composite. This composite measures intelligence on the K-ABC and concentrates on the child's ability to solve unfamiliar problems simultaneously and sequentially. The simultaneous processing scales have a greater impact on the mental processing composite score than do the sequential processing scales. The mental processing composite score is considered the global estimate of a child's level of intellectual functioning.

Results

The K-ABC is a standardized test, which means that a large sample of children in the two years, six months to 12 years, six months age range was administered the exam as a means of developing test norms. Children in the sample were representative of the population of the United States based on age, gender, race or ethnic group, geographic region, community size, parental education, educational placement (normal versus special classes), etc. From this sample, norms were established.

Based on these norms, the global scales on the K-ABC each have a mean or average score of 100 and a standard deviation of 15. For this test, as with most measures of intelligence, a score of 100 is in the normal or average range. The standard deviation indicates how far above or below the norm a child's score is. For example, a score of 85 is one standard deviation below the norm score of 100.

Test scores provide an estimate of the level at which a child is functioning based on a combination of many different subtests or measures of skills. A trained psychologist is needed to evaluate and interpret the results, determine strengths and weaknesses, and make overall recommendations based on the findings and behavioral observations.

See also Intelligence tests; Luria-Nebraska Neuropsychological Battery

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The American Psychological Association. 750 First St., NE, Washington, DC 20002-4242. (202) 336-5500. <www.apa.org>.

The National Association of School Psychologists. 4340 East West Highway, Suite 402, Bethesda, MD 20814. (301) 657-0270. <www.nasponline.com>.

Jenifer P. Marom, Ph.D.

Kaufman Short Neurological Assessment Procedure

Definition

The Kaufman Short Neurological Procedure, often abbreviated as K-SNAP, is a brief test of mental functioning appropriate for adolescents and adults between the ages of 11 and 85 years. It is administered on an individual basis, and measures mental functioning at varying levels of cognitive complexity as well as addressing possible neurological damage.

Purpose

The K-SNAP is intended as a short measure of mental functioning and is sometimes preferable to other longer mental status and intelligence exams. Compared to the **Kaufman Adolescent and Adult Intelligence Test** (KAIT), which is given to people in the same age range and takes over an hour to complete, the K-SNAP

takes only 20–30 minutes. The K-SNAP provides a measure of general mental status, as well as addressing specific mental abilities. It also allows for assessment of damage to the nervous system.

The K-SNAP was developed by Alan S. Kaufman and Nadeen L. Kaufman. Other Kaufman tests include the KAIT and the **Kaufman Assessment Battery for Children** (K-ABC). The Kaufmans based their tests on Horn and Cattell's formulation of the distinction between fluid and crystallized intelligence, sometimes referred to as the Gf-Gc Theory. Gf refers to such general fluid abilities as problem solving and reasoning. Fluid intelligence is thought not to be influenced by a person's cultural experience and education. Gc refers to such general crystallized abilities as acquired knowledge. Crystallized intelligence, unlike fluid intelligence, is thought to be shaped by a person's cultural experience and education.

Because the K-SNAP provides a measure of possible neurological impairment, it is often preferable to other measures of mental status and intelligence. If the doctor suspects that a patient may have a disorder of the nervous system, the doctor can use the K-SNAP as a short initial assessment. Depending on the results of the K-SNAP, the doctor can give more specific tests.

Precautions

One should be careful when using the results of the K-SNAP to assess neurological impairment. It should be used as a supplement to other more extensive and more specific measures of neuropsychological functioning.

The K-SNAP is primarily a test of mental and neuropsychological functioning. Although it measures cognitive skills, it should not be used to measure someone's overall intelligence.

Description

The K-SNAP consists of four subtests administered in the following order of complexity: Mental Status; Gestalt Closure; Number Recall; and Four-Letter Words. Each subtest contains between 10 and 25 items.

The Mental Status subtest assesses the test taker's alertness, attentiveness, and orientation to the environment. In this subtest, the examiner asks the examinee to answer verbal questions. It is the easiest and shortest of the four subtests, containing only 10 items.

The Gestalt Closure subtest provides an assessment of visual closure and simultaneous processing. In this subtest, the examinee is shown partially completed inkblot pictures and is asked to name the objects in the pictures.

KEY TERMS

Gestalt—A German word that means "form" or "structure." The Gestalt Closure subtest on the K-SNAP measures a person's ability to identify a whole object from a partially completed drawing of its form.

Mean—The mathematical average of all scores in a set of scores. The K-SNAP Composite Score has been standardized to have a mean of 100. The K-SNAP subtests have been standardized to have a mean of 10. The means are based on a comparison to others in the same age group. Standardizing in this way then allows the scores to be comparable across age groups.

Orientation—In psychiatry, the ability to locate oneself in one's environment with respect to time, place and people.

Reliability—The ability of a test to yield consistent, repeatable results.

Standard deviation—A measure of variability in a set of scores. The K-SNAP Composite Score has been standardized to have a standard deviation of 15. The subtests have been standardized to have a standard deviation of three. The standard deviations are based on a comparison to others in the same age group. Standardizing in this way then allows the scores to be comparable across age groups.

Standardization—The administration of a test to a sample group of people for the purpose of establishing scoring norms. Prior to the publication of the K-SNAP, it was standardized in 1988 using a sample of 2,000 adults and adolescents.

Validity—The ability of a test to measure accurately what it claims to measure.

The Number Recall subtest assesses sequential processing and short-term auditory memory. In this subtest, the examiner recites series of numbers and the examinee repeats the numbers.

The Four-Letter Words subtest measures the test taker's ability to solve problems and make plans. In this subtest, the examinee is asked to guess a secret word by analyzing a series of four-letter words that provide clues to the answer. It is the most complex of the subtests.

The K-SNAP is a relatively easy test to administer. Except for the Mental Status subtest, the test items are

presented on an easel, which is visually appealing to many test takers. Also, because the test is brief and includes a variety of tasks, the test takers often find the test engaging and interesting.

The K-SNAP is considered to be useful in evaluating elderly people, especially with regard to decline in fluid intelligence. The Mental Status subtest can also detect possible age-related impairment in mental functioning.

Compared to other neurological and cognitive assessments, there are smaller than usual differences in K-SNAP performance between African-American and Caucasian individuals, especially with regard to fluid intelligence. This cultural neutrality makes the K-SNAP a preferred method for testing African-Americans.

Results

The K-SNAP yields several scores, including raw scores, scaled scores, a composite score, and an impairment index. Raw scores and scaled scores are calculated for each of the four subtests. Raw scores are calculated first; they refer simply to the number of points that the examinee scored on a particular subtest. The raw scores are converted to scaled scores to simplify comparisons between the subtests and between examinees. The subtest scaled scores are standardized to have a mean of 10 and a standard deviation of three.

One composite score is obtained on the K-SNAP. The composite score has a mean of 100 and a standard deviation of 15 and is based on the scores of the four subtests.

The results of the Mental Status subtest are primarily of interest when working with middle-aged or elderly people, as well as people with neurological or cognitive impairments. Most people find the Mental Status subtest very easy, and they get most, if not all, of the items correct.

Some of the interpretation of the K-SNAP involves comparisons of performance on tasks of varying complexity. For example, Gestalt Closure is considered a less complex task than Number Recall. Someone who performs better on the more difficult Number Recall subtest may exhibit some kind of **brain** dysfunction. On the other hand, that person may simply prefer sequential processing tasks.

An impairment index is also calculated and provides an objective measure of cognitive and neurological impairment. The impairment index is based on the following four factors: the K-SNAP composite score; the test taker's performance on the Mental Status subtest; the difference between the scaled scores on the Number Recall and Gestalt Closure subtests; and the difference between the actual composite score and the predicted composite score based on the test taker's level of education. These four factors determine whether a more comprehensive assessment of impairment is necessary. For example, if an examinee has a composite score below 70, a low score on the Mental Status subtest, a large difference in performance in the Number Recall and Gestalt Closure subtests, and a difference of at least 24 points between the predicted and actual composite scores, there may be indications of impairment. One example of such impairment is damage to one hemisphere of the brain.

Overall, the K-SNAP has above-average to good reliability. As a mental status examination, it has been shown to have good validity as well. There have been no studies, however, demonstrating the K-SNAP's validity as a measure of neuropsychological impairment. Because the K-SNAP is based on similar theories and on the same standardization sample as other Kaufman tests, such as the KAIT, interpretation across the range of Kaufman tests is easier than comparing results from the K-SNAP to results from tests designed by other persons.

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Ali Fahmy, Ph.D.

Kava kava

Definition

Kava kava is a dioecious (having male and female reproductive parts of the plant on different individuals) shrub native to the Pacific islands. Its botanical name is *Piper methysticum*; it is a member of the Piperaceae, or pepper, family. It is also known as asava pepper or intoxicating pepper. The narcotic drink made from the roots of this shrub is also called kava kava. Kava kava has been widely recommended in recent years as a mild tranquilizer due to its painkilling properties. *As of 2002, howev-*

er, kava kava has been the subject of official safety warnings from the U. S. Food and Drug Administration (FDA) and its counterparts in Canada, France, Germany, Switzerland, and Spain.

Captain James Cook is credited with introducing kava kava to Europeans when he visited the South Pacific in 1773. Previously, the inhabitants of the Pacific islands used kava kava as a ceremonial beverage. It was consumed at weddings, funerals, and birth rituals, and it was offered to honored guests. Kava kava was also drunk as part of healing rituals. The first commercial products containing kava kava were offered to European consumers around 1860.

As of 2001, kava kava ranked ninth in sales of all herbal dietary preparations sold in the United States through mainstream retailers, with total sales of $15 million. Health food stores, health professionals, and mail order firms accounted for another $15 million in sales of kava kava.

KEY TERMS

Dioecious—A category of plants that reproduce sexually but have male and female reproductive organs on different individuals. Kava kava is a dioecious plant.

Kavalactones—Medically active compounds in kava root that act as local anesthetics in the mouth and as minor tranquilizers

Kavapyrones—Compounds in kava root that act as muscle relaxants and anticonvulsants.

Purpose

The German Commission E, a panel of physicians and pharmacists that reviews the safety and efficacy of herbal preparations, at one time approved the use of kava kava as a nonprescription dietary supplement for the relief of nervous anxiety, **stress**, and restlessness. That approval was withdrawn in the fall of 2001.

In addition to relief of stress and anxiety, kava kava has also been recommended by health care providers for **insomnia**, sore or stiff muscles, toothache or sore gums, **attention-deficit/hyperactivity disorder**, menstrual cramps, uncontrolled epilepsy, and jet lag.

Description

The beverage form of kava kava was traditionally prepared in the Pacific islands by chewing the roots of the kava plant and spitting them into a bowl. The active compounds, known as kavalactones and kavapyrones, are found primarily in the root of the plant and are activated by human saliva. Contemporary Pacific islanders prepare kava kava by pounding or grinding the roots and mixing them with coconut milk or water. Modern Western manufacturers use alcohol or acetate in making liquid kava preparations. Kava kava is also available in capsules, tablets, powdered, or crushed forms. Experts in herbal medicine recommended the use of kava preparations standardized to contain 70% kavalactones.

Kavalactones are chemicals that affect the **brain** in the same way as benzodiazepines such as valium, which is prescribed for depression or anxiety. Kavalactones cause the tongue or gums to feel numb. Kavapyrones are chemicals that have anticonvulsant and muscle relaxant properties.

Recommended dosage

Kava kava should never be given to children, particularly in view of recent health warnings concerning adults.

The usual dose of kava kava that has been recommended to relieve stress or insomnia in adults is 2–4 g of the plant boiled in water, up to three times daily. Alternately, 60–600 mg of kavalactones in a standardized formula could be taken per day.

Precautions

Before 2002, the usual precautions regarding kava kava stated that it should not be used at all by pregnant or lactating women, or by any individual when driving or operating heavy machinery. The American Herbal Products Association (AHPA) advised consumers in 1997 not to take kava kava for more than three months at a time, and not to exceed the recommended dosages. In light of more recent findings, however, it may be prudent to completely avoid preparations of or products containing kava kava.

Side effects

Prior to 2002, most reports of side effects from kava kava concerned relatively minor problems, such as numbness in the mouth, headaches, mild dizziness, or skin rashes. In the nineteenth-century, missionaries to the Pacific islands noted that people who drank large quantities of kava kava developed yellowish scaly skin. A recent study found the same side effect in test subjects who took 100 times the recommended dose of the plant.

As of 2002, kava kava has also been associated with causing damage to the liver, including hepatitis, cirrhosis, and liver failure. Most of the research on kava kava has been done in Europe, where the herb is even more popular than it is in the United States. By the late fall of 2001, there had been at least 25 reports from different European countries of liver damage caused by kava kava; French health agencies reported one death and four patients requiring liver transplants in connection with kava kava consumption. On December 19, 2001, the Medwatch advisory of the FDA posted health warnings about the side effects of kava kava; on January 16, 2002, Health Canada advised Canadians to avoid all products containing the herb. France banned the sale of preparations containing kava kava in February 2002. The U. S. National Center for Complementary and Alternative Medicine (NCCAM) has put two research studies of kava kava on hold while awaiting further action by the FDA. NCCAM advised consumers in the United States on January 7, 2002, to avoid products containing kava.

In addition to causing liver damage, kava kava appears to produce psychological side effects in some patients. A team of Spanish physicians has reported that beverages containing kava kava may cause anxiety, depression, and insomnia. In addition, kava kava may cause tremors severe enough to be mistaken for symptoms of Parkinson's disease.

Interactions

Kava kava has been shown to interact adversely with beverage alcohol and with several categories of prescription medications. It increases the effect of **barbiturates** and other psychoactive medications; in one case study, a patient who took kava kava together with **alprazolam** (a benzodiazepine used to treat anxiety) went into a coma. It may produce dizziness and other unpleasant side effects if taken together with phenothiazines (medications used to treat **schizophrenia**). Kava kava has also been reported to reduce the effectiveness of levodopa, a drug used in the treatment of Parkinson's disease. To avoid potential reactions with prescription medications, people should inform their physician if they are taking kava kava.

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NIH Office of Dietary Supplements. Building 31, Room 1B25. 31 Center Drive, MSC 2086. Bethesda, MD 20892-2086. (301) 435-2920. Fax: (301) 480-1845. <www.odp.od.nih.gov/ods>.

OTHER

American Botanical Council (ABC). P.O. Box 144345, Austin, TX 78714-4345. (512) 926-4900. Fax: (512) 926-2345. <www.herbalgram.org>.

FDA Center for Food Safety and Applied Nutrition. <www.fda.gov/medwatch/safety/2001/kava.htm>.

NIH National Center for Complementary and Alternative Medicine (NCCAM) Clearinghouse. P. O. Box 8218, Silver Spring, MD 20907-8218. TTY/TDY: (888) 644-6226. Fax: (301) 495-4957. <www.nccam.nih.gov>.

Rebecca J. Frey, Ph.D.

Kleptomania

Definition

Kleptomania is an impulse control disorder characterized by a recurrent failure to resist stealing.

Description

Kleptomania is a complex disorder characterized by repeated, failed attempts to stop stealing. It is often seen in patients who are chemically dependent or who have a coexisting mood, anxiety, or eating disorder. Other coexisting mental disorders may include major depression, panic attacks, **social phobia**, **anorexia nervosa**, **bulim-**

ia nervosa, substance abuse, and **obsessive-compulsive disorder**. People with this disorder have an overwhelming urge to steal and get a thrill from doing so. The recurrent act of stealing may be restricted to specific objects and settings, but the affected person may or may not describe these special preferences. People with this disorder usually exhibit guilt after the theft.

Detection of kleptomania, even by significant others, is difficult and the disorder often proceeds undetected. There may be preferred objects and environments where theft occurs. One theory proposes that the thrill of stealing helps to alleviate symptoms in persons who are clinically depressed.

Causes and symptoms

Causes

The cause of kleptomania is unknown, although it may have a genetic component and may be transmitted among first-degree relatives. There also seems to be a strong propensity for kleptomania to coexist with obsessive-compulsive disorder, bulimia nervosa, and clinical depression.

Symptoms

The handbook used by mental health professionals to diagnose mental disorders is the ***Diagnostic and Statistical Manual of Mental Disorders***. Published by the American Psychiatric Association, the *DSM* contains diagnostic criteria and research findings for mental disorders. It is the primary reference for mental health professionals in the United States. The 2000 edition of this manual (fourth edition, text revision), known as the *DSM-IV-TR,* lists five diagnostic criteria for kleptomania:

- Repeated theft of objects that are unnecessary for either personal use or monetary value.
- Increasing tension immediately before the theft.
- Pleasure or relief upon committing the theft.
- The theft is not motivated by anger or vengeance, and is not caused by a delusion or hallucination.
- The behavior is not better accounted for by a **conduct disorder**, **manic episode**, or antisocial personality disorder.

Demographics

Studies suggest that 0.6% of the general population may have this disorder and that it is more common in females. In patients who have histories of obsessive-compulsive disorder, some studies suggest a 7% correlation with kleptomania. Other studies have reported a particularly high (65%) correlation of kleptomania in patients with bulimia.

KEY TERMS

Anorexia nervosa—An eating disorder characterized by an intense fear of weight gain accompanied by a distorted perception of one's own underweight body.

Bulimia nervosa—An eating disorder characterized by binges in which large amounts of food are consumed, followed by forced vomiting.

Cognitive-behavioral therapy—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing oneís behaviors accordingly.

Obsessive-compulsive disorder—Disorder in which the affected individual has an obsession (such as a fear of contamination, or thoughts he or she doesn't like to have and can't control) and feels compelled to perform a certain act to neutralize the obsession (such as repeated handwashing).

Panic disorder—An anxiety disorder in which an individual experiences sudden, debilitating attacks of intense fear.

Phobia—Irrational fear of places, things, or situations that lead to avoidance.

Rational emotive therapy—A form of psychotherapy developed by Albert Ellis and other psychotherapists based on the theory that emotional response is based on the subjective interpretation of events, not on the events themselves.

Diagnosis

Diagnosing kleptomania is usually difficult since patients do not seek medical help for this complaint, and initial psychological assessments may not detect it. The disorder is often diagnosed when patients seek help for another reason, such as depression, bulimia, or for feeling emotionally unstable (labile) or unhappy in general (dysphoric). Initial psychological evaluations may detect a history of poor parenting, relationship conflicts, or acute stressors—abrupt occurrences that cause stress, such as moving from one home to another. The recurrent act of stealing may be restricted to specific objects and settings, but the patient may or may not describe these special preferences.

Treatments

Once the disorder is suspected and verified by an extensive psychological interview, therapy is normally directed towards impulse control, as well as any accompanying mental disorder(s). Relapse prevention strategies, with a clear understanding of specific triggers, should be stressed. Treatment may include psychotherapies such as **cognitive-behavioral therapy** and **rational emotive therapy**. Recent studies have indicated that **fluoxetine** (Prozac) and **naltrexone** (Revia) may also be helpful.

Prognosis

Not much solid information is known about this disorder. Since it is not usually the presenting problem or chief complaint, it is frequently not even diagnosed. There are some case reports that document treatment success with antidepressant medications, although as with almost all psychological disorders, the outcomes vary.

Prevention

There is little evidence concerning prevention. A healthy upbringing, positive intimate relationships, and management of acutely stressful situations may lower the incidence of kleptomania and coexisting disorders.

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Klonopin *see* **Clonazepam**

K-SNAP *see* **Kaufman Short Neurological Assessment Procedure**

L

Laboratory tests *see* **Urine drug screening**

Lamictal *see* **Lamotrigine**

Lamotrigine

Definition

Lamotrigine is an anticonvulsant drug commonly used to prevent **seizures**. It is also used as a mood stabilizer in some people with bipolar (manic-depressive) disorder. In the United States, lamotrigine is available under the trade name of Lamictal.

Purpose

Lamotrigine is used to prevent seizures in individuals with seizure disorders. It is also used as a mood stabilizer in people with **bipolar disorder**.

Description

The United States Food and Drug Administration (FDA) approved Lamotrigine in 1994. This drug appears to suppress the activity of neurons (nerve cells) in the **brain**. By stabilizing neurons, lamotrigine prevents seizure activity and may also stabilize abnormal mood swings.

Lamotrigine is available as both oral and chewable tablets. It is broken down in the liver.

Recommended dosage

The dosage of lamotrigine varies depending upon the age and weight of the patient, other medications that the patient is taking, and whether the patient has heart, liver, or kidney disease. It is common for patients to start with a low dosage of lamotrigine. The dosage is then increased slowly over several weeks to help prevent side effects. The dosage may be adjusted frequently by the prescribing physician.

A common dose for an adult who takes no other medications and has no other diseases is 150–250 mg taken twice daily.

Precautions

A serious and permanently disfiguring rash may occur as a result of lamotrigine. The rash, which is a symptom of a systemic reaction to the drug, may be life-threatening. If a rash occurs, a doctor should be contacted immediately, and the drug stopped. People who have experienced any kind of rash while taking lamotrigine should never take the drug again.

Lamotrigine should be used with physician supervision after assessing the risks and benefits in people with heart, kidney, or liver disease. The dosage is usually reduced in these individuals.

Side effects

Side effects that occur in more than 10% of people taking lamotrigine are: headache, dizziness, unsteadiness while walking, blurred vision, double vision, nausea, cold-like symptoms involving runny noses or sore throats, and infections.

Although relatively rare, any rash that develops while taking lamotrigine should be evaluated by a health care professional, since life-threatening rashes may occur.

Other side effects include confusion, impaired memory, **sleep disorders**, nonspecific pain all over the body, and disruption of menstrual cycles.

Interactions

Some drugs can decrease the levels of lamotrigine in the body. This may make the drug less effective. Examples include **carbamazepine**, phenobarbital, primidone, phenytoin, and **valproic acid**. Interestingly, val-

KEY TERMS

Bipolar disorder—A mental disorder characterized by dramatic, and sometimes rapid mood swings, resulting in both manic and depressive episodes; formerly called manic-depressive disorder.

Folic acid—An essential B-vitamin that humans obtain through diet.

Manic—Referring to mania, a state characterized by excessive activity, excitement or emotion.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals approximately 0.035 ounces.

proic acid and its close relative, **divalproex sodium**, have also been reported to increase lamotrigine levels in some people, which could increase the side effects of the drug. When lamotrigine and valproic acid are used together, there is a greater chance that a serious rash may develop. Very specific dosage guidelines must be followed when these two drugs are used at the same time.

Lamotrigine may increase the levels of carbamazepine in the body, increasing adverse effects associated with carbamazepine.

An increased risk of certain side effects may occur if lamotrigine is used with drugs that inhibit folic acid synthesis, such as methotrexate.

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Kelly Karpa, RPh, Ph.D.

Lavender

Definition

Lavender is the shrub-like aromatic plant, *Lavandula officinalis*, sometimes called *Lavandula vera* or true lavender.

Purpose

Lavender is a mild sedative and antispasmodic. The essential oil derived from lavender is used in **aromatherapy** to treat anxiety, difficulty sleeping, nervousness, and restlessness. Other preparations of the plant are taken internally to treat sleep disturbances, stomach complaints, loss of appetite, and as a general tonic.

Description

Lavender is a shrubby evergreen bush that grows to about 3 feet (1 m) tall and 4 feet (1.4 m) in diameter. The plant produces aromatic spiky flowers from June to September. An essential oil used for healing and in perfume is extracted from the flowers just before they open.

Lavender is native to the Mediterranean region and is cultivated in temperate regions across the world. There are many species and subspecies. The preferred lavender for medicinal use is *L. officinalis* or true lavender. In Europe, lavender has been used as a healing herb for centuries. It was a prominent component of smelling salts popular with women in the late 1800s.

Lavender is used both externally and internally in healing. Externally the essential oil is used in aromatherapy as a relaxant and to improve mood. Aromatherapy can be facilitated through massage, used in the bath, in potpouri jars, and burned in specially-dsigned oil burners. Lavender is also used to treat **fatigue**, restlessness, nervousness, and difficulty sleeping. Pillows stuffed with lavender have been used as a sleep aid in Europe for many years. Lavender oil applied to the forehead and temples is said to ease headache.

Researchers have isolated the active compounds in lavender. The most important of these is an aromatic volatile oil. Lavender also contains small amounts of coumarins, compounds that dilate (open up) the blood vessels and help control spasms. Some modern scientific research supports the claim that lavender is effective as a mild sedative and a calming agent. In one Japanese study, people exposed to the odor of lavender were found to show less mental **stress** and more alertness than those not exposed to the fragrance when evaluated by psychological tests. In a peer-reviewed British study, when the sleeping room was perfumed with lavender, elderly nursing home residents with **insomnia** slept as well as they did when they took sleeping pills and better than they did when they were given neither sleeping pills nor exposed to lavender fragrance.

Other external uses of the essential oil of lavender are as an antiseptic to disinfect wounds. When used on wounds, lavender oil often is combined with other essential oil extracts to enhance its antiseptic and dehydrating

properties. Lavender oil added to bathwater is believed to stimulate the circulation.

Taken internally as a tea made from lavender flowers or as a few drops of lavender oil on a sugar cube, this herb is used as a mild sedative and antispasmodic. The German Federal Health Agency's Commission E, established to independently review and evaluate scientific literature and case studies pertaining to medicinal plants, has approved the use of lavender tea or lavender oil on a sugar cube to treat restlessness and insomnia. Despite conflicting scientific claims, this organization has also endorsed the internal use of lavender for stomach upsets, loss of appetite, and excess gas. Animal research confirms that lavender oil has an antispasmodic effect on smooth muscle of the intestine and uterus. These results have not been confirmed in humans.

Recommended dosage

Lavender tea is made by steeping 1 to 2 teaspoons of flowers per cup of boiling water. One cup of tea can be drunk three times a day. Alternatively, 1 to 4 drops of lavender oil can be placed on a sugar cube and eaten once a day. Externally, a few drops of oil can be added to bath water or rubbed on the temples to treat headache. Like any herbal product, the strength of the active ingredients can vary from batch to batch, making it difficult to determine exact dosages.

Precautions

The use of lavender, either alone or in combination with other herbs, is not regulated by the United States Food and Drug Administration. Unlike pharmaceuticals, herbal and dietary supplements are not subjected to rigorous scientific testing to prove their claims of safety and effectiveness. The strength of active ingredients varies from manufacturer to manufacturer, and the label may not accurately reflect the contents.

Particular problems with lavender oil revolve around substitution of oil from species of lavender other than *Lavandula officinalis*, the preferred medicinal lavender. Most often true lavender oil is adulterated with less expensive lavadin oil. Lavadin oil comes from other species of lavender. It has a pleasant lavender odor, but its chemical compositions, and thus its healing actions, are different from true lavender oil. People purchasing lavender oil or tonics containing lavender should be alert to substitutions.

Side effects

When used in the recommended dosage, lavender is not considered harmful. Some people have reported developing contact dermatitis (a rash) when lavender oil is used directly on the skin.

KEY TERMS

Antispasmodic—A medication or preparation given to relieve muscle or digestive cramps.

Interactions

There are no studies on interactions of lavender with conventional pharmaceuticals. Traditionally lavender has been used in combination with other herbs such as tea oil and lemon balm without adverse interactions.

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Tish Davidson, A.M.

Learning disabilities *see* **Learning disorders**

Learning disorders

Definition

Learning disorders, or learning disabilities, are disorders that cause problems in speaking, listening, reading, writing, or mathematical ability.

Description

A learning disability, or specific developmental disorder, is a disorder that inhibits or interferes with the skills of learning. Under federal law, public schools consider a child to be learning disabled if his or her level of academic achievement is two or more years below the

KEY TERMS

IQ—Intelligence quotient, or a measure of the intelligence of an individual based on the results of a written test.

standard for age and IQ level. The Fourth Edition Text Revision of the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*, a handbook that mental health professionals use to diagnose mental disorders) uses the term *learning disorder* and defines this as cognitive difficulties arising from **brain** dysfunction.

It is estimated that 5% to 20% of school-age children in the United States, mostly boys, suffer from learning disabilities. Often, learning disabilities appear together with other disorders, such as **attention-deficit/hyperactivity disorder** (ADHD). Learning disorders are thought to be caused by irregularities in the functioning of certain parts of the brain. Evidence suggests that these irregularities are often inherited (i.e. a person is more likely to develop a learning disability if other family members have them). Learning disabilities are also associated with certain conditions occurring during fetal development or birth, including maternal use of alcohol, drugs, and tobacco; exposure to infection; injury during birth; low birth weight; and sensory deprivation.

Aside from underachievement, other warning signs that a person may have a learning disability include overall lack of organization, forgetfulness, and taking unusually long amounts of time to complete assignments. In the classroom, the child's teacher may observe one or more of the following characteristics: difficulty paying attention, unusual sloppiness and disorganization, social withdrawal, difficulty working independently, and trouble switching from one activity to another. In addition to the preceding signs, which relate directly to school and schoolwork, certain general behavioral and emotional features often accompany learning disabilities. These include impulsiveness, restlessness, distractibility, poor physical coordination, low tolerance for frustration, low self-esteem, daydreaming, inattentiveness, and anger or sadness.

Types of learning disabilities

Learning disabilities are associated with brain dysfunctions that affect a number of basic skills. Perhaps the most fundamental is sensory-perceptual ability—the capacity to take in and process information through the senses. Difficulties involving vision, hearing, and touch will have an adverse effect on learning. Although learning is usually considered a mental rather than a physical pursuit, it involves motor skills, and it can also be impaired by problems with motor development. Other basic skills fundamental to learning include memory, attention, and language abilities.

The three most common academic skill areas affected by learning disabilities are reading, writing, and arithmetic. Some sources estimate that between 60% and 80% of children diagnosed with learning disabilities have reading as their only or main problem area. Learning disabilities involving reading have traditionally been known as dyslexia; currently, the preferred term is *reading disorder*. A wide array of problems is associated with **reading disorder**, including difficulty identifying groups of letters, problems relating letters to sounds, reversals and other errors involving letter position, chaotic spelling, trouble with syllabication (breaking words into syllables), failure to recognize words, hesitant oral reading, and word-by-word rather than contextual reading.

Writing disabilities, known as dysgraphia or **disorder of written expression**, include problems with letter formation and writing layout on the page, repetitions and omissions, punctuation and capitalization errors, "mirror writing" (writing right to left), and a variety of spelling problems. Children with dysgraphia typically labor at written work much longer than their classmates, only to produce large, uneven writing that would be appropriate for a much younger child.

Learning abilities involving math skills, generally referred to as dyscalcula (or dyscalculia) or **mathematics disorder**, usually become apparent later than reading and writing problems—often at about the age of eight. Children with dyscalcula may have trouble counting, reading and writing numbers, understanding basic math concepts, mastering calculations, and measuring. This type of disability may also involve problems with nonverbal learning, including spatial organization.

In order to meet the criteria established by the American Psychiatric Association (APA) for these various diagnoses, the child's skills in these areas must be significantly below that of their peers on standardized tests (taking age, schooling, and level of intelligence into account), and the disorders must significantly interfere with academic achievement and/or daily living.

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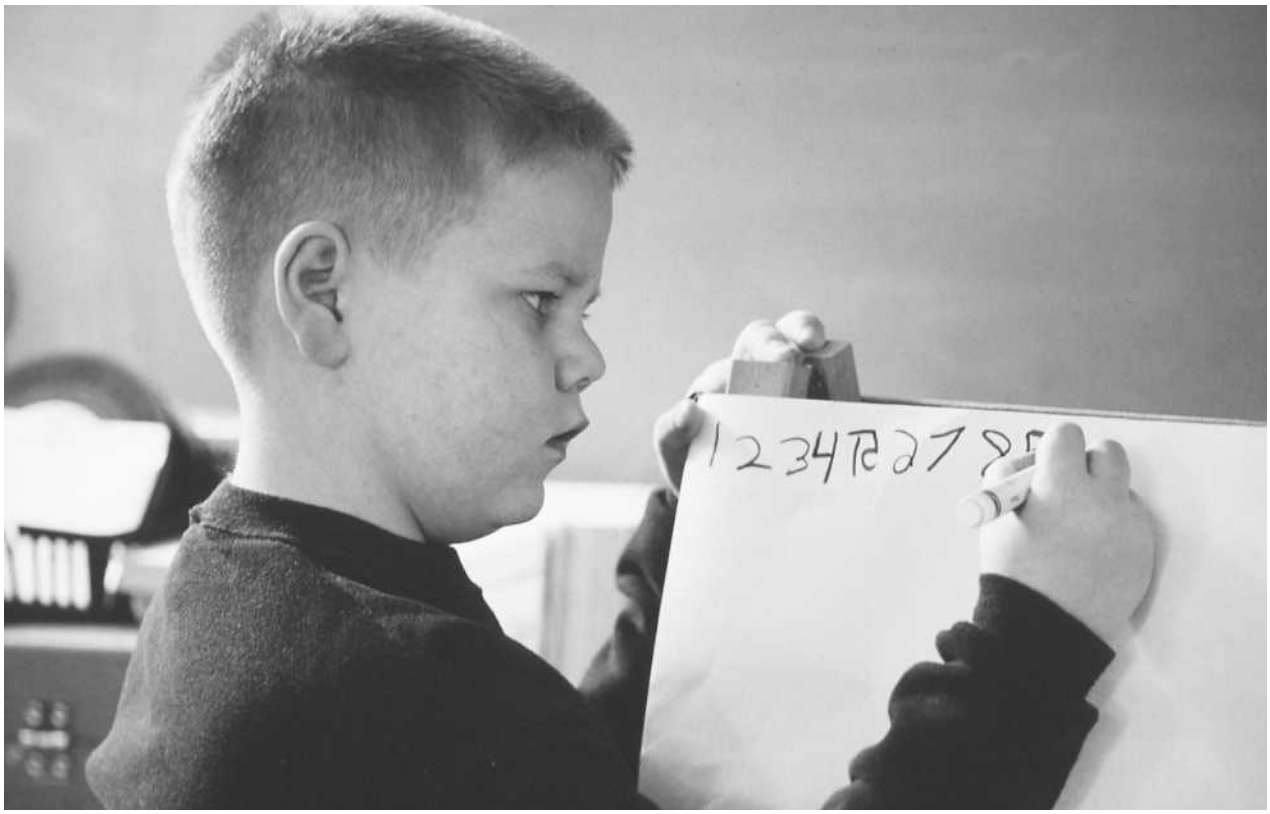

This eight-year-old boy has a learning disability that causes him to write some of these numbers backwards. *(Ellen B. Senisi. Photo Researchers, Inc. Reproduced by permission.)*

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Learning Disabilities Association of America (formerly ACLD, the Association for Children and Adults with Learning Disabilities). 4156 Library Road, Pittsburgh, PA 15234-1349. Telephone: (412) 341-1515. Web site: <http://www.ldanatl.org>.

National Center for Learning Disabilities. 381 Park Avenue South Suite 1401, New York, NY 10016. Telephone: (212) 545-7510, or toll-free at (888) 575-7373. Web site: <http://www.ncld.org>.

Librium *see* **Chlordiazepoxide**

Lidone *see* **Molindone**

Light therapy

Definition

Light therapy refers to two different categories of treatment, one used in mainstream medical practice and the other in alternative/complementary medicine. Mainstream light therapy (also called phototherapy) includes the use of ultraviolet light to treat psoriasis and other skin disorders, and the use of full-spectrum or bright light to treat **seasonal affective disorder** (SAD). Light therapy for SAD was first introduced in the 1980s and is now a widely approved form of treatment for the disorder.

Light therapy in alternative or complementary approaches includes such techniques as the use of colored light or colored gemstones directed at or applied to various parts of the body. In some alternative forms of light therapy, the person visualizes being surrounded by and breathing in light of a particular color.

KEY TERMS

Aura—An energy field that is thought to emanate from the human body and to be visible to people with special psychic or spiritual powers.

Chakra—One of seven major energy centers in the body, according to traditional systems of Eastern medicine. The chakras are associated with the seven colors of light in the rainbow.

Chromatherapy—An alternative form of light therapy in which colored light is directed toward a specific chakra or part of the body in order to heal or to correct energy imbalances. Practitioners of chromatherapy are sometimes called chromapaths.

Dawn simulation—A form of light therapy in which the patient is exposed while asleep to gradually brightening white light over a period of an hour and a half.

Lux—The International System unit for measuring illumination, equal to one lumen per square meter.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Phototherapy—Another name for light therapy in mainstream medical practice.

Seasonal affective disorder (SAD)—A mood disorder characterized by depression, weight gain, and sleepiness during the winter months. An estimated 4–6% of the population of Canada and the northern United States suffers from SAD.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Purpose

Mainstream light therapy

The purpose of light therapy in mainstream psychiatric treatment is the relief of seasonal affective disorder, a form of depression most often associated with shortened daylight hours in northern latitudes from the late fall to the early spring. It is occasionally employed to treat such sleep-related disorders as **insomnia** and jet lag. Recently, light therapy has also been found effective in the treatment of such nonseasonal forms of depression as **bipolar disorder**. Light therapy for SAD and nonseasonal forms of depression is thought to work by triggering the brain's production of serotonin, a neurotransmitter related to mood disorders. Other researchers think that light therapy may relieve depression or jet lag by resetting the body's circadian rhythm, or inner biological clock.

In dermatology, ultraviolet (UV) light therapy is used to treat rashes, psoriasis, other skin disorders, and jaundice. Outpatient treatment for psoriasis usually requires three treatment sessions per week until the skin clears, which takes about seven weeks.

Alternative light therapies

Alternative light therapies are generally used to treat energy imbalances in the seven major chakras. Chakras are defined in Eastern systems of traditional medicine as energy centers in the human body located at different points along the spinal column. Each chakra is thought to absorb a certain vibration of light in the form of one of the seven colors of the rainbow, and to distribute this color energy through the body. When a specific chakra is blocked, light in the color associated with that chakra can be used to unblock the energy center and balance the flow of energy in the body.

The seven major chakras in the body and their associated colors are:

- red: the root chakra, located at the base of the spine
- orange: the sacral chakra, located in the small of the back
- yellow: the solar plexus chakra
- green: the heart chakra
- blue: the throat or thyroid chakra
- indigo: the so-called "third eye," located in the head at the level of the pineal gland
- violet or white: the crown chakra, located at the level of the pituitary gland

Alternative forms of light therapy also use colored light to heal different parts of the body associated with the various chakras. For example, yellow light would be used to heal digestive disorders, green to treat the circulatory system, and so on. Concentrating colored light into a narrow beam or applying a colored gemstone is thought to stimulate the **acupuncture** or acupressure points that govern the various organ systems of the body. This application of light therapy is sometimes called chromatherapy.

Precautions

Patients with eye disorders should consult an ophthalmologist before being treated with any form of phototherapy. Patients who are taking medications that make their skin sensitive to UV rays or bright light should also consult their health care provider. Although there are no reports of permanent eye damage from either light box therapy or UV treatment for skin disorders, patients sometimes experience headaches, dry eyes, mild sunburn, or **fatigue**. These problems can usually be relieved by adjusting the length of time for light treatments and by using a sunscreen and nose or eye drops. Lastly, patients who should have UV treatment for skin disorders should receive it from a board-certified dermatologist or other licensed health care professional; they should not attempt to treat themselves with sunlamps or similar tanning appliances.

There are no precautions needed for alternative light therapies.

Description

Mainstream light therapy

Mainstream phototherapy for skin disorders involves the exposure of the affected areas of skin to ultraviolet light. It is most often administered in an outpatient clinic or doctor's office. Light therapy for seasonal affective disorder and other forms of depression can be self-administered at home or in a private room in the workplace. The patient sits in front of a light box mounted on or near a desk or table for a period of time each day ranging from 15 minutes to several hours, depending on the severity of the SAD symptoms. Some SAD patients may have two or three sessions of light therapy each day. Treatment typically begins in the fall, when the days grow noticeably shorter, and ends in the spring.

The light box itself may be equipped with full-spectrum bulbs, which do emit UV rays as well as visible light; or it may use bulbs that filter out the UV rays and emit bright light only. Most light boxes emit light ranging from 2500–10,000 lux, a lux being a unit of light measurement equivalent to 1 lumen per square meter. For purposes of comparison, average indoor lighting is 300–500 lux, and the sunlight outdoors on a sunny day in summer is about 100,000 lux. Patients are instructed to sit facing the light box but to avoid staring directly at it. They can read or work at their desk while sitting in front of the light box. Light boxes cost between $200 and $500, but can often be rented from medical supply companies.

Newer forms of light therapy for SAD include the light visor, which resembles a baseball cap with a light source attached underneath the front of the device, above the wearer's eyes. The light visor allows the patient to walk or move about while receiving light treatment. Another new treatment is dawn simulation, which appears to be especially helpful for SAD patients who have difficulty getting up in the morning. In dawn simulation, the lighting fixture is programmed to turn gradually from dim to brighter light to simulate the sunrise. Dawn simulation is started around 4:30 or 5 o'clock on the morning, while the patient is still asleep.

Alternative light therapies

Chromatherapy may be administered in several different ways. The first step is determining the source or location of the patient's energy imbalance. Some color therapists or chromapaths are sensitive to the colors in the aura, or energy field surrounding a person's physical body that is invisible to most people. Dark or muddy colors in the aura are thought to indicate the locations of energy imbalance. Another technique involves suspending a quartz crystal on a pendulum over each chakra while the patient lies on a table or on the floor. The crystal swings freely if the chakra is open and energy is moving normally, but stops or moves irregularly if the chakra is blocked.

In the second stage of treatment, colored light is directed at specific areas of the body. The chromapath may use either colored light bulbs or may filter white light through a colored plastic filter. The red, orange, and yellow rays are thought to enter the body more effectively through the soles of the feet; patients receiving these colors of light may be asked to sit on the floor with their bare feet 12–14 inches from the light source. The green ray is thought to enter through the solar plexus and the blue, indigo, and violet rays through the crown of the head. Blue light can be used to irradiate the whole body for the relief of physical pain, and violet light can be similarly used to relieve nervous strain and mental disorders.

Another form of colored light therapy involves the use of gemstones in the colors appropriate to each chakra. The crystal structures of gemstones are thought to reflect and transmit energy vibrations, including color vibrations. In gemstone treatment, the chromapath first cleanses the patient's aura with a clear quartz crystal and then places colored gemstones (usually semiprecious rather than expensive precious stones) on the parts of the body corresponding to the location of the chakras while the patient is lying on his or her back or stomach. The colored stones are thought to both cleanse the aura and recharge the energy centers.

A third form of colored light therapy is called color breathing or color visualization. It can be self-administered at home or any other private space. The patient sits

Light therapy is a common treatment for seasonal affective disorder (SAD)— depression caused by change in seasons and decreased sunlight. People who undergo light therapy may sit in front of a light box mounted on or near a desk or table for a period of time each day ranging from 15 minutes to several hours, depending on the severity of the SAD symptoms. Treatment typically begins in the fall, when the days grow noticeably shorter, and ends in the spring. *(Stock Boston, Inc. Reproduced by permission.)*

in a chair with both feet on the floor, or sits on the floor in the lotus position. He or she then breathes slowly and rhythmically while visualizing being surrounded by light of the appropriate color and breathing in that color. The patient may also repeat a verbal affirmation related to the color, such as "The orange ray is filling me with vitality and joy," or "The violet ray is healing every part of my being."

Preparation

Patients should consult their health care provider before mainstream phototherapy, in order to determine possible sensitivity to bright light and adjust medication dosages if necessary.

Holistic and alternative practitioners usually ask patients to bathe or shower before chromatherapy, and to wear loosely fitting white or neutral-colored clothing. Washing is considered necessary to remove any negative energies that the patient has picked up from other people or from the environment. Wearing light-colored loose clothing is thought to minimize interference with the vibrations from the colored light or gemstones. The final step in preparation is a brief period of **meditation** or creative visualization for the practitioner as well as the patient. This step helps to create an atmosphere of calm and relaxation for the treatment.

Aftercare

No aftercare is necessary for mainstream light treatments.

Practitioners of alternative light therapies recommend that patients sit or rest quietly for a few minutes after the treatment rather than returning abruptly to their daily routines. This brief rest is thought to maximize the benefits of the treatment.

Risks

As was previously mentioned, mainstream light therapies may produce minor side effects (headache, insomnia, mild sunburn or skin irritation, dry eyes) in some patients. In addition, some patients receiving phototherapy for SAD may experience hypomania, which is a feeling of euphoria or an exaggeratedly "upbeat" mood. As with the physical side effects, hypomania can usually be managed by adjusting the frequency or length of light therapy sessions.

There are no known risks associated with alternative light therapies.

Normal results

Normal results for mainstream light treatments are clearing of the skin disorder or a lifting of depressed mood or jet lag.

Normal results for alternative light therapies include a sense of heightened energy and relief from negative thoughts or preoccupations. Some chromapaths also consider relief of physical pain or symptoms to be normal results for chromatherapy.

See also Circadian rhythm sleep disorder

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American Holistic Medicine Association. Suite 201, 4101 Lake Boone Trail, Raleigh, NC 27607.

Colour Therapy Association. P. O. Box 16756, London SW20 8ZW, United Kingdom.

International Society for the Study of Subtle Energies and Energy Medicine (ISSSEEM). 356 Goldco Circle. Golden, CO 80401. (303) 278-2228. <www.vitalenergy.com/ISSSEEM>.

National Depressive and Manic Depressive Association. 730 Franklin Street, Suite 501, Chicago, IL 60610. (800) 826-3632. <www.ndmda.org>.

National Institute of Mental Health. Mental Health Public Inquiries, 5600 Fishers Lane, Room 15C-05, Rockville, MD 20857. (301) 443-4513. (888) 826-9438. <www.nimh.nih.gov>.

Society for Light Treatment and Biological Rhythms. 824 Howard Ave., New Haven, CT 06519. Fax (203) 764-4324. <www.sltbr.org>. E-mail: sltbr@yale.edu.

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Limbic system *see* **Brain**

Lithium carbonate

Definition

Lithium is a naturally occurring element that is classified as an anti-manic drug. It is available in the United States under the brand names Eskalith, Lithonate, Lithane, Lithotabs, and Lithobid. It is also sold under its generic name.

Purpose

Lithium is commonly used to treat mania and bipolar depression (manic-depression or **bipolar disorder**). Less commonly, lithium is used to treat certain mood disorders, such as **schizoaffective disorder** and aggressive behavior and emotional instability in adults and children. Rarely is lithium taken to treat depression in the absence of mania. When this is the case, it is usually taken in addition to other antidepressant medications.

Description

Lithium salts have been used in medical practice for about 150 years. Lithium salts were first used to treat gout. It was noted in the 1880s that lithium was somewhat effective in the treatment of depression, and in the 1950s lithium was seen to improve the symptoms of bipolar disease. The way lithium works in the body is unclear, but its therapeutic benefits are probably related to its effects on other electrolytes such as sodium, potassium, magnesium, and calcium. Lithium is taken either as lithium carbonate tablets or capsules or as lithium citrate syrup.

The therapeutic effects of lithium may appear slowly. Maximum benefit is often not evident for at least two weeks after starting the drug. People taking lithium should be aware of this and continue taking the drug as directed even if they do not see immediate changes in mood.

Lithium is available in 300-mg tablets and capsules, 300-mg and 450-mg sustained-release tablets, and a syrup containing approximately 300 mg per teaspoonful.

Recommended dosage

Depending on the patient's medical needs, age, weight, and kidney function, doses of lithium can range from 600 to 2,400 mg per day, although most patients will be stabilized on 600 to 1,200 mg per day. Patients who require large amounts of lithium often benefit from the addition of another anti-manic drug, which may allow the dose of lithium to be lowered.

Generally, lithium is taken two or three times daily. However, the entire dose may be taken at once if the physician believes that a single daily-dose program will increase patient **compliance**. The single dose schedule is especially helpful for people who are forgetful and may skip doses on a multiple dose schedule. Additionally, evidence indicates that once-daily doses are associated with fewer side effects.

More than with any other drug used in the treatment of mental disorders, it is essential to maintain lithium blood levels within a certain narrow range to derive the maximum therapeutic benefit while minimizing serious negative side effects. It is important that people taking lithium have their blood levels of lithium measured at regular intervals.

KEY TERMS

Bipolar disease—A mental disorder characterized by periods of mania alternating with periods of depression.

Compliance—In medicine or psychiatry, cooperation with a treatment plan or schedule of medications.

Electrocardiograph—(EKG) A test that measures the electrical activity of the heart as it beats. An abnormal EKG can indicate possible cardiac disease.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Schizoaffective disorder—A mental disorder that shows a combination of symptoms of mania and schizophrenia.

Thyroid—A gland in the neck that produces the hormone thyroxin, which is responsible for regulating metabolic activity in the body. Supplemental synthetic thyroid hormone is available as pills taken daily when the thyroid fails to produce enough hormone.

Precautions

Because lithium intoxication may be serious and even life-threatening, blood concentrations of lithium should be measured weekly during the first four weeks of therapy and less often after that.

Patients taking lithium should have their thyroid function monitored and maintain an adequate sodium (salt) and water balance. Lithium should not be used or used only with very close physician supervision by people with kidney impairment, heart disease, and other conditions that affect sodium balance. Dosage reduction or complete discontinuation may be necessary during infection, diarrhea, vomiting, or prolonged fast. Patients who are pregnant, breast-feeding, those over age 60, and people taking diuretics ("water pills") should discuss the risks and benefits of lithium treatment with their doctor before beginning therapy. Lithium should be discontinued 24 hours before a major surgery, but may be continued normally for minor surgical procedures.

Side effects

Tremor is the most common neurological side effect. Lithium tremor is an irregular, non-rhythmic twitching of the arms and legs that is variable in both intensity and frequency. Lithium-induced tremor occurs in approximately half of persons taking this medication. The chance of tremor decreases if the dose is reduced. Acute lithium toxicity (poisoning) can result in neurological side effects, ranging from confusion and coordination impairment, to coma, **seizures**, and death. Other neurological side effects associated with lithium therapy include lethargy, memory impairment, difficulty finding words, and loss of creativity.

About 30 to 35% of patients experience excessive thirst and urination, usually due to the inability of the kidneys to retain water and sodium. However, lithium is not known to cause kidney damage.

Lithium inhibits the synthesis of thyroid hormone. About 10 to 20% of patients treated with lithium develop some degree of thyroid insufficiency, but they usually do not require supplementation with thyroid hormone tablets.

Gastrointestinal side effects include loss of appetite, nausea, vomiting, diarrhea, and stomach pain. Weight gain is another common side effect for patients receiving long-term treatment. Changes in saliva flow and enlargement of the salivary glands may occur. An increase in tooth cavities and the need for dental care among patients taking lithium has been reported.

Skin reactions to lithium are common but can usually be managed without discontinuing lithium therapy. Lithium may worsen folliculitis (inflammation of hair follicle), psoriasis, and acne. Thinning of the hair may occur, and, less commonly, hair loss may be experienced. Swollen feet are an uncommon side effect that responds to dose reduction.

Electrocardiographic (EKG) abnormalities may occur with lithium therapy, but significant cardiovascular effects are uncommon except as the result of deliberate or accidental overdose.

A mild-to-moderate increase in the number of white blood cells is a frequent side effect of lithium. Conversely, lithium may slow the formation of red blood cells and cause anemia.

Increased risk of fetal cardiovascular disease may be associated with the use of lithium during pregnancy, especially during the first trimester (first three months). For this reason, once a woman becomes pregnant, lithium should be discontinued until the second or third trimester and the patient receives alternative treatments for her mania.

Interactions

People taking lithium should always be concerned that other medications they are taking may adversely interact with it; patients should consult their physician or pharmacists about these interactions. The following list represents just some of the medications that lithium may interact with to either (a) increase or decrease the effectiveness of the lithium or (b) increase or decrease the effectiveness of the other drug:

- angiotensin-converting enzyme inhibitors such as captopril, lisinopril, or enalapril
- non-steroidal anti-inflammatory drugs such as ibuprofen or naprosyn
- diuretics (water pills) such as hydrochlorothiazide, furosemide, or ethacrynic acid
- asthma drugs such as theophylline and aminophylline
- anticonvulsants such as phenytoin and carbamazepine
- calcium channel blockers such as verapamil or diltiazem
- muscle relaxants such as methocarbamol, carisoprodol, and cyclobenzaprine
- metronidazole, a commonly prescribed antibiotic, used to treat infections
- antidiabetic therapy
- amiodarone, an anti-arrhythmic drug
- antacids containing sodium bicarbonate
- antidepressants

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Jack H. Raber, Pharm.D.

Lithobid *see* **Lithium carbonate**

Lithonate *see* **Lithium carbonate**

Lithotabs *see* **Lithium carbonate**

Lobotomy *see* **Psychosurgery**

Lorazepam

Definition

Lorazepam, a mild tranquilizer in the class of drugs known as benzodiazepines is a sold in the United States under brand names Alzapam, Ativan, or Loraz. It is also available generically.

Purpose

Lorazepam is used for management of anxiety, nausea and vomiting, **insomnia**, and **seizures**. Lorazepam is also used prior to surgery to produce sedation, sleepiness, drowsiness, relief of anxiety, and a decreased ability to recall the events surrounding the surgery.

Description

Lorazepam is a member of the benzodiazepine family. Benzodiazepines primarily work by enhancing the function of a certain naturally occurring **brain** chemical, gamma aminobutyric acid (GABA), that is responsible for inhibiting the transmission of nervous impulses in the brain and spinal cord. At the same time, the enhancement of GABA in the brain decreases symptoms associated with anxiety. Lorazepam differs from drugs such as **diazepam** (Valium) and **chlordiazepoxide** (Librium) in that it is shorter acting and does not accumulate in the body after repeated doses.

Lorazepam is available in 0.5-mg, 1-mg, and 2-mg tablets and in an injectable form.

Recommended dosage

Lorazepam is taken several times daily by mouth or injected to treat anxiety. Dosage ranges from 1–2 mg taken either every 12 or every eight hours. The maximum daily total dosage for anxiety is 10 mg given in two to three divided doses. For sleep, patients may take from 2–4 mg at bedtime. Doses taken before surgery range from 2.5–5 mg.

Between 0.5 mg and 1 mg of lorazepam may be taken every six to eight hours to help control treatment-related nausea and vomiting (nausea and vomiting that occur as a side effect of a drug or medical treatment). Two mg of lorazepam is often given half an hour before chemotherapy to help prevent stomach upset. An additional 2 mg may be taken every four hours as needed.

The usual dose to treat seizures is 4 mg given intravenously (through a vein). This dose may be increased to 8 mg in patients who do not respond to the 4-mg dose.

KEY TERMS

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Precautions

Lorazepam, like other drugs of this type, can cause physical and psychological dependence. Patients should not increase the dose or frequency of this drug on their own, nor should they suddenly stop taking this medication. Instead, when stopping the drug, the dosage should gradually be decreased, then discontinued. If the drug is stopped abruptly, the patient may experience agitation, irritability, difficulty sleeping, convulsions, and other withdrawal symptoms.

Patients allergic to benzodiazepines should not take lorazepam. Those with narrow-angle glaucoma, pre-existing depression of the central nervous system, severe uncontrolled pain, or severe low blood pressure should not take lorazepam. This drug should be used with caution in patients with a history of drug abuse. Children under age 12 should not take lorazepam. Children between the ages of 12 and 18 may take the drug by mouth, but not intravenously. Pregnant women and those trying to become pregnant should not take lorazepam. This drug has been associated with damage to the developing fetus when taken during the first three months of pregnancy. Patients taking this drug should not breast-feed.

Side effects

Drowsiness and sleepiness are common and expected effects of lorazepam. Patients should not drive, operate machinery, or perform hazardous activities that require mental alertness until they have a sense of how lorazepam will affect their alertness. Patients over age 50 may experience deeper and longer sedation after taking lorazepam. These effects may subside with continued use or dosage reduction.

The effects of an injection may impair performance and driving ability for 24–48 hours. The impairment may last longer in older people and those taking other central nervous system depressants, such as some pain medications.

Lorazepam may also make patients feel dizzy, weak, unsteady, or clumsy. Less frequently, people may feel depressed, disoriented, nauseous, or agitated while taking this drug. Other side effects include headache, difficulty sleeping, rash, yellowing eyes, vision changes, and **hallucinations**. Redness and pain may occur at the injection site.

Patients may experience high or low blood pressure and difficulty breathing after an injection of lorazepam. Nausea, vomiting, dry mouth, and constipation may also occur. The patient's sex drive may decrease, but this side effect is reversible once the drug is stopped. Patients should alert their physician to confusion, depression, excitation, nightmares, impaired coordination, changes in personality, changes in urinary pattern, chest pain, heart palpitations, or any other side effects.

Interactions

Alcohol and other central nervous system depressants can increase the drowsiness associated with this drug. Some over-the-counter medications depress the central nervous system. The herbal remedies **kava kava** and **valerian** may increase the effects of lorazepam. Patients should check with a doctor before starting any new medication while taking lorazepam. People should not drink alcoholic beverages when taking lorazepam and for 24–48 hours before receiving an injection prior to surgery.

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Loss *see* **Grief**

Loxapine

Definition

Loxapine is a prescription-only drug used to treat serious mental, nervous, and emotional disorders. Loxapine is sold under the brand name Loxitane in the United States. Loxapine is also be available in generic form.

Purpose

Loxapine is used to treat a variety of mental disorders including anxiety, mania, depression, and psychotic disorders.

Description

Loxapine is in the class of drugs known as antipsychotic agents. The exact mode of action of loxapine has not been precisely determined, but this drug has a tranquilizing effect on patients with anxiety, mania, and other psychotic disorders. It is known that loxapine reduces the amount of dopamine transmitted within the **brain**. Loxapine is available in 5-, 10-, 25-, and 50-mg tablets.

Recommended dosage

Loxapine is available in oral solution, capsules, tablets, and injectable form. The typical starting dose for adults and children over the age of 16 years is 10 mg given two to four times daily. The maximum range after the initial period is between 60 mg and 100 mg given two to four times per day. After a period of time, the dose is usually lowered to 20–60 mg per day given in divided doses. Injections are usually given only during the initial phase and are delivered into muscle (IM) in doses ranging from 12.5 mg to 50 mg every four to six hours until a desired level of response is reached. Then, the patient is usually put on the oral (PO) form for maintenance therapy. Guidelines for use in persons under the age of 16 years have not been established.

Precautions

Persons taking loxapine should not stop taking this medication suddenly. The dosage should be gradually decreased over time. Loxapine should not be combined with other agents that depress the central nervous system, such as antihistamines, alcohol, tranquilizers, sleeping medications, and seizure medications. Loxapine can cause the skin to become more sensitive to the sun. Sunscreen with a skin protection factor (SPF) greater than 15 should be used when taking this drug.

Loxapine is typically not used in persons who are in severe drug-induced states or in a coma. People with a history of **seizures**, heart disease, prostate enlargement, glaucoma, or chronic obstructive pulmonary disorder should receive loxapine only after careful evaluation. Guidelines for use in children under the age of 16 years have not been established. Loxapine has not been thoroughly studied in pregnant and nursing women, but great caution should be exercised when women in these groups use loxapine.

Side effects

Rare side effects, but ones that need to be reported immediately to a doctor, include seizures, breathing difficulties, irregular heartbeat, significant changes in blood pressure, increased sweating, severe stiffness, extreme weakness, and unusually pale skin. These symptoms are considered an emergency, and the patient should stop using the medication immediately. More common but less serious side effects include uncontrolled movement of the arms or legs, lip smacking, unusual movements of the tongue, puffing of the cheeks, and uncontrolled chewing movements. These symptoms should also be reported immediately to a doctor.

More common and even less serious side effects include difficulty in speaking or swallowing, restlessness, stiffness of arms and legs, trembling, and loss of balance. These symptoms also need to be reported to a doctor. Less common and not especially significant side effects include urination problems, muscle spasms, skin rash, and severe constipation. Rare and not particularly serious side effects include uncontrolled twisting and movement

KEY TERMS

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Chronic obstructive pulmonary disease—Disorder characterized by the decreasing ability of the lungs to ventilate adequately.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Psychotic—Mental disorder characterized by disturbances of personality and a loss of normal association with reality.

Respiratory depression—Significant impairment of the respiratory system.

of the neck, fever, sore throat, unusual bleeding, yellowing of the eyes or skin, and changes in facial expression.

Overdose symptoms include significant drowsiness, severe dizziness, significant breathing difficulties, severe weakness, trembling muscles, and severe uncontrolled movements.

Interactions

Loxapine should not be combined with anticholinergic drugs because of the potential of decreased antipsychotic effects. Loxapine should not be combined with bromocriptine because the combination can decrease the effectiveness of bromocriptine in patients with pituitary tumors. The combination of loxapine with lithium increases the toxicity of both drugs significantly. Likewise, loxapine and **lorazepam** should not be combined because the combination of the two has produced very low blood pressure, severe drowsiness, and respiratory depression in rare cases.

See also Anxiety and anxiety disorders; Depression and depressive disorders

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Loxitane *see* **Loxapine**

LSD *see* **Hallucinogens and related disorders**

Ludiomil *see* **Maprotiline**

Luria-Nebraska Neuropsychological Battery

Definition

The Luria-Nebraska Neuropsychological Battery, also known as LNNB or Luria-Nebraska Battery, is a standardized test battery used in the screening and evaluation of neuropsychologically impaired individuals.

Purpose

The LNNB was developed in an attempt to combine the qualitative techniques of some neuropsychological tests with the quantitative techniques of others. However, the scoring system that most clinicians use is primarily quantitative. The battery measures specific neuropsychological functioning in several areas including motor skills, language abilities, intellectual abilities, nonverbal auditory skills, and visual-spatial skills.

The battery is used by clinicians as a screening tool to determine whether a significant **brain** injury is present or to learn more about known brain injuries. It is also used to determine what the patient is or is not able to do with regard to neuropsychological functioning. For example, the LNNB may be used to determine which intellectual or cognitive tasks a patient may or may not be able to complete. The battery can also be used to arrive at underlying causes of a patient's behavior. More specifically, information regarding the location and nature of the brain injury or dysfunction causing a patient's problems is collected.

The LNNB is also used to help distinguish between brain damage and functional mental disorders such as **schizophrenia**. Also, within the category of schizophrenia, the battery can be used to help distinguish between patients with normal neuropsychological functioning and those with clear deficits. Besides its specifically clinical use, the battery is sometimes used for legal purposes—the presence or severity of a brain injury may be measured as part of an evaluation used in the court system.

Precautions

Because of the length of the test and complexity in interpretation, the examiner must be competent and properly trained. Also, the fact that many patients are, indeed, brain damaged can make test administration difficult or frustrating.

Description

The LNNB is based on the work of A. R. Luria, a Russian neuropsychologist who performed pioneering theoretical and clinical work with regard to brain function. Luria believed in a primarily qualitative approach to assessment and was opposed to standardization. He did not believe that neuropsychological functioning could be measured quantitatively. Thus, although his name is part of the test itself, his contribution to the LNNB is entirely

theoretical. Also, the LNNB is based, in part, on Luria's Neuropsychological Investigation, a measure developed by Christensen in 1975. This test included items asked by Luria in his clinical interviews, some of which are used in the LNNB.

The battery, written in 1981 by Charles Golden, is appropriate for people aged 13 and older and takes between 90 and 150 minutes to complete. It consists of 269 items in the following 11 clinical scales:

- reading
- writing
- arithmetic
- visual
- memory
- expressive language
- receptive language
- motor function
- rhythm
- tactile
- intellectual

Scores for three summary scales can also be calculated: pathognomonic, right hemisphere, and left hemisphere. A children's version of the battery, called the Luria-Nebraska Neuropsychological Battery for Children (LNNB-C), appropriate for children aged eight to 12, is also available.

Results

The probability of brain damage is assessed by comparing an individual's score on each of the battery's 11 clinical scales to a critical level appropriate for that person's age and education level. For example, if a person has five to seven scores above the critical level, they most likely have some sign of neurological impairment. Eight or more scores above the critical level indicate a clear history of neurological disorder.

The battery has been criticized by researchers on the grounds that it overestimates the degree of neuropsychological impairment. In other cases, it has been found to fail to detect neuropsychological problems. Also, the intellectual processes scale has not been found to correspond well to other measures of intelligence, such as the **Wechsler Adult Intelligence Scale** (WAIS).

Other research, however, has found it to be a useful measure. It has been found as effective as the **Halstead-Reitan Battery** in distinguishing between brain-damaged individuals and nonbrain-damaged individuals with psychiatric problems. Part of the inconsistencies in opinion regarding the LNNB may be due to the specific nature of the population being tested by the battery and the difficulties in administration and scoring that some clinicians experience.

KEY TERMS

Pathognomonic—Describing symptoms characteristic of a particular disease.

Reliability—The ability of a test to yield consistent, repeatable results.

Standardization—The administration of a test to a sample group of people for the purpose of establishing test norms. The Luria-Nebraska Neuropsychological Battery was standardized using a sample of neurologically impaired individuals.

Validity—The ability of a test to measure accurately what it claims to measure.

See also Intelligence tests; Kaufman Assessment Battery for Children; Kaufman Short Neuropsychological Assessment; Neuropsychological testing

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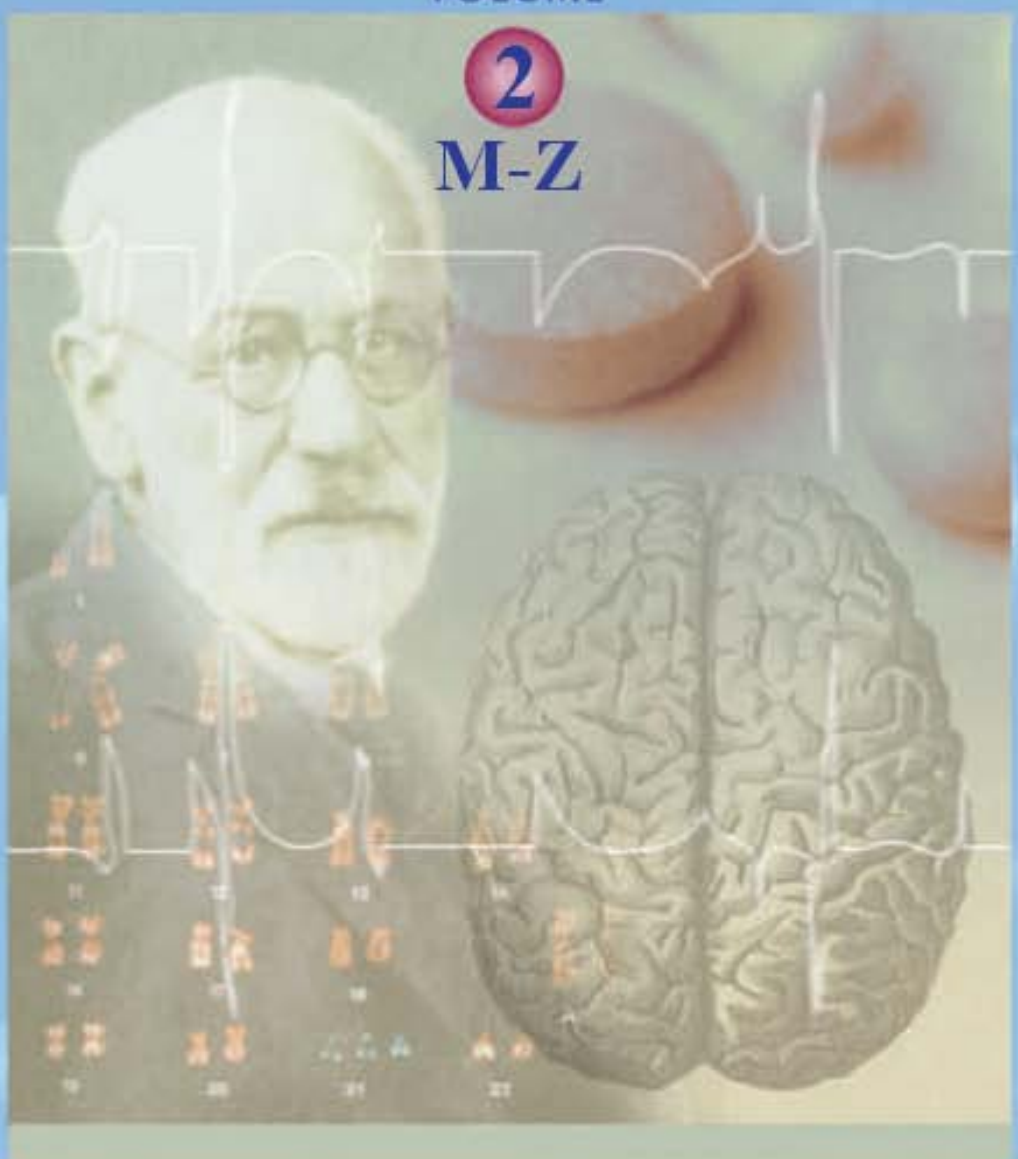
Luvox *see* **Fluvoxamine**

The GALE ENCYCLOPEDIA of MENTAL DISORDERS

VOLUME

2

M-Z



The GALE ENCYCLOPEDIA *of* MENTAL DISORDERS

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VOLUME

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M-Z

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PLEASE READ—IMPORTANT INFORMATION

The *Gale Encyclopedia of Mental Disorders* is a medical reference product designed to inform and educate readers about a wide variety of mental disorders, diagnostic techniques and tests, therapies, and psychiatric medications. The Gale Group believes the product to be comprehensive, but not necessarily definitive. It is intended to supplement, not replace, consultation with a physician or other health care practitioner. While the Gale Group has made substantial efforts to provide information that is accurate, comprehensive, and up-to-date, the Gale Group makes no representations or warranties of any kind, including without limitation, warranties of merchantability or fitness for a particular purpose, nor does it guarantee the accuracy, comprehensiveness, or timeliness of the information contained in this product. Readers should be aware that the universe of medical knowledge is constantly growing and changing, and that differences of medical opinion exist among authorities. Readers are also advised to seek professional diagnosis and treatment of any medical condition, and to discuss information obtained from this book with their health care provider.

INTRODUCTION

The *Gale Encyclopedia of Mental Disorders* is a valuable source of information for anyone who wants to learn more about mental disorders and their treatments. This collection of approximately 400 entries provides in-depth coverage of specific disorders recognized by the American Psychiatric Association (as well as some disorders not formally recognized as distinct disorders), diagnostic procedures and techniques, therapies, and psychiatric medications. In addition, entries have been included to facilitate understanding of related topics, such as Advance directives, Crisis housing, and Neurotransmitters.

This encyclopedia minimizes medical jargon and uses language that laypersons can understand, while still providing thorough coverage that will benefit health science students as well.

Entries follow a standardized format that provides information at a glance. Rubrics include:

Disorders	**Medications**
Definition	Definition
Description	Purpose
Causes and symptoms	Description
Demographics	Recommended dosage
Diagnosis	Precautions
Treatments	Side effects
Prognosis	Interactions
Prevention	Resources
Resources	

INCLUSION CRITERIA

A preliminary list of mental disorders and related topics was compiled from a wide variety of sources, including professional medical guides and textbooks, as well as consumer guides and encyclopedias. The advisory board, made up of professionals from a variety of health care fields including psychology, psychiatry, pharmacy, and social work, evaluated the topics and made suggestions for inclusion. Final selection of topics to include was made by the advisory board in conjunction with the Gale editor.

ABOUT THE CONTRIBUTORS

The essays were compiled by experienced medical writers, including physicians, pharmacists, and psychologists. The advisors reviewed the completed essays to ensure that they are appropriate, up-to-date, and accurate.

HOW TO USE THIS BOOK

The *Gale Encyclopedia of Mental Disorders* has been designed with ready reference in mind.

- Straight **alphabetical arrangement** of topics allows users to locate information quickly.
- **Bold-faced terms** within entries direct the reader to related articles.
- **Cross-references** placed throughout the encyclopedia direct readers from alternate names, drug brand names, and related topics to entries.
- A list of **key terms** is provided where appropriate to define unfamiliar terms or concepts. A **glossary** of key terms is also included at the back of Volume II.
- The **Resources** sections direct readers to additional sources of information on a topic.
- Valuable **contact information** for organizations and support groups is included with many of the disorder entries.
- A leaf graphic () inserted next to the entry title denotes entries about herbals (such as Ginkgo biloba) or dietary supplements (such as SAMe). These entries have the same rubrics as the medication entries; however, the graphic is to draw attention to the fact that these entries are not about prescription medications.
- A **Symptoms list** at the back of Volume II has been included *not* for diagnosis but to reveal patterns in

symptoms and disorders and to provide a starting point for research or discussion with a health care provider.

- A comprehensive **general index** guides readers to all topics mentioned in the text.

GRAPHICS

The *Gale Encyclopedia of Mental Disorders* contains 100 illustrations, photos, and tables. A color insert in each volume has been included to enhance certain photos shown in the text in black and white.

ADVISORY BOARD

A number of experts in medicine, psychology, psychiatry, and pharmacy provided invaluable assistance in the formulation of this encyclopedia. The members of the advisory board performed a myriad of duties, from defining the scope of coverage to reviewing individual entries for accuracy and accessibility. The editor would like to express appreciation to them for their time and for their contributions.

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M

Magnetic resonance imaging

Definition

Magnetic resonance imaging (MRI) is one of the newest diagnostic medical imaging technologies that uses strong magnets and pulses of radio waves to manipulate the natural magnetic properties in the body to generate a visible image. In the field of mental health, an MRI scan may be used when a patient seeks medical help for symptoms that could possibly be caused by a **brain** tumor. These symptoms may include headaches, emotional abnormalities, or intellectual or memory problems. In these cases, an MRI scan may be performed to "rule out" a tumor, so that other tests can be performed in order to establish an accurate **diagnosis**.

Purpose

MRI was developed in the 1980s. Its technology has been developed for use in magnetic resonance angiography (MRA), magnetic resonance spectroscopy (MRS), and, more recently, magnetic resonance cholangiopancreatography (MRCP). MRA was developed to study blood flow, whereas MRS can identify the chemical composition of diseased tissue and produce color images of brain function. MRCP is evolving into a non-invasive potential alternative for the diagnostic procedure endoscopic retrograde cholangiopancreatography (ERCP).

Advantages

DETAIL. MRI creates precise images of the body based on the varying proportions of magnetic elements in different tissues. Very minor fluctuations in chemical composition can be determined. MRI images have greater natural contrast than standard x rays, **computed tomography** scan (CT scan), or ultrasound, all of which depend on the differing physical properties of tissues. This sensitivity allows MRI to distinguish fine variations in tissues deep within the body. It is also particularly useful for spotting and distinguishing diseased tissues (tumors and other lesions) early in their development. Often, doctors prescribe an MRI scan to investigate more fully earlier findings of other imaging techniques.

SCOPE. The entire body can be scanned, from head to toe and from the skin to the deepest recesses of the brain. Moreover, MRI scans are not obstructed by bone, gas, or body waste, which can hinder other imaging techniques. (Although the scans can be degraded by motion such as breathing, heartbeat, and bowel activity.) The MRI process produces cross-sectional images of the body that are as sharp in the middle as on the edges, even of the brain through the skull. A close series of these two-dimensional images can provide a three-dimensional view of the targeted area. Along with images from the cross-sectional plane, the MRI can also provide images sagitally (from one side of the body to the other, from left to right for example), allowing for a better three-dimensional interpretation, which is sometimes very important for planning a surgical approach.

SAFETY. MRI does not depend on potentially harmful ionizing radiation, as do standard x ray and computed tomography scans. There are no known risks specific to the procedure, other than for people who might have metal objects in their bodies.

Despite its many advantages, MRI is not routinely used because it is a somewhat complex and costly procedure. MRI requires large, expensive, and complicated equipment, a highly trained operator, and a doctor specializing in radiology. Generally, MRI is prescribed only when serious symptoms or negative results from other tests indicate a need. Many times another test is appropriate for the type of diagnosis needed.

Uses

Doctors may prescribe an MRI scan of different areas of the body.

BRAIN AND HEAD. MRI technology was developed because of the need for brain imaging. It is one of the few

KEY TERMS

Angiography—A procedure in which a contrast medium is injected into the bloodstream (through an artery in the neck) and its progress through the brain is tracked. This illustrates where a blockage or hemorrhage has occurred.

Gadolinium—A very rare metallic element useful for its sensitivity to electromagnetic resonance, among other things. Traces of it can be injected into the body to enhance the MRI pictures.

Hydrogen—The simplest, most common element known in the universe. It is composed of a single electron (negatively charged particle). It is the nuclear proton of hydrogen that makes MRI possible by reacting resonantly to radio waves while aligned in a magnetic field.

Ionizing radiation—Electromagnetic radiation that can damage living tissue by disrupting and destroying individual cells. All types of nuclear decay radiation (including x rays) are potentially ionizing. Radio waves do not damage organic tissues they pass through.

Magnetic field—The three-dimensional area surrounding a magnet, in which its force is active. During MRI, the patient's body is permeated by the force field of a superconducting magnet.

Radio waves—Electromagnetic energy of the frequency range corresponding to that used in radio communications, usually 10,000 cycles per second to 300 billion cycles per second. Radio waves are the same as visible light, x rays, and all other types of electromagnetic radiation, but are of a higher frequency.

imaging tools that can see through bone (the skull) and deliver high-quality pictures of the brain's delicate soft tissue structures. MRI may be needed for patients with symptoms of a brain tumor, **stroke**, or infection (like meningitis). MRI may also be needed when cognitive or psychological symptoms suggest brain disease (like Alzheimer's or Huntington's diseases, or multiple sclerosis), or when developmental retardation suggests a birth defect. MRI can also provide pictures of the sinuses and other areas of the head beneath the face. In adult and pediatric patients, MRI may be better able to detect abnormalities than compared to computed tomography scanning.

SPINE. Spinal problems can create a host of seemingly unrelated symptoms. MRI is particularly useful for identifying and evaluating degenerated or herniated spinal discs. It can also be used to determine the condition of nerve tissue within the spinal cord.

JOINT. MRI scanning is most commonly used to diagnose and assess joint problems. MRI can provide clear images of the bone, cartilage, ligament, and tendon that comprise a joint. MRI can be used to diagnose joint injuries due to sports, advancing age, or arthritis. MRI can also be used to diagnose shoulder problems, such as a torn rotator cuff. MRI can also detect the presence of an otherwise hidden tumor or infection in a joint, and can be used to diagnose the nature of developmental joint abnormalities in children.

SKELETON. The properties of MRI that allow it to see through the skull also allow it to view the inside of bones. Accordingly, it can be used to detect bone cancer, inspect the marrow for leukemia and other diseases, assess bone loss (osteoporosis), and examine complex fractures.

HEART AND CIRCULATION. MRI technology can be used to evaluate the circulatory system. The heart and blood flow provides a good natural contrast medium that allows structures of the heart to be clearly distinguished.

THE REST OF THE BODY. Whereas computed tomography and ultrasound scans satisfy most chest, abdominal, and general body imaging needs, MRI may be needed in certain circumstances to provide better pictures or when repeated scanning is required. The progress of some therapies, like liver cancer therapy, needs to be monitored, and the effect of repeated x-ray exposure is a concern.

Precautions

MRI scans and metal

MRI scanning should not be used when there is the potential for an interaction between the strong MRI magnet and metal objects that might be embedded in a patient's body. The force of magnetic attraction on certain types of metal objects (including surgical steel) could move them within the body and cause serious injury. Metal may be embedded in a person's body for several reasons.

MEDICAL. People with implanted cardiac pacemakers, metal aneurysm clips, or who have broken bones repaired with metal pins, screws, rods, or plates must tell their radiologist prior to having an MRI scan. In some cases (like a metal rod in a reconstructed leg), the difficulty may be overcome.

INJURY. Patients must tell their doctor if they have bullet fragments or other metal pieces in their body from old wounds. The suspected presence of metal, whether

from an old or recent wound, should be confirmed before scanning.

OCCUPATIONAL. People with significant work exposure to metal particles (working with a metal grinder, for example) should discuss this with their doctor and radiologist. The patient may need prescan testing—usually a single, regular x ray of the eyes to see if any metal is present.

Chemical agents

Chemical agents designed to improve the picture or allow for the imaging of blood or other fluid flow during MRA may be injected. In rare cases, patients may be allergic to, or intolerant of, these agents, and these patients should not receive them. If these chemical agents are to be used, patients should discuss any concerns they have with their doctor and radiologist.

General

The potential side effects of magnetic and electric fields on human health remain a source of debate. In particular, the possible effects on an unborn baby are not well known. Any woman who is, or may be, pregnant, should carefully discuss this issue with her doctor and radiologist before undergoing a scan.

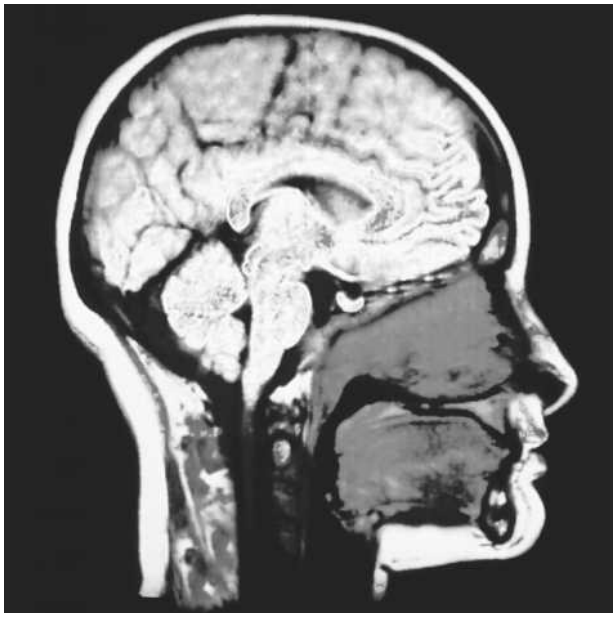
As with all medical imaging techniques, **obesity** greatly interferes with the quality of MRI.

Description

In essence, MRI produces a map of hydrogen distribution in the body. Hydrogen is the simplest element known, the most abundant in biological tissue, and one that can be magnetized. It will align itself within a strong magnetic field, like the needle of a compass. The earth's magnetic field is not strong enough to keep a person's hydrogen atoms pointing in the same direction, but the superconducting magnet of an MRI machine can. This comprises the magnetic part of MRI.

Once a patient's hydrogen atoms have been aligned in the magnet, pulses of very specific radio wave frequencies are used to knock them back out of alignment. The hydrogen atoms alternately absorb and emit radio wave energy, vibrating back and forth between their resting (magnetized) state and their agitated (radio pulse) state. This comprises the resonance part of MRI.

The MRI equipment records the duration, strength, and source location of the signals emitted by the atoms as they relax and translates the data into an image on a television monitor. The state of hydrogen in diseased tissue differs from healthy tissue of the same type, making MRI particularly good at identifying tumors and other lesions.



MRI scan of human brain. *(Scott Camazine and Sue Trainor. Photo Researchers, Inc. Reproduced by permission.) See color insert for color version of photo.*

In some cases, chemical agents such as gadolinium can be injected to improve the contrast between healthy and diseased tissue.

A single MRI exposure produces a two-dimensional image of a slice through the entire target area. A series of these image slices closely spaced (usually less than half an inch) makes a virtual three-dimensional view of the area.

Regardless of the exact type of MRI planned, or area of the body targeted, the procedure involved is basically the same. In a special MRI suite, the patient lies down on a narrow table and is made as comfortable as possible. Transmitters are positioned on the body and the table moves into a long tube that houses the magnet. The tube is as long as an average adult lying down, and is open at both ends. Once the area to be examined has been properly positioned, a radio pulse is applied. Then a two-dimensional image corresponding to one slice through the area is made. The table then moves a fraction of an inch and the next image is made. Each image exposure takes several seconds and the entire exam will last anywhere from 30 to 90 minutes. During this time, the patient must remain still as movement can distort the pictures produced.

Depending on the area to be imaged, the radio-wave transmitters will be positioned in different locations.

- For the head and neck, a helmet-like covering is worn on the head.

- For the spine, chest, and abdomen, the patient will be lying on the transmitters.
- For the knee, shoulder, or other joint, the transmitters will be applied directly to the joint.

Additional probes will monitor vital signs (like pulse, respiration, etc.) throughout the test.

The procedure is somewhat noisy and can feel confining to many patients. As the patient moves through the tube, the patient hears a thumping sound. Sometimes, music is supplied via earphones to drown out the noise. Some patients may become anxious or feel claustrophobic while in the small, enclosed tube. Patients may be reassured to know that throughout the study, they can communicate with medical personnel through an intercom-like system.

Recently, open MRIs have become available. Instead of a tube open only at the ends, an open MRI also has opening at the sides. Open MRIs are preferable for patients who have a fear of closed spaces and become anxious in traditional MRI machines. Open MRIs can also better accommodate obese patients, and allow parents to accompany their children during testing.

If the chest or abdomen is to be imaged, the patient will be asked to hold his to her breath as each exposure is made. Other instructions may be given to the patient as needed. In many cases, the entire examination will be performed by an MRI operator who is not a doctor. However, the supervising radiologist should be available to consult as necessary during the exam, and will view and interpret the results sometime later.

Magnetic resonance spectroscopy (MRS) is different from MRI because MRS uses a continuous band of radio wave frequencies to excite hydrogen atoms in a variety of chemical compounds other than water. These compounds absorb and emit radio energy at characteristic frequencies, or spectra, which can be used to identify them. Generally, a color image is created by assigning a color to each distinctive spectral emission. This comprises the spectroscopy part of MRS. MRS is still experimental and is available only in a few research centers.

Doctors primarily use MRS to study the brain and disorders like epilepsy, **Alzheimer's disease**, brain tumors, and the effects of drugs on brain growth and metabolism. The technique is also useful in evaluating metabolic disorders of the muscles and nervous system.

Magnetic resonance angiography (MRA) is another variation on standard MRI. MRA, like other types of angiography, looks specifically at fluid flow within the blood (vascular) system, but does so without the injection of dyes or radioactive tracers. Standard MRI cannot make a good picture of flowing blood, but MRA uses specific radio pulse sequences to capture usable signals. The technique is generally used in combination with MRI to obtain images that show both vascular structure and flow within the brain and head in cases of stroke, or when a blood clot or aneurysm is suspected.

MRI technology is also being applied in the evaluation of the pancreatic and biliary ducts in a new study called magnetic resonance cholangiopancreatography (MRCP). MRCP produces images similar to that of endoscopic retrograde cholangiopancreatography (ERCP), but in a non-invasive manner. Because MRCP is new and still very expensive, it is not readily available in most hospitals and imaging centers.

Preparation

In some cases (such as for MRI brain scanning or MRA), a chemical designed to increase image contrast may be given immediately before the exam. If a patient suffers from anxiety or claustrophobia, drugs may be given to help the patient relax.

The patient must remove all metal objects (watches, jewelry, eye glasses, hair clips, etc.). Any magnetized objects (like credit and bank machine cards, audio tapes, etc.) should be kept far away from the MRI equipment because they can be erased. The patient cannot bring any personal items such as a wallet or keys into the MRI machine. The patient may be asked to wear clothing without metal snaps, buckles, or zippers, unless a medical gown is worn during the procedure. The patient may be asked not to use hair spray, hair gel, or cosmetics that could interfere with the scan.

Aftercare

No aftercare is necessary, unless the patient received medication or had a reaction to a contrast agent. Normally, patients can immediately return to their daily activities. If the exam reveals a serious condition that requires more testing or treatment, appropriate information and counseling will be needed.

Risks

MRI poses no known health risks to the patient and produces no physical side effects. Again, the potential effects of MRI on an unborn baby are not well known. Any woman who is, or may be, pregnant, should carefully discuss this issue with her doctor and radiologist before undergoing a scan.

Normal results

A normal MRI, MRA, MRS, or MRCP result is one that shows the patient's physical condition to fall within normal ranges for the target area scanned.

Abnormal results

Generally, MRI is prescribed only when serious symptoms or negative results from other tests indicate a need. There often exists strong evidence of a condition that the scan is designed to detect and assess. Thus, the results will often be abnormal, confirming the earlier diagnosis. At that point, further testing and appropriate medical treatment is needed. For example, if the MRI indicates the presence of a brain tumor, an MRS may be prescribed to determine the type of tumor so that aggressive treatment can begin immediately without the need for a surgical biospy.

Resources

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Goldman, L., and Claude Bennett, eds. *Cecil Textbook of Medicine.* 21st Edition. Philadelphia: W. B. Saunders, 2000: pp 977–970.

Kevles, Bettyann Holtzmann. *Naked to the Bone: Medical Imaging in the Twentieth Century.* New Brunswick, NJ: Rutgers University Press, 1997.

Roth, Carolyn K. *Tech's Guide to MRI: Imaging Procedures, Patient Care and Safety.* Malden: Blackwell Science, 2001.

Zaret, Barry L., and others, eds. *The Patient's Guide to Medical Tests.* Boston: Houghton Mifflin Company, 1997.

PERIODICALS

Carr-Locke, D., and others, "Technology Status Evaluation: Magnetic Resonance Cholangiopancreatography." *Gastrointestinal Endoscopy* (June 1999): 858–61.

ORGANIZATIONS

American College of Radiology. 1891 Preston White Drive, Reston, VA 22091. (800) ACR-LINE. <http://www.acr.org>.

American Society of Radiologic Technologists. 15000 Central Avenue SE, Albuquerque, NM 87123–3917. (505) 298–4500. <http://www.asrt.org>.

Kurt Richard Sternlof
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Magnetocephalogram *see* **Electroencephalography**

Major depressive disorder

Definition

Major depressive disorder (MDD) is a condition characterized by a long-lasting depressed mood or marked loss of interest or pleasure (anhedonia) in all or nearly all activities. Children and adolescents with MDD may be irritable instead of sad. These symptoms, along with others described below, must be sufficiently severe to interfere significantly with the patient's daily functioning in order for a person to be diagnosed with MDD.

Description

Major depressive disorder is a serious mental disorder that profoundly affects an individual's quality of life. Unlike normal bereavement or an occasional episode of "the blues," MDD causes a lengthy period of gloom and hopelessness, and may rob the sufferer of the ability to take pleasure in activities or relationships that were previously enjoyable. In some cases, depressive episodes seem to be triggered by an obviously painful event, but MDD may also develop without a specific stressor. Research indicates that an initial episode of depression is likely to be a response to a specific stimulus, but later episodes are progressively more likely to start without a triggering event. A person suffering major depression finds job-related responsibilities and such other tasks as parenting burdensome and carried out only with great effort. Mental efficiency and memory are affected, causing even simple tasks to be tiring and irritating. Sexual interest dwindles; many people with MDD become withdrawn and avoid any type of social activity. Even the ability to enjoy a good meal or a sound night's sleep is frequently lost; many depressed people report a chronic sense of malaise (general discomfort or unease). For some, the pain and suffering accompanying MDD becomes so unendurable that **suicide** is viewed as the only option; MDD has the highest mortality rate of any mental disorder.

Major depressive disorder may be limited to a single episode of depression; more commonly, it may become a chronic condition with many episodes of depressed mood. Other symptoms that may develop include psychotic symptoms (bizarre thoughts, including delusional beliefs and **hallucinations**); **catatonia**; postpartum

KEY TERMS

Agitation—Excessive restlessness or emotional disturbance that is often associated with anxiety or psychosis. May be a symptom of major depressive disorder.

Anhedonia—Loss of the capacity to experience pleasure. Anhedonia is one of the so-called negative symptoms of schizophrenia, and is also a symptom of major depression.

Catatonia—Disturbance of motor behavior with either extreme stupor or random, purposeless activity.

Cortisol—A steroid hormone released by the cortex (outer portion) of the adrenal gland when a person is under stress.

Delusion—A false belief that is resistant to reason or contrary to actual fact. Women suffering from postpartum depression sometimes have delusions about their new baby.

Dysthymia—Depression of low intensity.

Dysthymic disorder—A mood disorder that is less severe than depression but usually more chronic.

Etiology—The cause or origin of a disease or disorder. The word is also used to refer to the study of the causes of disease.

Gingko—A shade tree native to China with fan-shaped leaves and fleshy seeds with edible kernels. Gingko extract has been approved in Europe as a complementary or adjunctive treatment for major depressive episodes.

Intrapsychic—Occurring inside a person's mind or psyche.

Labile—Subject to frequent change, particularly in reference to mood.

Limbic system—A group of structures in the brain that includes the amygdala, hippocampus, olfactory bulbs, and hypothalamus. The limbic system is associated with homeostasis and the regulation and arousal of emotions.

Malaise—The medical term for a general condition of unease, discomfort, or weakness.

Melancholia—A form of severe depression characterized by weight loss, insomnia, and an inability to experience pleasure.

Rumination—A tendency to dwell on certain thoughts, particularly negative ones, repeatedly or obsessively.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

onset (sometimes accompanied by psychotic symptoms); and **seasonal affective disorder**, or SAD.

Such conditions as **postpartum depression** and seasonal affective disorder accompany MDD only under certain circumstances. Postpartum depression begins within four weeks of giving birth. Women with this disorder experience labile mood (frequent drastic mood changes). They may feel helpless and unable to care adequately for their infant, or they may be completely uninterested in the child. The symptoms of postpartum depression are much more severe than those of the relatively common "new baby blues," which affect up to 70% of new mothers. The presence of psychotic symptoms in the mother, too many ruminations (obsessive thoughts), or **delusions** about the infant are associated with a heightened risk of serious harm to the child. The symptoms of postpartum depression are usually attributed to fluctuations in the woman's hormone levels and the emotional impact of bearing a child. The condition is especially likely to occur in women who were highly anxious during pregnancy or had a previous history of mood disorder. Seasonal affective disorder (SAD) is also more common in women than in men; in this case, symptoms of MDD typically begin in fall and winter, especially in northern latitudes in the United States and Canada. Exposure to natural light is limited during the winter in these areas, but the symptoms of SAD typically improve during the spring and summer.

Causes and symptoms

Causes

Because MDD is a relatively common mental disorder, researchers have performed a range of different studies to identify possible underlying causes. Three types of causes are commonly identified: intrapsychic, environmental, and biological.

INTRAPSYCHIC. Since Sigmund Freud attributed the development of mental disorders to intrapsychic (occurring inside the mind) conflicts occurring during early childhood, a sizeable number of theorists have suggested that MDD results from a tendency to internalize negative events. Cognitive behavioral treatment models assume that a person's interpretation of situations is responsible for the development of depression rather than the events themselves. Some people blame themselves for negative experiences while attributing positive outcomes to external sources; they may tend to feel guilty, undeserving, and eventually depressed. For example, they may think of their present job as something they obtained by a chance stroke of good luck; at the same time, they may regard being laid off as something they brought on themselves. When these patterns of thought become habitual, they lead to a style of coping characterized by a view of oneself as worthless, ineffectual, and inferior. In some cases, people pick up these patterns of thinking from their parents or other family members.

Another theory regarding intrapsychic causes attributes depression to so-called "learned helplessness." This theory grew out of research studies on animal learning, comparing dogs that were able to escape from mild electric shocks to dogs that could not escape. The researchers discovered that the dogs who could not escape the mild shocks became passive; later, when they were put in a situation in which they could escape the shocks, they made no attempt to do so but simply lay on their stomachs and whimpered. The animals had, in short, learned to be helpless; they had learned during the first part of the experiment that nothing they had done had any effect on the shocks. Applied to human beings, this theory holds that people tend to become depressed when they have had long-term experiences of helplessness— as would be the case for abused children. Later, when the children have become adults, they do not see themselves as grownups with some control over their lives; they continue to react to setbacks or losses with the same feelings of helplessness that they had as children, and they become depressed.

ENVIRONMENTAL. Environmental theories of the etiology (causation) of MDD emphasize the role of external events in triggering depression. According to this perspective, people become depressed primarily due to unfortunate circumstances that are difficult to change. In some cases, these misfortunes may include environmental disasters or personal losses; but such other factors as low socioeconomic status, oppression associated with one's sex or race, or unpleasant or frustrating relationships are also thought to contribute to depression.

BIOLOGICAL. Ancient medicine alleged that one's state of mind was related to the presence of specific "humors," or fluids, in the body, and various theories have emerged since the eighteenth century regarding possible constitutional factors in humans that affect mood. In recent years, researchers have found numerous abnormalities in the neuroendocrine systems, **neurotransmitters**, and neuroanatomy of the brains of both children and adults with MDD, as well as strong evidence for genetic factors in MDD.

Levels of cortisol, a hormone associated with the human "fight-or-flight" response, have long been studied as possible biological markers for depression. In many adults, cortisol levels rise when the person is acutely depressed and return to normal when the depression passes. Research findings have been inconsistent regarding cortisol levels in children and adolescents, although there is some evidence that higher levels of cortisol secretion are associated with more severe depressive symptoms and with a higher likelihood of recurrence. As of 2002, however, cortisol levels are not considered to be reliable enough to be useful in diagnosing MDD.

Another biological factor that has been studied in humans are changes in the levels of neurotransmitters, which are chemicals that conduct nerve impulses across the tiny gaps between nerve cells. Variations in the levels of certain neurotransmitters have been researched for many years due to their importance in the brain's limbic system, which is the center of emotions and has many important pathways to other parts of the **brain**. In depression, the system that regulates a neurotransmitter called serotonin does not function properly. A group of medications known as serotonin specific reuptake inhibitors, or SSRIs, are assumed to be effective in relieving depression because they prevent serotonin from being taken back up too quickly by receptors in the brain.

Differences in the anatomical structure of the brains of children and adults with MDD have suggested several possible explanations for its development. In particular, the prefrontal cortex has been thought to play a role, on the basis of findings in stroke patients with damage to the prefrontal area of the brain, and in children and adults with MDD. Researchers found that stroke patients experienced more severe depression if their stroke occurred closer to the frontal lobe of the brain; similarly, people with MDD have been found to have decreased frontal lobe volume. Studies of depressed children and adults included subjects who were currently depressed as well as those with a history of depression who were in remission, which suggests that abnormalities in the frontal lobe may be a structural marker of depression. Other neurological studies have reported lower levels of electrical activity in the left frontal cortex among depressed subjects (including the infants of depressed mothers) compared to persons who are not depressed.

Researchers have also been interested in the relationship of genetic factors to depression. It has been known for many years that depression tends to run in families. Convincing evidence of the heritability of depression has been obtained by comparing identical twins (who have identical genetic inheritances) with fraternal twins; these studies have consistently found a higher likelihood of depression between identical than between fraternal twins. Other data indicate that people with a higher genetic risk of depression are more likely to become depressed following a stressful event than people with fewer genetic risk factors.

Symptoms

The core symptom of major depression is a sad mood that does not go away. While most people have occasional days when they feel out of sorts, persons with MDD experience low feelings that build gradually over a period of days or weeks. They are usually not able to "snap out of it" even when something positive happens. In some cases, the symptoms are preceded by an obvious loss or painful event, such as divorce or a death in the family, but the disorder may also appear to begin "out of the blue." People with MDD often appear sad, irritable, and easily moved to tears. They may sleep poorly and complain of vague physical aches and pains; experience sexual difficulties or loss of interest in sex; drop out of social activities; and come across to others as unhappy or lacking in energy. Some people with MDD may deny that they feel depressed, but they lose their enthusiasm for hobbies or work they once found enjoyable and rewarding. Children and adolescents present with many of these same characteristics, but they may often appear easily frustrated and cranky instead of sad. The symptoms of MDD can be summarized as follows:

- Disturbed mood (sad, hopeless, discouraged, "down in the dumps") during most of the day.
- Loss of interest or pleasure in activities.
- Change in appetite nearly every day, leading either to weight gain or to loss of 5% of body weight. In children, this symptom may appear as a failure to make normal weight gains related to growth.
- **Insomnia** (waking in the middle of the night and having difficulty returning to sleep, or waking too early in the morning) or **hypersomnia** (sleeping much more than normal).
- Psychomotor retardation (slowed thinking, speech, body movements) or agitation (inability to sit still, hand-wringing, pulling at clothing, skin, or other objects) that is apparent to others.
- Sense of worthlessness or unreasonable guilt over minor failings.
- Problems with clear thinking, concentration, and decision-making.
- Recurrent thoughts of death or suicide, or making a suicide attempt.

Demographics

Recent research indicates that 4.9% of the population of the United States meets the diagnostic criteria for MDD at any given time, but 17.1% will experience at least one episode of the disorder at some point during their lives. While the disorder may affect people at any age, it is most commonly diagnosed in young adults in their twenties. For reasons that are not well understood, women are twice as likely to develop MDD as are men; prior to puberty, however, MDD is about equally common in girls and boys. Adolescence is a high-risk period for MDD; while suicide may result from impulsive behavior under **stress** rather than from MDD, it is noteworthy that about 14% of all teenage deaths are due to suicide. The figures for gay and lesbian youth indicate that as many as 20%–35% make suicide attempts. Other risk factors include Hispanic ethnicity; younger age at onset; lower levels of education or income; and being separated or divorced.

Depression appears to have become a more common disorder over the past century. Epidemiologists studying the incidence of depression across time compared groups of people born between 1917 and 1936, between 1937 and 1952, and between 1953 and 1966; their results indicated that the rate of depression increased progressively from one generation to the next. While no single explanation for the rise in depressive disorders emerged, some researchers have suggested that the breakdown of social support networks caused by higher rates of family disruption and greater social mobility may be important contributing factors.

Diagnosis

Major depressive disorder may be diagnosed when a person visits his or her family doctor with concerns about mood, changes in appetite or sleeping patterns, and similar symptoms. Doctors in family practice, in fact, are more likely to be consulted by patients with depression than doctors in any other medical specialty. In addition, a large proportion of people discuss depressed feelings with their clergyperson, who, in the mainstream Christian and Jewish bodies, has typically been trained to recognize the signs of depression and to encourage the person to see their doctor. In some cases the patient may be

brought to see the doctor by a concerned spouse or other family member.

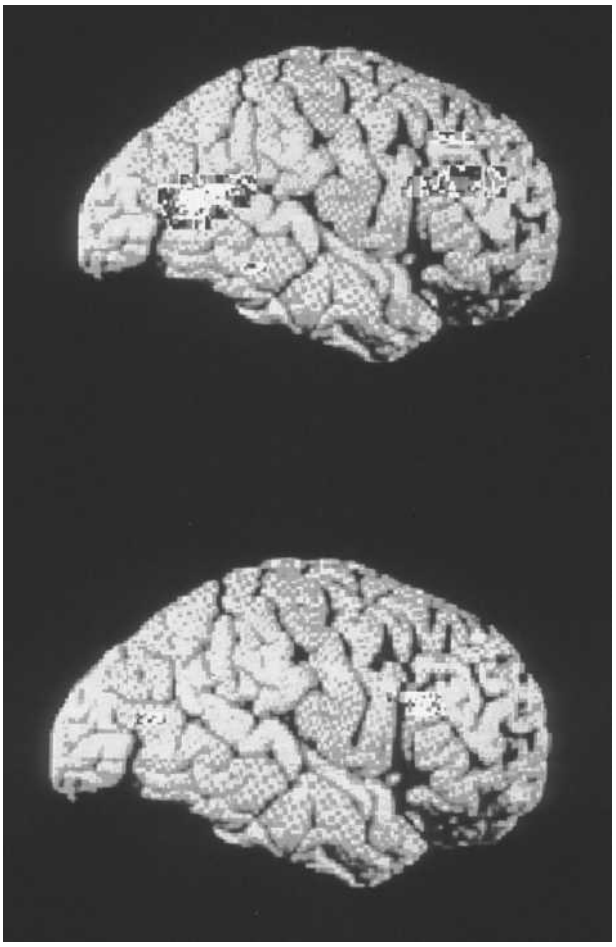
The **diagnosis** of MDD involves a constellation of symptoms in addition to depressed mood. After taking a careful history, including asking the patient about his or her sleeping patterns, appetite, sex drive, and mood, the doctor will give the patient a physical examination to rule out other possible causes of the symptoms. Certain other disorders may resemble MDD, including cognitive dysfunction caused by the direct effects of a substance (drug of abuse, medication, or toxic chemical); various medical conditions (i.e., an underactive thyroid gland; strokes; or early stages of **dementia**), or other mental disorders. Such stressful life events as normal bereavement may also produce behaviors similar to those associated with MDD; while a bereaved person may appear to have many of the characteristics of MDD, the disorder would not be diagnosed unless the symptoms continued for more than two months or were extreme in some way. As part of the diagnostic interview, the doctor may give the patient a brief screening questionnaire, such as the **Beck Depression Inventory**, in order to obtain a clearer picture of the symptoms. In addition to interviewing the patient, the doctor may talk to family members or others who can provide information that the patient may forget, deny, or consider unimportant.

The diagnosis of MDD is complicated by the fact that people with MDD frequently suffer from other mental illnesses at the same time, including anxiety disorders, substance abuse problems, and **personality disorders**. Given that the patient's symptoms may vary according to age, sex, and stage of the illness, some clinicians have suggested that MDD may actually be a collection or group of disorders with a small number of underlying core symptoms rather than a single entity.

The diagnosis of a person with MDD may also include certain specifiers, including the severity and chronicity of the disorder; the presence of psychotic features (delusions or hallucinations) or catatonia (remaining motionless for long periods of time, and other peculiarities of posture, movement, or speech); melancholia (depressed mood that is worse in the morning; early morning wakening; psychomotor retardation or agitation; significant weight loss; or inappropriate guilt); and information regarding postpartum status. If the depression is currently in remission, this fact is also commonly listed as a diagnostic specifier.

Treatments

Because MDD can have a devastating impact on a person'slife, the importance of effective treatment cannot be overestimated. Treatment strategies have evolved over the years according to researchers' varying opinions of the underlying causes of depression, but the outpouring of interest in MDD allows treatment providers to select from a variety of tested approaches.



Colored positron emission tomography (PET) scans comparing the brain of a depressed person (top) with the brain of a healthy person. *(Photo Reasearchers, Inc. Reproduced by permission.) See color insert for color version of photo.*

Psychotherapy

Cognitive psychotherapies for depression are based on the belief that depressed people perceive themselves and the world in unrealistically negative ways. Considerable research has been done regarding the cognitive dimension of depression; for example, studies find that depressed people pay more attention to negative events than to positive ones, and that dwelling on unpleasant experiences prolongs and worsens depressive episodes. Cognitive therapists help patients identify the automatic thoughts that lead them to anticipate poor outcomes or to interpret neutral events in negative ways.

The patient is also encouraged to challenge negative thoughts by comparing his or her expectations of events with actual outcomes.

Evidence that poor interpersonal relationships may heighten vulnerability to depression, along with findings that depressed adults and depressed children tend to provoke negative reactions from other people, has prompted the use of **social skills training** as a form of treatment. In this type of therapy, patients are trained to recognize actions and attitudes that annoy or distance other people, and to replace these behaviors with more appropriate ones. Social skills training may be particularly helpful to depressed persons who tend to isolate themselves and have lost confidence in their ability to develop healthy relationships. This treatment model promotes the idea that depression is likely to lift when the patient becomes adept at making new friends and establishing rewarding social supports.

Psychodynamic psychotherapy is often effective in treating patients with MDD whose depression is related to unresolved issues from the past, particularly abuse or other painful childhood experiences. The growth of insight into one's emotional patterns, as well as the supportive aspects of this form of therapy, offers considerable relief from emotional pain to many patients.

Medications

The use of medications in the treatment of depression began in the late 1950s with the successful introduction of tricyclic antidepressants and MAO inhibitors. Treatment of depression with medications has greatly increased since the advent of selective serotonin reuptake inhibitors (SSRIs) such as **fluoxetine** (Prozac) and **sertraline** (Zoloft). While these medications are no more effective than their predecessors, they have fewer side effects and are much safer for patients who may be likely to overdose. Selecting the optimal antidepressant medication is not always a straightforward process, however, and the patient may have to try out various drugs for a period of weeks or months before finding one that is effective for him or her. In addition, while the SSRIs have comparatively few side effects, such complaints as loss of sexual interest or functioning, nervousness, headaches, gastrointestinal complaints, drowsiness, and insomnia can be significant obstacles to the patient's taking the medication as directed.

Other mainstream approaches

The use of **electroconvulsive therapy** (ECT), initially introduced in the 1930s, was virtually abandoned as a treatment for MDD for many years, largely as a result of the effectiveness and convenience of psychotropic (mind-altering) medications. Since the 1980s, however, interest in the procedure has renewed; in 1990 the American Psychiatric Association published new guidelines for the use of ECT. Despite media portrayals of ECT as an outdated and cruel form of treatment that causes considerable pain, in actuality the patient is given a sedative and the electrical stimulation is calibrated precisely to produce the maximum therapeutic effects. ECT may be the first line of treatment when a patient cannot tolerate the customary medications or is at high risk of harming themselves; it is more commonly used with patients who fail to respond to drug treatment. In terms of effectiveness, however, ECT actually outperforms medications even among patients who are helped by antidepressants, as well as those who are resistant to drug treatment.

The use of phototherapy (**light therapy**) has proven to be the treatment of choice for patients diagnosed with seasonal affective disorder. Although the reasons for the effectiveness of phototherapy are not yet clear, treatment involves exposing the eyes to bright (2,500 lux) light for several minutes a day. Currently, however, there is little evidence to suggest that phototherapy is useful in the treatment of other types of MDD.

Alternative and complementary treatments

The National Center for Complementary and Alternative Medicine (NCCAM) is conducting an ongoing series of clinical tests of alternative and complementary treatments for depression. Those that have been shown to reduce symptoms of depression and compare favorably with conventional treatments include **acupuncture**; Ayurvedic medicine; **meditation**; and a therapeutic diet designed to be free of caffeine and refined sugar.

Herbal preparations are common alternative treatments for depression; in fact an NCCAM study found that depression is the single most common reason for people in the United States to purchase herbal remedies. Some, such as St. John's wort, have been used in Europe for decades. The German Commission E, which regulates government approval of herbal preparations in German-speaking Europe, recently approved the use of ***Gingko biloba*** extract as a treatment for depression. The most important caution is that persons who are using herbal remedies, whether to treat depression or other conditions, *should always tell their doctor what they are taking, how much, and how often*. This warning is crucial because some herbal preparations that are safe in themselves can interact with prescription medications. In particular, St. John's wort has been reported to cause interactions with fluoxetine (Prozac).

Some complementary approaches appear to be helpful to persons with depression because they offer pleasurable experiences for the senses or lift the person's spirit. These include **aromatherapy**; music therapy; pet therapy; humor; therapeutic massage; and **yoga**.

Prognosis

Major depression is increasingly viewed as a chronic condition for many people. Left untreated, a depressive episode may last four months or longer, regardless of the age of onset. While most people recover fully from a given depressive episode, eventual recurrence is common. Long-term studies of people with MDD indicate that about 60% of patients who have one episode of depression will have a second episode; with each succeeding episode, the chances of a subsequent episode increase. For example, persons having a third episode stand a 90% chance of having a fourth. Between depressive episodes, the patient's mood may return to a nondepressed state (in about two-thirds of the cases) or continue to show some degree of impairment (one-third of cases). Patients who recover only partially between episodes appear to be at especially high risk of recurrence.

Community studies indicate that about 60% of the people diagnosed with MDD are greatly improved or fully recovered by one year after diagnosis. A very severe initial episode of depression, the presence of a coexisting **dysthymic disorder**, or the existence of a serious medical condition are associated with a poorer prognosis.

Prevention

While programs specifically aimed at preventing MDD are not widespread, early interventions with children to address some of the issues related to depression have met with success. In particular, social skills training has been found to reduce symptoms of depression, perhaps by enabling children to develop the kinds of social supports and friendships that promote good mental health. Cognitive behavioral techniques that teach people to challenge dysfunctional thought patterns, such as the tendency to deny responsibility for good outcomes and to feel overly responsible for negative events, has been found to successfully reduce the rates of depressive symptoms in children and college students. In addition, psychoeducational work with parents having mood disorders has been effective in improving the adjustment of their children. Long-term follow-up of such approaches is incomplete, but these studies support the possibility that improved individual and family functioning may help to lower rates of depression in the future.

As the factors that increase an individual's vulnerability to depression become better understood, effective strategies for early **intervention** and possible prevention become possible. Brief therapies that target such symptoms as maladaptive thought patterns or interpersonal problems may lower the risk of serious mood disturbances. Knowledge of the mental health implications of natural or humanly caused disasters has already resulted in much improved mental health services to communities in need. It is realistic to expect that appropriate treatment will become more available and accessible to people experiencing less dramatic setbacks to their ability to function in the future.

See also Adjustment disorder; Catatonic disorder; Children's Depression Inventory (CDI); Creative therapies; Family psychoeducation; Genetic factors and mental disorders; Grief

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National Depressive and Manic-Depressive Association. 730 North Franklin Street, Suite 501, Chicago, IL 60610-3526. (800) 826-3632. <www.ndmda.org>.

OTHER

National Center for Complementary and Alternative Medicine (NCCAM) Clearinghouse. P.O. Box 7923, Gaithersburg, MD 20898. (888) 644-6226. TTY: (866) 464-3615. Fax: (866) 464-3616. <www.nccam.nih.gov>.

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Jane A. Fitzgerald, Ph.D.

Male erectile disorder *see* **Erectile dysfunction**

Male orgasmic disorder

Definition

Male orgasmic disorder may be defined as a persistent or recurrent inability to achieve orgasm despite lengthy sexual contact or while participating in sexual intercourse.

The mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders*** *(DSM-IV-TR)*, includes this disorder among the **sexual dysfunctions**, along with **premature ejaculation**, **dyspareunia**, and others.

Description

The individual affected by male orgasmic disorder is unable to experience an orgasm following a normal sexual excitement phase. The affected man may regularly experience delays in orgasm, or may be unable to experience orgasm altogether.

Normal orgasm

First, it is important to this discussion to understand the characteristics of a "normal" orgasm. The sensation of orgasm in the male includes emission followed by ejaculation. The term emission refers to a sensation of impending ejaculation produced by contractions of the prostate gland, seminal vesicles, and urethra accompanied by generalized muscular tension, perineal contractions, and involuntary pelvic thrusting. Orgasm is followed by a period of resolution characterized by feelings of well-being and generalized muscular relaxation. During this phase, men may be unable to respond to further sexual stimulation, erection, and orgasm for a variable period of time.

It is also important to distinguish orgasm from ejaculation, although in most instances they occur almost simultaneously. Orgasm is a peak emotional and physical experience, whereas ejaculation is simply a reflex action occurring at the lower portion of the spinal cord and resulting in ejection of semen. Some men have been able to recognize the separation of the two processes, enabling them to experience multiple orgasms without the occurrence of ejaculation. Once ejaculation takes place, a period of recovery time is required prior to a subsequent orgasm.

The sensation of orgasm differs between individuals, and individual orgasms may differ in the same person. All orgasms share certain characteristics in common including rhythmic body and pelvic contractions, elevation of the heart rate, systemic hypertension, hyperventilation, and muscle tension, followed by the sudden release of tension.

The physiological mechanism of normal orgasm

The cycle of sexual response is under the control of a balanced interplay between the two major nervous systems, the sympathetic and the parasympathetic. In general, the sympathetic nervous system prompts action whereas the parasympathetic system's main action is recovery and calming. In order for a penis to become erect, its smooth muscles are relaxed and it becomes congested with blood vessels. This process is mediated by a complex cascade of humoral, neurological and circulatory events in which the parasympathetic nervous system plays a key role. Orgasm and ejaculation and subsequent relaxation of the penis are predominantly functions of the sympathetic nervous system.

Thus, whereas emission is a balanced interplay between the parasympathetic and sympathetic nervous systems, orgasm and ejaculation are predominantly under the control of the sympathetic nervous system. The mechanisms of this system may be blocked by impaired function of the **brain** or of the hormonal, circulatory, and neurological systems. Additionally, certain medications may block these actions.

Abnormalities affecting the process of orgasm

Abnormalities in these processes may be "primary" or "secondary." Primary abnormalities are of lifelong duration with effective sexual performance never having been experienced. Secondary abnormalities are acquired after a period of normal function. If an orgasmic problem only occurs under a particular set of circumstances, or only with certain sexual partners, the condition is considered to be "situational" rather than "generalized" (occurring regardless of the circumstances or partner). The defect in sexual function may be total or partial.

The evidence strongly suggests that orgasm has more to do with the brain than with the body. Electrode stimulation of certain parts of the brain will produce sexual pleasure similar to that produced by physical stimulation. The fact that orgasm occurs during sleep is supportive of this concept.

Causes and symptoms

Causes

The cause of male orgasmic disorders may be organic (related to a condition in the body), but, in most cases, is of psychological origin. It is important for the physician to make every effort to find an underlying cause because the therapy and prognosis depend upon it. A detailed history (including an interview with the sexual partner, if feasible), a general physical examination, the performance of certain laboratory and, in some cases, special tests, are important in the investigation of the underlying cause of the male orgasmic disorder.

Organic causes of male orgasmic disorder include the following:

- Hypogonadism, in which the testes do not produce enough testosterone.
- Thyroid disorders (both hyperthyroidism—too much thyroid hormone— and hypothyroidism, or abnormally low levels of thyroid hormone).
- Pituitary conditions (Cushing's syndrome, excessive production of the hormone that induces lactation called prolactin).
- Diseases that affect the nervous system, such as strokes, multiple sclerosis, diabetic neuropathy, spinal cord injuries.
- Surgery affecting the prostate and other pelvic organs.
- Diseases of the penis.
- Substance abuse, including alcohol.

KEY TERMS

Antihypertensive—An agent used in the treatment of hypertension (high blood pressure).

Diabetes mellitus—A chronic disease affecting the metabolism of carbohydrates that is caused by insufficient production of insulin in the body.

Diabetic neuropathy—Condition existing in people with diabetes in which the nerves at the extremities, especially the feet, are less sensitive to touch and injury.

Humoral—A term describing a hormonal substance secreted by an endocrine gland (such as the thyroid).

Perineal—An anatomical area located between the external genitals and the anus.

Phenothiazine—A class of drugs widely used in the treatment of psychosis.

Prostate gland—The gland at the base of a male's urethra that produces a component of semen.

Retroperitoneal—The anatomical area between the peritoneum (lining of the abdominal cavity) and the muscular and connective tissues of the abdominal wall.

Seminal fluid—Fluid composed of semen from the testes and prostatic secretions.

Seminal vesicles—Sac-like structures bordering the male urethra and serving as storage depots for the seminal fluid.

Urethra—The tubular passage conducting urine from the bladder to the exterior. In the male, the urethra traverses the penis.

- Certain medications. Some of these medications include: the phenothiazines [antipsychotics such as **chlorpromazine** (Thorazine) or **trifluoperazine** (Stelazine)]; certain medications used to treat high blood pressure, including the thiazides [such as triamterene (Dyazide) or spironolactone (Aldactone)] and **beta blockers** [such as **propranolol** (Inderal)]; and the tricyclic antidepressants such as **doxepin** (Sinequan) and **protriptyline** (Vivactil).

The most common causes of the male orgasmic syndrome are psychological in nature. The responsible psychological mechanisms may be "intrinsic" (due to basic internal factors), or "extrinsic" (due to external or environmental factors).

Intrinsic psychological factors that may cause male orgasmic disorder include:

- depression
- feelings of guilt, anger, fear, low self-esteem, and anxiety
- fear of getting the partner pregnant or of contracting a sexually transmitted disease or HIV

Extrinsic psychological factors that may cause male orgasmic disorder include:

- living under conditions that cause undue **stress**
- unsatisfactory relationship with sexual partner
- past history of traumatic sexual encounters such as sexual abuse, rape or incest
- having been raised in an atmosphere of strict sexual taboos

Environmental factors may interfere with sexual functioning. There may be no safe, private place in which the patient can exercise sexual activity or he may be too fatigued from other activities to participate sexually. The difficulties in striving for "safe sex" and the psychological effects and stresses that may result from homosexuality may also interfere with sexual function.

Symptoms

In order to be diagnosed with male orgasmic disorder, the following symptoms must be present according to the *DSM-IV-TR*:

- Persistent or recurrent delay in, or absence of, orgasm following a normal sexual excitement phase during sexual activity that the clinician judges to be adequate. The affected man's age is considered, as well.
- As with all of the sexual dysfunctions, the manual states that the dysfunction must cause the affected man "distress or interpersonal difficulty." According to the *DSM-IV-TR,* the orgasmic dysfunction cannot be better accounted for by another disorder (except another sexual disorder), and cannot be due exclusively to the direct effects of substance abuse, a medication, or a general medical condition. This entry, however, discusses the full scope of male orgasmic difficulties, and so discusses general medical conditions and medications as well as psychological factors.

In addition to specific symptoms involving sexual function (inability or delay in reaching orgasm after sufficient stimulation), most patients complain of anxiety, guilt, shame and frustration, and many develop bodily complaints on a psychological basis. Although sexual dysfunction usually occurs during sexual activity with a partner, the clinician should inquire about sexual function during masturbation. If problems occur during masturbation, the problem probably has nothing to do with the sexual partner.

The physician should differentiate male orgasmic disorder from other sexual disorders such as retarded or delayed ejaculation and retrograde ejaculation. In both of these conditions, orgasm occurs but is delayed or, in the case of retrograde ejaculation, occurs in a retrograde direction (into the bladder).

Demographics

Male orgasmic disorder is found in all races and ethnic groups. In the case of the lifelong type of the disorder, manifestations will occur around the age of puberty. In certain genetic hypogonadism disorders, such as Klinefelter's syndrome, certain bodily signs and symptoms may alert the physician. Similarly, in associated thyroid, testicular and pituitary abnormalities, there may be other manifestations of the underlying disorder. In the acquired type of male orgasmic disorder, the patient will have had the previous experience of normal sexual function. In these cases, it is usually a situational factor that precipitates the disorder.

Diagnosis

The **diagnosis** is usually readily made on the basis of the patient's history and the presence of the *DSM-IV-TR* diagnostic criteria. Male orgasmic disorder may be part of a complex of sexual malfunctioning that may include **erectile dysfunction**, abnormalities in ejaculation (such as premature ejaculation or retrograde ejaculation), and **hypoactive sexual desire disorder**.

In order to differentiate between the various potential disorders, the physician may request laboratory tests and/or may perform further diagnostic evaluations. Blood plasma levels of testosterone are of help in diagnosing hypogonadism. A number of tests of thyroid, pituitary and adrenal function are available to diagnose hormonal abnormalities of those glands. A test for nocturnal penile erections may be performed to diagnose erectile dysfunction.

Treatments

If an extrinsic mechanism is discovered as the cause of the orgasmic disorder, steps should be taken to eliminate or ameliorate the problem. An example would be substance or alcohol abuse or the use of certain provocative medications. In the case of antihypertensives, for example, a number of equally effective agents are available if the one in current use is suspect. Therapy should be directed toward improvement of concurrent conditions such as diabetes that may be having an adverse

effect on sexual function. Environmental factors that interfere with sexual activity should be corrected.

In the majority of cases, **psychotherapy** will be suggested even in those cases where psychological factors are secondary rather than the primary mechanism for the disorder. Such treatment should be rendered by therapists with special training in the disorders of sexual function and who can tactfully evaluate the sexual compatibility of the patient and his partner. Treatment usually requires the support of the sexual partner in improving both the psychological as well as the physical aspects of the problem. A step-wise program of partner stimulation of the patient to initially ejaculate outside the vagina, then at the vaginal labia, and finally inside the vagina may be helpful.

Prognosis

The prognosis of the patient with male orgasmic syndrome is dependent on whether the condition is lifelong or acquired and the condition's causes. Prognosis is best when it can be demonstrated that the condition is related to some extrinsic or environmental factor that can be corrected or ameliorated. The prognosis is also favorable in those cases that are due to a remedial organic condition such as a thyroid disorder or hypogonadism. The prognosis is guarded when the disorder is found to be secondary to a deep-seated and chronic psychological or actual psychiatric problem that, in itself, carries an unfavorable prognosis.

Prevention

There are no definitive steps that can be taken to prevent the onset of the male orgasmic disorder. Prompt recognition of the syndrome is important so that appropriate therapy can be attempted as early as possible. As with many chronic conditions, the longer the condition exists, the more difficult therapy becomes.

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Malingering

Definition

The central theme to all definitions of malingering is that the term applies to persons who deliberately pretend to have an illness or disability in order to receive financial or other gain, or to avoid punishment or responsibility.

Description

Personal gain is always the motivation for malingering. Some external reward is sought and is the rationale for feigning an illness. For example, the criminal who does not want to pay for his/her crime, the soldier who does not want to fight, or the person who wishes to be paid for a nonexistent disability all may be tempted to feign an illness.

Malingering can take many forms. However, as specifically related to mental illness, the tendency is to fake more common disorders such as **major depressive disorder**, **post-traumatic stress disorder**, and **panic disorder** with **agoraphobia**. With very little coaching or research, even a beginner can simulate symptoms of these disorders. Generalized symptoms such as headaches, dizziness, low back pain, stomach pain, etc., are easily manufactured, and x rays, **magnetic resonance imaging** (MRIs), or CAT scans (computed axial tomography) are unable to determine a physical cause.

Malingerers tend to avoid symptoms such as those associated with more serious psychiatric disorders, because the pretense is very difficult to maintain and objective measures could detect the difference. For example, hearing voices and seeing demons, or living with the idea that others can hear unspoken thoughts, would become a difficult act to maintain over time. On the other hand, to feign a sad mood, loss of interest in formerly enjoyed activities, or a low energy level may not be so difficult to demonstrate. Likewise, responding positively to a series of questions about having heart palpitations, sweating, dizziness, or fear of impending death, could be done readily.

The concept that fakers use less severe symptoms to escape detection was validated in 2001 in a research study. Individuals were asked to fake mental illnesses in such a way as to avoid detection by sophisticated psychological tests. All or portions of the following tests were employed in the research: the Structured Inventory of Malingered Symptomatology, the Psychopathic Personality Inventory, the M-Test, and the Trauma Symptom Inventory. Slightly over 11% of the 540 research participants successfully avoided detection and were diagnosed with real disorders instead of with malin-

gering. Questionnaires completed by those who successfully faked symptoms showed that they avoided detection by endorsing fewer actual symptoms, staying away from unduly strange or bizarre symptoms, and responding based upon personal experience.

Although ordinarily an intended fraud, malingering may serve an adaptive purpose under circumstances of duress, such as while being held captive. Faking an illness at such a time may allow a person to avoid cooperating with their captors or to avoid punishment.

Causes and symptoms

Lying for personal benefit has existed since the beginning of time. As previously stated, personal gain is the goal of the malingerer.

The symptoms may vary a great deal from person to person.

Demographics

Due to the difficulty of determining and exposing malingering, the incidence is unknown.

Diagnosis

When attempting to diagnose malingering, mental health professionals have three possibilities to consider. First, there is the possibility that the illness feigned by the malingerer is real. However, once it is determined that the disorder has no basis in fact, the professional is left with two viable diagnoses: **factitious disorder** and malingering. Factitious disorder is a legitimate malady, but malingering is not. Both have to do with feigned illnesses.

Unlike malingering, the individual with factitious disorder produces fake symptoms to fulfill the need to maintain the "sick role"—a sort of emotional gain. Being "sick" gives the person with factitious disorder attention from physicians and sympathy from friends and loved ones. Thus, this individual's goal is not the same as the malingerer's.

With malingering, motivation is always external and is designed to accomplish one of three things: (1) evade hard or dangerous situations, punishment, or responsibility; (2) gain rewards such as free income, source for drugs, sanctuary from police, or free hospital care; or (3) avenge a monetary loss, legal ruling, or job termination.

Mental health practitioners become alert to the possibility of malingering when circumstances exist that might help promote such a facade. Malingering is suspected when any combination of events such as the following occur:

- A person is referred by his/her attorney for an evaluation.
- There is a noticeable and distinct difference between the level of distress or disability claimed by the person when compared to information obtained by objective means. (Objective means could take the form of personal observation, task performance ability by the person, or a psychological test like those mentioned above.)
- There is a lack of cooperation from the individual.
- A **diagnosis** of **antisocial personality disorder** exists.

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American Psychiatric Association. 1400 K Street NW, Washington D.C. 20005. <http://www.psych.org>.

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Managed care

Definition

Managed care is a generic term for various health care payment systems that attempt to contain costs by controlling the type and level of services provided. Health maintenance organization (HMO) is a term that is often used synonymously with managed care, but HMOs are actually a particular type of managed care organization.

Purpose

Health care reform has been an increasingly urgent concern in the United States over the past 40 years. Until recently, the primary source of health care coverage was indemnity insurance, which pays or reimburses the cost

KEY TERMS

Capitated payment system—A contract between managed care organizations and health care providers involving a prepaid amount for blocks of services.

Carve-out plans—Managed care plans that make provision for mental health services by creating subcontracts involving different terms of payment and utilization review from those used for general health care.

Case manager—A professional who designs and monitors implementation of comprehensive care plans (i.e., services addressing medical, financial, housing, psychiatric, vocational, social needs) for individuals seeking mental health or social services.

Case rate—A type of contract between managed care organizations and health care providers involving a prepaid amount for services on a case-by-case basis.

Deductible—The amount of money that must be paid out of pocket by health care consumers before the insurance provider will make payments.

Health maintenance organization (HMO)—A type of managed care system that involves payment contracts with a group or panel of health care providers.

Health Maintenance Organization Act of 1973—Federal legislation that provided aid to develop HMOs.

Indemnity insurance—Insurance plans that pay on a fee-for-service basis in the event of illness or injury.

Medicaid—A program jointly funded by state and federal governments that reimburses hospitals and physicians for the care of individuals who cannot pay for their own medical expenses. These individuals may be in low-income households or may have chronic disabilities.

Medicare—A federally funded health insurance program for individuals age 65 and older, and certain categories of younger persons with disabilities.

Preferred provider organization (PPO)—A type of managed care system involving payment contracts with a group or panel of health care providers.

Premium—The cost of enrollment in a health insurance plan. Premiums are usually paid on a monthly basis.

Utilization review—A process used by managed care organizations involving scrutiny of service care delivery to determine whether services are necessary.

of medical services in the event of a person's illness or injury. Indemnity insurance gives health care providers few reasons to use less expensive forms of treatment—the insurance companies generally pay for any treatment deemed necessary by a physician. Presumably, this type of system encourages providers to overuse expensive, unnecessary treatments and diagnostic procedures. Patient co-pays and deductibles attempt to limit excessive use of medical services. Yet costs continue to rise, resulting in insurance companies' frequently raising premium prices.

The primary intent of managed care is to reduce health care costs. Emphasis is placed on preventive care and early **intervention**, rather than care provided after an illness or injury has occurred. The responsibility of limiting services is placed on the service provider rather than the consumer. This limitation is achieved by (a) "gatekeeper" policies that require individuals to get referrals for specialized treatment from their primary physicians; (b) financial incentives (either bonuses or withholding money) for providers to restrict services and contain costs; (c) guidelines requiring adherence by providers at the cost of being dropped from the plan for noncompliance; (d) review of services by the managed care organization and **denial** of payment if services are considered unnecessary.

Description

Health maintenance organizations have been in existence in the United States since the late 1800s. It was not until the 1950s, however, that the government began to encourage the development of HMOs. In 1973, the Health Maintenance Organization Act was passed; and in 1978, a Congressional amendment increased federal aid for HMO development. From 1980 to 1989, enrollment in HMOs increased from 9 million to 36 million Americans. By 1990, 95% of private insurance companies used some form of managed care. In the 1990s, managed care was incorporated into Medicare and Medicaid plans as well.

Managed care organizations frequently contract with a group or panel of health care providers. HMOs and PPOs (preferred provider organizations) are examples of these types of contracts. Individuals insured under an HMO or PPO may receive care only from providers on the panel. These providers are expected to deliver services according to specific stipulations. Payment is often subject to utilization review, in which delivery of medical services is scrutinized to determine whether the services are necessary. The review may occur with each episode of treatment, or may be ongoing through the use of a case manager. If the managed care organization thinks that the services were unnecessary, payment is denied.

Payment arrangements between managed care organizations and care providers are often made in advance. Capitated payment systems are typically used with large health care facilities that serve many people. The health care provider receives a set amount of money each month based on the number of individuals covered by the plan. The provider may or may not serve that many people in one month. Capitation systems provide a steady, reliable cash flow, but involve some economic risk because the services provided may exceed the dollar amount allotted. Another type of payment system uses case rates. The provider receives a predetermined amount of money per individual on a case-by-case basis. The amount of money reflects the estimated service costs to treat the individual patient's condition. Again, the provider takes the risk that unanticipated services will be required.

In the past, mental health services (including substance abuse treatment) were routinely excluded from managed care plans. In the 1970s, some mental health care coverage was required in order to meet federal qualifications. Carve-out plans were developed in the 1990s. These plans essentially create a separate managed care plan for mental health services. Mental health services tend to be covered at a lower rate than general health services and have also been cut back more severely. From 1988 to 1997, mental health care spending decreased by 54%, which reflects cutbacks 670% higher than those for general health care benefits. Mental health care providers are also subjected to higher levels of utilization review than medical care providers.

Ethical concerns

Managed care has been successful in fulfilling its primary purpose of lowering health care costs in the United States. Statistics show drastic decreases in the use of inpatient care and accompanying overall reduction in costs. Many observers, however, would argue that the quality of care has suffered as a result. Individuals have fewer choices regarding the locations where they can receive treatment. If a managed care organization closes, individuals under that plan must switch to other care providers under a new plan, which disrupts ongoing treatment. Care providers often feel that their clients are denied essential care in favor of saving money. Employers have become disillusioned because of increasing disability claims due to employees having received inadequate treatment for illnesses or injuries. In addition to disability claims, inadequate treatment results in hidden costs to employers in terms of lost productivity.

Another factor in decreased quality of care involves conflicting loyalties for health care providers. On the one hand, providers want to ensure quality care for their clients. On the other hand, they are encouraged to provide the least amount of care possible in order to receive financial benefits. Just as dishonest practice was suspected in conjunction with indemnity insurance, managed care creates a powerful potential for inappropriately addressing patients' needs.

Future directions

Due to growing popular discontent with managed care organizations, many critics believe that the system will not continue in its current state. No one, however, expects managed care to disappear completely and indemnity plans to rise to their former prominence. Changes are expected to occur as managed care programs begin competing among themselves. Cost and efficiency will no longer be the main selling point; quality of services will take precedence. One researcher has suggested that along with new systems of managed care and continuing systems of indemnity plans, health care providers may even organize and offer services directly to employers, thus eliminating the middlemen. This development would be beneficial to all involved: employers would pay less; providers would be better compensated; and clients would receive better care.

See also Case management

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Department of Managed Health Care. California HMO Help Center, 980 Ninth Street, Suite 500, Sacramento, CA 95814-2725. <http://www.hmohelp.ca.gov>.

Medicare. 1-800-MEDICARE. <www.medicare.gov>.

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Mania *see* **Manic episode**

Manic episode

Definition

A discrete period lasting a week or more during which a person experiences mania, an abnormally elevated, cheerful, or euphoric mood.

Description

A person experiencing a manic episode shows persistent and often inappropriate enthusiasm which may involve taking on new projects for which he or she is ill suited. It might also involve engaging strangers in detailed conversations, acting without concern for consequences of one's actions, or increased sexual activities. Less commonly, a person may be abnormally irritable during a manic episode. On average, the episodes begin before age 25. This means that some individuals experience their first episode while in their teens and others during middle age.

Psychiatrists use five criteria to identify someone in the midst of this type of mood episode. First, the period of abnormal behavior must persist for at least one week unless the person is admitted to a hospital. Typically, the episodes last from a few weeks to a few months. Second, the **diagnosis** requires three additional symptoms if the mood change results in expansive behavior, or four if it results in unnatural irritability. These symptoms include an unwarranted sense of self-importance, a tendency to be easily distracted, a decreased need for sleep, a rapid flow of ideas with one replacing another before the first is acted upon, an inability to sit still or increased activity directed at achieving some goal, an irrepressible need to talk, and finally, a devotion to some activity the patient finds pleasurable but could be harmful. The third criterion is that the symptoms do not qualify the patient for a diagnosis of **mixed episode**. Fourth, the patient can not function normally at home or at work, or shows signs of **psychosis**. The fifth and last criterion is that the cause of the episode can not be attributed to side effects from any drug abuse, medication, medical treatment, or medical condition.

Many of these symptoms are also present in a hypomanic episode. A hypomanic episode is similar to a manic episode, but the symptoms may be experienced to a lesser extent. The main differences between a manic and hypomanic episode are the following:

- A hypomanic episode may only last four days, whereas a manic episode, by definition, lasts one week.
- In a manic episode, psychotic features (**hallucinations** and **delusions**) may be present, but in a hypomanic episode, they cannot be.
- A manic episode significantly impairs the affected person's functions, but a hypomanic episode does not.

Both of these kinds of episodes may be seen in patients with **bipolar disorder**.

Dean A. Haycock, Ph.D.

Maprotiline

Definition

Maprotiline is an oral antidepressant. It is a member of the tetracyclic antidepressant family of compounds. In the United States, it is sold under the trade name Ludiomil.

Purpose

Maprotiline is an antidepressant intended for use by persons with depressive **neurosis** and bipolar syndrome. It is also occasionally used for the relief of anxiety associated with depression.

Description

Maprotiline elevates mood. The precise pharmacological mode of action is not fully understood but it is

KEY TERMS

Barbiturates—A class of medications (including Seconal and Nembutal) that causes sedation and drowsiness. They may be prescribed legally, but may also be used as drugs of abuse.

Bipolar syndrome—An abnormal mental condition characterized by periods of intense elation, energy and activity followed by periods of inactivity and depression.

Guanethidine—An antihypertensive drug used to treat high blood pressure.

Hallucination—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Manic—Referring to mania, a state characterized by excessive activity, excitement or emotion.

Monoamine oxidase inhibitors—A group of antidepressant drugs that decrease the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Physostigmine—A short-acting drug that enhances levels of a substance (acetylcholine) between neurons in the brain.

Syncope—A brief lapse of consciousness caused by a temporarily insufficient flow of blood to the brain.

Tachycardia—A pulse rate above 100 beats per minute.

thought to inhibit the reuptake of the neurotransmitter norepinephrine at nerve endings in the **brain**. It is prescribed in 25-, 50-, and 75-mg tablets.

Recommended dosage

The recommended initial dosage of maprotiline is 75 mg, given by mouth in three 25-mg administrations. The initial dosage should be maintained for at least two weeks. Therapeutic results maybe observed in three to seven days. Typically, initial administration may have to be continued for two to three weeks before results are observed.

The recommended total dosage is 150 mg per day. Dosage should be increased 25 mg at a time. The maximum daily dosage in severely depressed persons is 225 mg. The elderly may require a total initial dosage of 25 mg per day.

Precautions

Maprotiline should be discontinued or reduced in dosage prior to surgery. This is due to the potential for interactions with anesthetic agents.

Maprotiline may promote seizure activity: of all the cyclic antidepressants it probably causes the highest incidence of **seizures** and has thus fallen out of favor with most psychiatrists. Also for this reason, it should not be combined with other neuroleptics (antipsychotics) that can also cause seizures. The drug increases the effect of alcohol and should not be taken with products containing alcohol or **barbiturates**. Persons taking monoamine oxidase inhibitors (MAOIs), such as Parnate (**tranylcypromine**) and Nardil (**phenelzine**), should not take maprotiline.

The possibility of **suicide** is a component of depression. A minimal number of doses should be dispensed at any one time to minimize the potential for use as a suicide agent. Because the drug may lower the threshold for a **manic episode** among persons with **bipolar disorders**, it should be used only with caution and under close supervision.

Side effects

The most commonly reported side effect of maprotiline is dry mouth. Slightly more than one person in five (22%) experiences this effect. Approximately 16% of users experience drowsiness, dizziness is reported by 8%, and nervousness and constipation by 6%. Other less common side effects include anxiety, agitation, **insomnia**, blurred vision, tremor, weakness, **fatigue**, nausea, and headache with blurred vision are also reported. Other rare side effects are similar to those experienced by users of tricyclic antidepressants. These include abnormally high or low blood pressure, tachycardia, and syncope. **Hallucinations**, disorientation, and mania have been reported, as have vomiting, diarrhea, and gastric distress.

Interactions

Cimetidine and **fluoxetine** reduce the elimination of maprotiline, thus increasing its plasma concentration. Barbiturates and phenytoin increase the elimination of maprotiline, thus decreasing its plasma concentration.

Cardiovascular toxicity has been reported when maprotiline is used simultaneously with thyroid-replacement medications such as levothyroxine, and maprotiline blocks the pharmacological effect of guanethidine.

An increased risk of seizures has been reported with the simultaneous use of physostigmine and maprotiline. A similar effect is observed when maprotiline is taken simultaneously with phenothiazine compounds.

See also Anxiety and anxiety disorders; Bipolar disorder; Bipolar disorders; Depression and depressive disorders

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: 703-836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

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Marijuana *see* **Cannabis and related disorders**

Marital and family therapists

Definition

A marriage and family therapist is a person who has received advanced, specialized training and has practiced therapy for an extended period, typically a minimum of 3,000 hours, under the close supervision of a competent, licensed professional. A marital and family counselor must be licensed by passing both written and oral examinations as well as completing continuing education requirements. Licenses to practice are issued by individual states.

Description

A marital and family counselor concentrates on these two aspects of human behavior. While individuals may seek and receive individual counseling, complete families or marital pairs are more commonly seen together during counseling sessions.

Different theoretical models exist for marital and **family therapy**. However, these share a common thread of concentrating on interactions between and among members of a dysfunctional unit.

The goal of marital and family therapists is to improve relationships between marital partners or family members, or to help with the dissolution of a difficult relationship with minimum harm to all. Various techniques are employed. These include active listening, role-playing, **behavior modification**, and changing expectations concerning the behaviors of others. Persons receiving therapy are helped to understand the motivations and actions of others. They are taught techniques to modify their own behaviors, or how to accept more readily the behaviors of others.

Success in marital and family counseling requires patience, time, and a commitment to succeed. As dysfunctional behaviors are acquired over long periods of time, long periods are required to first unlearn troublesome habits and then replace them with more appropriate patterns of behavior. Patience and understanding facilitate this process. A commitment to succeed is mandatory for success. Therapists must be able to identify persons who enter therapy without a commitment to succeed.

Individual states regulate the activities in which marriage and family therapists may legally engage. This is done to protect consumers from incompetence and negligence of service providers who may potentially exploit them. Most state regulations closely delineate the minimum training and education requirements for marital and family therapists. Thus, the possession of a license to practice marital and family therapy certifies minimum competency and ensures that consumers receive safe and fair treatment. As of 2002, there are 42 states that license practitioners of marital and family therapy.

Marital and family therapists receive training in the following three areas to qualify for a license to practice the profession.

- Academic program. A person must earn a master's degree with an emphasis in marital and family therapy from an accredited academic institution. Most programs of study are 48 semester credit hours in length. The curriculum must include theoretical as well as practical training. Specific areas of competency such as human sexuality, assessing victims of child **abuse** and substance abuse must be embedded in the curriculum. Students must receive 30 hours of directly supervised counseling and an additional 150 hours of directed counseling practice.
- Supervised clinical experience. Prior to becoming eligible to sit for a licensure exam, candidates must complete a total of approximately 3,000 hours of supervised counseling experiences. The 3,000 hours may include activities related to personal **psychotherapy**, supervision, direct counseling experience, professional enrichment experiences, and maintaining records. Some (approximately one-quarter) of these hours may be included in the graduate degree training curriculum. All of the clinical experiences are closely supervised.
- Licensure examination. The examination has written and oral components. A license to practice is granted with the successful passage of both parts of the exam. A minimum of 36 hours of continuing education training must be completed every two years as a requirement for re-licensure.

See also Psychotherapy; Behavior modification; Play therapy

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American Family Therapy Academy. 2020 Pennsylvania Avenue, NW, #273, Washington, DC 20006. Telephone: (202) 333-3690. Fax: (202) 333-3692. Web site: <www.afta.org>.

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Marital therapy *see* **Couples therapy**

Masochism *see* **Sexual masochism**

Massage *see* **Bodywork therapies**

Mathematics disorder

Definition

Mathematics disorder, formerly called developmental arithmetic disorder, developmental acalculia, or dyscalculia, is a learning disorder in which a person's mathematical ability is substantially below the level normally expected based on his or her age, intelligence, life experiences, educational background, and physical impairments. This disability affects the ability to do calculations as well as the ability to understand word problems and mathematical concepts.

Description

Mathematics disorder was first described as a developmental disorder in 1937. Since then, it has come to encompass a number of distinct types of mathematical deficiencies. These include:

- difficulty reading and writing numbers
- difficulty aligning numbers in order to do calculations
- inability to perform calculations
- inability to comprehend word problems

The range and number of mathematical difficulties that have been documented suggests that there are several different causes for mathematics disorder. In addition, several known physical conditions cause mathematics disorder. Turner syndrome and fragile X syndrome, both genetic disorders that affect girls, are associated with difficulty in mathematics. Injury to certain parts of the **brain** can also cause inability to perform calculations. These conditions appear to be independent of other causes of mathematics disorder. Mathematics disorder is often associated with other **learning disorders** involving reading and language, although it may also exist independently in children whose reading and language skills are average or above average.

Causes and symptoms

The causes of mathematics disorder are not understood. Different manifestations of the disorder may have different causes. Symptoms of the disorder, however, can be grouped into four categories: language symptoms; recognition or perceptual symptoms; mathematical symptoms; and attention symptoms.

People with language symptoms have trouble naming mathematical terms; understanding word problems; or understanding such mathematical concepts as "greater than" or "less than." People with recognition symptoms have difficulty reading numbers and such operational signs as the plus or minus signs, or aligning numbers properly in order to perform accurate calculations. Mathematical symptoms include deficiencies in the ability to count; to memorize such basic arithmetical data as the multiplication tables; or to follow a sequence of steps in problem solving. Attention symptoms are related to failures in copying numbers and ignoring operational signs. Sometimes these failures are the result of a person's carelessness. At other times, however, they appear to result from a lack of understanding of the factors or operations involved in solving the problem.

KEY TERMS

Individual education plan (IEP)—A plan of instruction drawn up for an individual student who is having specific difficulties with mathematics, reading, or other skills necessary to progress beyond elementary school.

In practical terms, parents and teachers may see the following signs of mathematics disorder in a child's schoolwork:

- problems counting
- difficulty memorizing multiplication tables
- inability to grasp the difference between such operations as addition and subtraction
- poor computational skills; many errors in simple arithmetic
- slowness in performing calculations
- difficulty arranging numbers in order (from smallest to largest, for example)
- inability to grasp information on graphs
- difficulty copying numbers or problems
- inability to grasp the concept of place value
- inability to align two or three digit numbers to do calculations
- difficulty understanding word problems
- inability to understand mathematical symbols

These symptoms must be evaluated in light of the person's age, intelligence, educational experience, exposure to mathematics learning activities, and general cultural and life experience. The person's mathematical ability must fall substantially below the level of others with similar characteristics. In most cases, several of these symptoms are present simultaneously.

Demographics

The number of children with mathematics disorder is not entirely clear. The ***Diagnostic and Statistical Manual of Mental Disorders***, which is the basic manual consulted by mental health professionals in assessing the presence of mental disorders, indicates that about 1% of school age children have mathematics disorder. Other studies, however, have found higher rates of arithmetical dysfunction in children. Likewise, some studies find no gender difference in the prevalence of mathematics disorder, while others find that girls are more likely to be affected. Mathematics disorder, like other learning disabilities, however, appears to run in families, suggesting the existence of a genetic component to the disorder.

Diagnosis

Mathematics disorder is not usually diagnosed before a child is in the second or third grade because of the variability with which children acquire mathematical fluency. Many bright children manage to get through to fourth- or fifth-grade level in mathematics by using memorization and calculation tricks (such as counting on fingers or performing repeated addition as a substitute for multiplication) before their disability becomes apparent. Requests for testing usually originate with a teacher or parent who has observed several symptoms of the disorder.

To receive a **diagnosis** of mathematics disorder according to the criteria established by the American Psychiatric Association, a child must show substantially lower than expected ability in mathematics based on his or her age, intelligence, and background. In addition, the child's deficiencies must cause significant interference with academic progress or daily living skills.

In addition to an interview with a child **psychiatrist** or other mental health professional, the child's mathematical ability may be evaluated with such individually administered diagnostic tests as the Enright Diagnostic Test of Mathematics, or with curriculum-based assessments. If the results of testing suggest mathematics disorder, such other causes of difficulty as poor vision or hearing, **mental retardation**, or lack of fluency in the language of instruction, are ruled out. The child's educational history and exposure to opportunities for learning mathematics are also taken into account. On the basis of this information, a qualified examiner can make the diagnosis of mathematics disorder.

Treatments

Children who receive a diagnosis of mathematics disorder are eligible for an individual education plan (IEP) that details specific accommodations to learning. Because of the wide variety of problems found under the diagnosis of mathematics disorder, plans vary considerably. Generally, instruction emphasizes basic mathematical concepts, while teaching children problem-solving skills and ways to eliminate distractions and extraneous information. Concrete, hands-on instruction is more successful than abstract or theoretical instruction. IEPs also address other language or reading disabilities that affect a child's ability to learn mathematics.

Prognosis

Progress in overcoming mathematics disorder depends on the specific type of difficulties that the child has with mathematics, the learning resources available, and the child's determination to work on overcoming the disorder. Some children work through their disability, while others continue to have trouble with mathematics throughout life. Children who continue to suffer from mathematics disorder may develop low self-esteem and social problems related to their lack of academic achievement. Later in life they may be more likely to drop out of school and find themselves shut out of jobs or occupations that require the ability to perform basic mathematical calculations.

Prevention

There is no known way to prevent mathematics disorder.

See also Reading disorder; Disorder of written expression

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National Center for Learning Disabilities. 381 Park Avenue South, Suite 1401, New York, NY 10016. (888) 575-7373 (toll-free) or (212) 545-7510. <www.ncld.org>.

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Medication-induced movement disorders

Definition

Medication-induced movement disorder occurs due to treatment with antipsychotic medications. Most medication-induced movement disorders are caused by medications that block the action of dopamine, a neurotransmitter that allows communication between two neurons to take place and that is necessary for coordination of movements of different parts of the body. When the receptor where dopamine is supposed to bind is blocked, certain movement-related side effects occur. All of the medications that block dopamine receptors are called neuroleptics.

Neuroleptics include both conventional or typical antipsychotic agents, such as **chlorpromazine** (Thorazine), **haloperidol** (Haldol), and **fluphenazine** (Prolixin), as well as the newer, or atypical, antipsychotic agents such as **clozapine** (Clozaril), **risperidone** (Risperdal), **olanzapine** (Zyprexa), and **quetiapine** (Seroquel). In general, the newer, atypical antipsychotics appear to have a lower likelihood to cause movement disorders than the older, typical medications. Other neuroleptics include certain drugs used in the treatment of physical symptoms such as nausea, and include prochlorperazine, promethazine, and metoclopramide, as well as **amoxapine** (Asendin), which is marketed as an antidepressant.

There are other medications, however, that do not block dopamine action but still cause movement disorders. They are not referred to as neuroleptics, and they include **lithium carbonate**, **valproic acid** and a class of drugs called selective serotonin reuptake inhibitors (SSRIs). The disorder caused by these medications is called medication-induced postural tremor.

All of the disorders caused by neuroleptics, which include antipsychotics and other medications that block dopamine, as well as disorders caused by non-neuroleptic medications, are collectively referred to as medication-induced movement disorders.

Description

Neuroleptics

Medication-induced movement disorders caused by neuroleptics are divided into three time periods. The early-onset type, which usually occurs within the first seven days of treatment with neuroleptics, is known as neuroleptic-induced acute dystonia. Neuroleptic-induced acute dystonia is characterized by abnormal contractions of various muscle groups resulting in spasm and/or twisting of the head, neck, jaw, lips, tongue, and eye muscles as well as abnormal movements and postures of the limbs and the trunk.

The intermediate-onset types of movement disorders associated with the use of neuroleptics usually develop within the first three months of treatment. They are known as neuroleptic-induced Parkinsonism and neuroleptic-induced akathisia. Neuroleptic-induced Parkinsonism is associated with difficulty initiating movements. Once movements are initiated, they are very slow. Other characteristics of neuroleptic-induced Parkinsonism are tremor and rigidity in muscles. Neuroleptic-induced akathisia is associated with uncontrollable restlessness that may involve compulsive foot tapping, pacing, and a sense of inner tension.

The late-onset type of neuroleptic-related movement disorder is known as neuroleptic-induced **tardive dyskinesia** and the onset is usually seen many months to years after starting the neuroleptic treatment. Neuroleptic-induced tardive dyskinesia involves grotesque, repetitive, and involuntary movements. They are usually seen in the mouth and face.

A movement disorder that can occur at any time during the course of neuroleptic treatment is known as neuroleptic malignant syndrome. It is a serious condition and is characterized by changes in consciousness, ranging from agitation to coma. The patient may experience high fever, and increases in blood pressure and heart rate, as well as severe muscular rigidity.

Non-neuroleptics

All of the movement disorders mentioned above are related to the use of neuroleptic medications. However, other drugs, such as lithium, valproic acid, isoproterenol, amphetamine, theophylline, as well as a class of drugs known as tricyclic antidepressants, may also cause a movement disorder that is mainly characterized by postural tremor, a rhythmic alteration in movement. Lithium-induced tremor may take the form of twitching in the arms and legs.

KEY TERMS

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

Hyperthyroidism—Condition resulting from the thyroid glands secreting excessive thyroid hormone, causing increased basal metabolic rate, and causing an increased need for food to meet the demand of the metabolic activity; generally, however, weight loss results.

Neuroleptic—Another name for antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

Neuroleptic malignant syndrome—An unusual but potentially serious complication that develops in some patients who have been treated with antipsychotic medications. NMS is characterized by changes in blood pressure, altered states of consciousness, rigid muscles, and fever. Untreated NMS can result in coma and death.

Neuroleptic-induced acute dystonia—A severe form of the neurological movement disorder caused by the use of neuroleptic drugs.

Neuroleptic-induced akathisia—Refers to the disorder characterized by a physical restlessness (the inability to sit still, for example), and manifested by excessive voluntary movements, as a result of the use of neuroleptic drugs; research indicates it is likely the most common of neuroleptic-induced movement disorders.

Neuroleptic-induced Parkinsonism—Symptoms similar to Parkinson's disease that may appear in people taking neuroleptic (antipsychotic) medications. These symptoms include tremors in muscles and a shuffling gait.

Neuroleptic-induced tardive dyskinesia—A potentially irreversible neurological disorder caused by the use of antipsychotic/neuroleptic medications, with symptoms involving uncontrollable movement of various body parts.

Postural tremor—A continuous quiver that affects body posture and movement.

SLE (Systemic Lupus Erythematosus)—An autoimmune disease that leads to inflammation and damage to various body tissues and parts, including joints, skin, kidneys, heart, lungs, blood vessels, and brain.

Sydenham's chorea—A serious manifestation of acute rheumatic fever that commonly occurs in children ages seven through 14, peaking at age eight. This disease of the central nervous system is characterized by emotional instability, purposeless movements, and muscular weakness. At its peak in the 1950s it occured in nearly 50% of the acute rheumatic fever cases, but by 2002 had subsided to a degree of less than 10% of the acute cases.

Causes and symptoms

Causes

Neuroleptic-induced movement disorders are caused because the actions of dopamine are blocked. Dopamine is a neurotransmitter necessary for coordination of movements of different parts of the body.

Other medications, which are not classified as neuroleptics, block the action of other **neurotransmitters** as well as dopamine. However, because they essentially block the action of dopamine, they cause similar unwanted effects associated with movements.

Symptoms

Neuroleptic-induced acute dystonia is associated with primarily abnormal postures and muscular spasms. They are usually characterized by abnormal positioning of the head and neck in relation to the body, spasms of the jaw muscles, impaired swallowing, speaking or breathing, thickened or slurred speech due to a slow movement of the tongue, tongue protrusion or tongue dysfunction, eyes deviated up, down, or sideways, and abnormal positioning of the limbs or trunk. Patients experience pain and cramps in the affected muscles. In addition, many patients experiencing dystonia due to the neuroleptic treatment also experience fear and anxiety. This is especially present in patients who are not aware of the possibility of developing dystonia and who mistakenly associate these side effects as part of their mental illness.

Neuroleptic-induced Parkinsonism includes rigidity, tremor, and bradykinesia (slow movements). The tremor is a rhythmic, three- to six-cycle-per-second motion that is present at rest. The tremor can affect the limbs, head, mouth, or lips. Rigidity signifies the degree of tension present in the muscle. It can be either continuous or inter-

mittent in the affected limbs or joints. Bradykinesia includes decreased arm movements related to walking, as well as difficulty initiating movement. Drooling may occur due to a decrease in pharyngeal motor activity. People experiencing neuroleptic-induced akathisia usually feel anxious, agitated, and unable to relax. They also may pace, rock while sitting and standing, and often rapidly alternate between sitting and standing.

Neuroleptic-induced tardive dyskinesia manifests itself in involuntary movements of the tongue, jaw, trunk, or extremities. It occurs most commonly in patients who have taken older antipsychotic medications for many years, although the condition may appear earlier than that (after one year of treatment with neuroleptics, or even earlier than that, especially in elderly people). The movements can be rapid and jerky, slow and continual, or rhythmic in nature. Over three-fourths of the individuals with neuroleptic-induced tardive dyskinesia have abnormal movements of the face and the mouth. This may include licking, sucking or smacking of the lips, chewing movements, jaw deviations, grimacing, grunting and other peculiar sounds, or brow furrowing. About one-half of patients with tardive dyskinesia have abnormal limb movements, while about one-quarter have disposition of the trunk.

The basic features of neuroleptic malignant syndrome is the development of high fever and severe muscle rigidity. These can be accompanied by tremor, changes in level of consciousness ranging from confusion to coma, increased heart rate and blood pressure. The fever can be mildly elevated (99–100°F) or severe (106°F). Neuroleptic malignant syndrome can be fatal in some cases, while it is relatively benign in others. There are no known predictors of neuroleptic malignant syndrome. However, it usually develops four weeks after starting neuroleptics, and about two-thirds of cases develop within the first week of treatment. A very small number of patients develop neuroleptic malignant syndrome many months after taking the neuroleptic.

Medication-induced postural tremor is characterized by a regular, rhythmic oscillation of hands and fingers, head, mouth, or tongue. The frequency of the tremor ranges from eight to 12 cycles per second. These are most easily observed when the affected part is in a sustained position (for example if hands are outstretched or the mouth is held open).

Demographics

Neuroleptic-induced acute dystonia occurs most commonly in young males. It is far less likely to occur with the newer medications known as atypical neuroleptic medications, such as clozapine, risperidone, olanzapine, and quetiapine. The possibility of neuroleptic-induced acute dystonia occurring with these atypical medications is less than 5%. The possibility of this side effect occurring with the conventional or typical neuroleptics is about 15-20%. The incidence is inversely correlated with age, meaning that younger persons are more likely to experience dystonia.

Neuroleptic-induced Parkinsonism is directly correlated with age. This means that older patients are more likely to experience this effect. It occurs in about 30% of patients. Neuroleptic-induced acute akathisia is not related to age and occurs in about 20% of patients being treated with neuroleptics.

The incidence of neuroleptic-induced tardive dyskinesia is related to total lifetime of treatment with antipsychotics. The cumulative incidence is about 5% per year of therapy. This essentially means that there is a 50% chance of developing tardive dyskinesia with 10 years of treatment with neuroleptics.

The incidence of neuroleptic malignant syndrome is about 0.5%. This condition is fatal in about 20 to 30% of cases.

Most available information on medication-induced postural tremor is about lithium-induced tremor. The prevalence of this condition is about 40%.

Diagnosis

People taking antipsychotic medications and other medications that block dopamine action must be regularly evaluated by a physician to monitor for medication-induced movement disorders. In order for these conditions to be officially diagnosed, certain criteria must be met.

Neuroleptic-induced acute dystonia must have one or more of the following developed in association with the use of neuroleptic: abnormal positioning of the head and neck in relation to the body, spasms of the jaw muscles, impaired swallowing, thickened or slurred speech, tongue protrusion or dysfunction, eyes deviated up, down, or sideways, or abnormal positioning of limbs or trunk. These symptoms need to have developed within seven days of starting the neuroleptic medication. Moreover, the symptoms cannot be associated with an underlying mental disorder, and they can't be due to a medication other than a neuroleptic. Dystonia due to neuroleptics needs to be distinguished from dystonia due to neuroleptic malignant syndrome.

Neuroleptic-induced Parkinsonism needs to have the triad of symptoms described above which include tremor, rigidity, and bradykinesia (slow movements). These symptoms cannot be related to a non-neuroleptic medication, or a psychiatric condition, such as Parkinson's dis-

ease, Wilson's disease, neuroleptic malignant syndrome, or substance withdrawal. Neuroleptic-induced akathisia is due to the use of a neuroleptic and not to anxiety, substance withdrawal or psychotic agitation. At least one of the symptoms of fidgety movements or swinging the legs, rocking from foot to foot while standing, pacing to relieve restlessness, or inability to sit and stand needs to be present. These symptoms must have developed within four weeks of initiating the therapy with neuroleptics.

Neuroleptic-induced tardive dyskinesia needs to include involuntary movements over a period of at least four weeks that manifest themselves as rapid and jerky, slow and continual, or rhythmic movements. The exposure to neuroleptics needs to be for at least three months, and the symptoms cannot be due to a neurologic condition, such as Huntington's disease, Wilson's disease, Sydenham's (rheumatic) chorea, systemic lupus, or hyperthyroidism.

Neuroleptic malignant syndrome must include severe muscle rigidity and elevated temperature as well as at least two of the following symptoms: sweating, difficulty swallowing, tremor, incontinence, changes in level of consciousness, mutism, increased heart rate, elevated blood pressure, or laboratory evidence of muscle injury. These symptoms cannot be due to another substance or a medical condition, such as viral encephalitis, or mood disorder with catatonic features.

The criteria for diagnosing medication-induced postural tremor includes a development of tremor associated with the use of a medication other than a neuroleptic. The tremor cannot be due to a non-medication condition that was present prior to starting the medication and cannot continue to be present following discontinuation of the medication. These criteria are helpful in distinguishing the tremor due to medication use from the tremor due to anxiety, alcohol withdrawal, **stress**, or **fatigue**. The tremor must have a frequency between eight and 12 cycles per second, and the tremor must not be caused by neuroleptic-induced Parkinsonism.

Treatments

In an attempt to prevent acute dystonia from developing, physicians may prescribe a preventative medication along with the antipsychotic (see "Prevention," below). Once neuroleptic-induced acute dystonia has appeared, however, there are several treatment options. A medication called **benztropine** in doses ranging from 1 mg to 8 mg is effective in reducing symptoms associated with dystonia. Most patients take 2 mg twice daily for seven days for prevention of dystonia at the time they are starting neuroleptic treatment. When benztropine therapy is initiated, the dose is slowly increased. Moreover, when discontinuing the treatment with benztropine, the dose should be slowly decreased to prevent the nausea and vomiting associated with abrupt withdrawal. Another medication that may be useful in treating neuroleptic-induced acute dystonia is called **trihexyphenidyl**. The doses can vary from 10 mg to 45 mg daily. Younger patients may respond better to the treatment with trihexyphenidyl because they can tolerate higher doses. The third pharmacological option is **diphenhydramine** (Benadryl). This medication can be taken for the period dystonic symptoms last. Another option may include switching the patient to one of the newer antipsychotics, such as clozapine, risperidone, or olanzapine, since each of these has a low incidence of causing dystonia.

There are a couple of ways to treat intermediate-onset movement disorders due to neuroleptics. **Amantadine** is a medication that is approved by the United States Food and Drug Administration for the treatment of Parkinsonian symptoms. Another helpful medication called **propranolol** comes from a class of drugs called **beta blockers**. Propranolol has been reported effective in the treatment of akathisia. The doses that are effective range from 20 mg to 100 mg daily. The response to propranolol is usually seen within the 24 hours of administration. Switching the patient to a newer or atypical antipsychotic, such as clozapine, or decreasing the dose of the current antipsychotic sometimes helps the condition.

There are no effective treatments for tardive dyskinesia once it develops. Tardive dyskinesia is associated and strongly correlated with the cumulative dose of the antipsychotic during years of treatment. Hence, the key to tardive dyskinesia is prevention. If possible, a newer medication, such as clozapine or risperidone, which have only a few case reports of tardive dyskinesia, should be used whenever possible. In many cases, if tardive dyskinesia is noticed early in a regular check-up with the physician, and if the medication causing the condition is stopped, the symptoms of tardive dyskinesia will subside. If the symptoms continue after the antipsychotic has been discontinued, the situation becomes difficult. Treatment will most likely involve movement disorder specialists and may or may not be successful. The medications reserpine and levodopa may be helpful for some patients.

The most common medications used to treat neuroleptic malignant syndrome are dantrolene, (a muscle relaxant that helps with the fever), bromocriptine, and amantadine.

In order to reduce medication-induced postural tremor, the lowest possible dose of the psychiatric drug should be used. Moreover, a medication from the beta

blockers, such as propranolol, can be used to help with the symptoms.

Prognosis

The prognoses for the early- and intermediate-onset of movement disorders are very good, especially with the option of switching the patient to a newer antipsychotic such as clozapine.

The prognosis for the late-onset disorder called tardive dyskinesia is very poor. Once the condition occurs, it is essentially irreversible and is very difficult to treat.

Neuroleptic malignant syndrome is a serious condition. It is deadly in about 20 to 30% of patients. Those who survive have a good chance of recovering.

Medication-induced postural tremor is very well-controlled with propranolol, and hence the prognosis is good while the patient is being treated with the medication causing the movement disorder.

Prevention

To prevent acute dystonia, some physicians prescribe benztropine, diphenhydramine, or other medications that treat dystonia, at the outset of treatment with an older antipsychotic.

The most important component of neuroleptic-induced tardive dyskinesia is prevention. If conventional antipsychotics are used, the drug use, drug dose, and the duration of use therefore should be minimized.

In order to avoid medication-induced postural tremor, patients should limit the amount of caffeine consumption. Also, in order to minimize the amount of daytime tremor they should take the psychiatric drug at bedtime.

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American Thyroid Association. 6066 Leesburg Pike, Suite 650, Falls Church, VA 22041. <http://www.thyroid.org>.

Canadian Movement Disorder Group, affiliate of Canadian Congress of Neurological Sciences. 709 7015 Macleod Trail SW, Calgary, AB T2H 2K6 Canada. <http://www.cmdg.org>.

We Move, worldwide education and awareness of movement disorders. 204 West 84th St., New York, NY 10024. <http://www.wemove.org>.

Ajna Hamidovic, Pharm.D.

Meditation

Definition

Meditation or contemplation involves focusing the mind upon a sound, phrase, prayer, object, visualized image, the breath, ritualized movements, or consciousness in order to increase awareness of the present moment, promote relaxation, reduce **stress**, and enhance personal or spiritual growth.

Purpose

Meditation can benefit people who are ill or overwhelmed by stress. It also promotes well-being in healthy people. In general, people who meditate regularly experience less anxiety and depression. They also report more enjoyment and appreciation of life, as well as better social relationships. Meditation produces a state of deep relaxation and a sense of balance, or equanimity. According to Michael J. Baime in *Essentials of Complementary and Alternative Medicine*, meditation allows one to fully experience intense emotions without losing composure. The consequence of emotional balance is greater insight regarding one's thoughts, feelings, and actions. Insight, in turn, promotes confidence and awareness. Meditation also facilitates a greater sense of calmness, empathy, and acceptance of self and others.

Meditation is sometimes suggested as a complement to medical treatments of disease; in particular, it is an important complementary therapy for both the treatment and prevention of many stress-related conditions. Regular meditation may reduce the number of symptoms

KEY TERMS

Anxiety—A feeling of apprehension and fear characterized by physical symptoms (heart palpitations, sweating, and feelings of stress, for example).

Anxiety disorders—Chronic conditions that can be characterized by an excessive and regular sense of apprehension, with physical symptoms such as sweating, palpitations, and feelings of stress. Anxiety disorders can be caused by biological and environmental events.

Anxiety-reduction techniques—Skills taught by a therapist to help an individual overcome anxiety, stress, and tension, and can include relaxation, visualization and imagery, diaphragmatic breathing, stress inoculation, and meditation.

Biofeedback—Biofeedback is a technique that uses monitoring instruments to measure and feed back information about muscle tension, heart rate, sweat responses, skin temperature, or brain activity.

Bodywork—Any technique involving hands-on massage or manipulation of the body.

Dervish—A person who belongs to one of the various mystical and ascetic Muslim orders, such as the Sufis. A whirling dervish meditates by whirling or spinning an ecstatic dance.

Hypnotherapy—The use of an induced trance state, or hypnosis, as a therapy.

Mantra—Originally, a sacred word or phrase repeated over and over to help focus the mind during meditation; in the Western world, this may refer to any repeated syllable, word, or phrase used to meditate.

Pain disorder—One of several somatoform disorders described in the revised, fourth edition of the mental health professional's handbook, the *Diagnostic and Statistical Manual of Mental Disorders*. The term "somatoform" means that symptoms are physical but are not entirely understood as a consequence of a general medical condition or as a direct effect of a substance, such as a drug.

Progressive relaxation—A technique for managing stress in which the person relaxes major muscle groups in a fixed sequence, often beginning with the feet and moving towards the head.

Transcendental meditation (TM)—A meditation technique based on Hindu practices that involves the repetition of a mantra.

Yoga—A system of exercises for achieving bodily or mental control and well-being.

experienced by patients with a wide range of illnesses and disorders. Based upon clinical evidence, as well as theory, meditation is seen as an appropriate therapy for **panic disorder**, **generalized anxiety disorder**, substance dependence and abuse, ulcers, colitis, chronic pain, psoriasis, and dysthymic disorder—a disorder that involves a steady, depressed mood for at least two years. Moreover, meditation is a valuable adjunct therapy for moderate hypertension (high blood pressure), prevention of cardiac arrest (heart attack), prevention of atherosclerosis (hardening of the arteries), arthritis (including fibromyalgia), cancer, **insomnia**, migraine, and **stroke**. It is a complementary therapy for moderating allergies and asthma because it reduces stress, which is prevalent in these conditions. Additionally, meditation may improve function or reduce symptoms of patients with neurologic disorders such as Parkinson's disease, multiple sclerosis, and epilepsy.

In 1995, the authors of a report to the National Institutes of Health on complementary or alternative medicine reviewed 30 years of research and reports of individuals and health care providers. They concluded that meditation and related methods for the enhancement of relaxation are cost-effective ways to improve health and quality of life.

Precautions

Meditation appears to be safe for most people. There are, however, case reports and studies noting some adverse effects. For example, 33% to 50% of people who participated in long, silent meditation retreats (two weeks to three months) reported increased tension, anxiety, confusion, and depression. On the other hand, they also reported that meditation was associated with very positive effects. It has been noted, however, that these studies failed to differentiate between serious psychiatric disturbances and normal mood swings. Nevertheless, the evidence suggests that meditation may not be appropriate for people with psychotic disorders, major depression, or severe **personality disorders**. Some researchers point out that the relaxed, trance-like state that characterizes deep meditation is similar to a hypnotic trance. Hence, meditation, as well as hypnosis, may be contraindicated

for people who have difficulty giving up control, such as people who are obsessive and compulsive.

Description

Background

Meditation has been practiced for millennia. Historically, meditation or contemplation was intended to develop spiritual understanding, awareness, or gratitude. It also was meant to help the person commune with God, or ultimate reality. The many different religious traditions in the world have given rise to a rich variety of meditative practices. These include the contemplative prayers and chants of Christian religious orders, the Buddhist practice of sitting meditation, and the whirling movements of the Sufi dervishes. Although meditation is an important spiritual practice in many traditions, it can be practiced by anyone to relieve stress and pain regardless of religious or cultural background.

In recent decades, a holistic approach to medicine has become increasingly popular. This approach developed in response to the ideas that health care providers treat whole persons, and that wellness and illness are better understood in terms of the body, mind, and soul. Some refer to this type of medicine as integrative, (that is, the Western biologic model of disease) and notions of appropriate treatment are modified by knowledge garnered from other cultures—especially those of China and India. When foreign ideas are tested in the U.S. both clinically and scientifically, if found to be valid, they are integrated into Western medicine.

With the increasing acceptance of holistic medicine, there has been more interest in the use of alternative or complementary therapies, such as meditation, hypnosis, and progressive relaxation. As a result, training in meditation and meditation sessions are offered in medical clinics and hospitals. Meditation has been used as primary therapy for treating certain diseases and as complementary therapy in a comprehensive treatment plan. Moreover, it has been employed as a means of improving the quality of life of people with debilitating, chronic, or terminal diseases.

When people are dying, they often cope with enduring pain, anxiety and fear, and end-of-life spiritual concerns. Meditation can be a way for the patient with terminal illness to self-manage pain and anxiety. This can partially reduce the amount of drugs required for effective pain control. People who are dying sometimes reject narcotics in an effort to preserve their consciousness and their communication with people who are important to them. Meditation is a means of preserving consciousness and life as the dying patient knows it. Also, meditation

This woman is sitting in the lotus position, practicing yoga. *(Duomo/Corbis. Reproduced by permission.)*

can be tailored to the religious or spiritual needs of the patient, and may be a means to spiritual solace.

In general, there are two main types of meditation: concentration, and mindful meditation. Concentration meditation involves focusing one's attention on the breath, an imagined or real image, ritualized movements (as in Tai chi, **yoga**, or qigong), or on a sound, word, or phrase that is repeated silently or aloud (mantra). In the Christian tradition, chanting and saying the rosary are forms of meditation. (A rosary is a string of beads used to keep track of the prayers recited.) One purpose of concentration meditation is to fully experience the present moment with serenity. The benefit of being fully present is that worries and anxieties fade, and a feeling of peace ensues. It is the feeling of peace that has physiological benefits, and has been referred to as the relaxation response. When thoughts or emotions arise, the person gently directs his or her mind back to the original focus of concentration.

In comparison, mindfulness meditation involves becoming aware of the entire field of attention. There is an awareness of all thoughts, feelings, perceptions or sensations as they arise from moment to moment. Mindfulness meditation is enhanced by the person's ability to quiet the mind and to accept all that is perceived

with composure. Many approaches to meditation are a blend of concentration and mindfulness.

Meditation may involve a quiet, relatively motionless seated posture or it may involve ritualized movement. Sitting meditation is generally done in an upright position, either in a chair or cross-legged on a cushion or mat on the floor. The spine is straight, yet relaxed. The eyes may be closed or open and gazing softly into the distance or at an object. Depending on the tradition, the person may be concentrating on the sensation of the movement of the breath; counting breaths; silently repeating a mantra; chanting a prayer; visualizing a peaceful and meaningful place; focusing awareness on the center of the body; or increasing awareness of all sensory experiences.

Movement meditation may be spontaneous and free form or it may involve highly structured, choreographed, repetitive patterns, as in the practice of Tai chi or qigong. (Tai chi and qigong are ancient Chinese forms of meditation with movement; both are believed to promote health by preserving or restoring the life force, or qi.) Movement meditation is particularly helpful for those people who find it difficult to remain still.

Meditation in health care settings

The use of meditation in health care settings often involves one of the following: transcendental meditation (TM); methods developed by Dr. Herbert Benson to elicit the relaxation response; or adaptations of the program of mindfulness-based stress reduction (MBSR) developed by Jon Kabat-Zinn.

Transcendental meditation (TM) has its origins in the Vedic tradition of India and was introduced to the West by Maharishi Mahesh Yogi. TM has been taught to several million people and is one of the most widely practiced forms of meditation in the West. Much of what is known about the physiology of meditation is based on studies of TM. In transcendental meditation, the person sits with closed eyes and concentrates on a single syllable or word (mantra) for 20 minutes, twice a day. When thoughts or feelings arise, the attention is brought back to the mantra. According to Charles Alexander, a TM researcher, the experience of TM involves a calming of thoughts and ordinary wakefulness, which is transcended and replaced by fully aware consciousness.

Eliciting the relaxation response involves a similar form of mental focusing. Dr. Herbert Benson, one of the first Western doctors to conduct research on the effects of meditation, developed his approach after observing the profound health benefits of a state of bodily calm (the relaxation response). In order to elicit this response, he teaches patients to repeat a word, sound, prayer, phrase, or activity (including swimming, jogging, yoga, or even knitting) for 10 to 20 minutes, twice a day. Patients also are taught not to pay attention to distracting thoughts and to return their focus to the original repetition. What is repeated is up to the individual. For example, instead of Sanskrit terms, the person may choose something personally meaningful, such as a phrase from a Christian or Jewish prayer.

Mindfulness meditation stems from traditional Buddhist meditation practices. **Psychologist** Jon Kabat-Zinn has been instrumental in bringing this form of meditation into medical settings. In formal mindfulness practice, the person sits with eyes closed, focusing the attention on the sensations and movement of the breath for approximately 45 to 60 minutes, at least once a day. Informal mindfulness practice involves bringing awareness to every activity in daily life. Wandering thoughts or distracting feelings are simply noticed, without resistance or reaction. The essence of mindfulness meditation is not that on which the individual is focusing, but rather the quality of dispassionate awareness the person brings to each moment. According to Kabat-Zinn, the purpose of mindfulness meditation is to become aware of one's body and mind in the present moment. Discerning observation differentiates mindfulness from other types of meditation. The MBSR program consists of a series of classes involving meditation, movement, and group participation. There are over 240 MBSR programs offered in health care settings around the world.

Meditation is not considered a medical procedure or **intervention** by most insurers; therefore, if there is a cost associated with training, patients pay for it themselves. Frequently, religious groups or meditation centers offer meditation instruction free of charge or for a nominal donation. Hospitals may offer MBSR classes to their patients for a reduced fee, and to the general public for a somewhat higher fee.

Normal results

The scientific study of the physiological effects of meditation began in the early 1960s. These studies demonstrated that meditation affects metabolism, the endocrine system, the central nervous system, and the autonomic nervous system. In particular, there is a slowing of cardiac and respiratory rates, a decrease in blood pressure, and an increase in alpha **brain** waves. These effects are typical of reduced anxiety.

There is a growing body of evidence supporting the medical benefits of meditation. For example, meditation is particularly effective as a treatment for chronic pain. Researchers have found that meditation reduces symptoms of pain and reliance on drugs used to control pain. For example, in one four-year follow-up study, the

majority of patients in an MBSR program reported improvement in the experience of pain as a result of participation in the program.

For many years, meditation has been recommended as a treatment for high blood pressure; however, there is a debate over the effectiveness of meditation compared with medical treatment. Although most studies show a reduction in blood pressure as a result of meditation, medication is relatively more effective.

Meditation may be an effective treatment for coronary artery disease (CAD). For example, a study of 21 patients practicing TM for eight months increased their tolerance of exercise and their capacity for work. Also, meditation is an important part of Dr. Dean Ornish's program for the prevention or reversal of CAD. His program involves a low-fat vegetarian diet, moderate exercise (for example, walking 30 minutes per day), and techniques for reducing stress, including meditation.

Researchers have found that meditation is effective in the treatment of chemical dependency. Gelderloos and others reviewed 24 studies and concluded that TM is helpful in programs that target smoking behavior and drug and alcohol abuse.

The scientific evidence also suggests that meditation is particularly helpful in treating anxiety-related disorders and in reducing symptoms of anxiety triggered by stress. For example, researchers conducted a study in 1998 of 37 patients with psoriasis—a chronic, stress-related skin condition. They found that patients who practiced mindfulness meditation and who received standard ultraviolet light treatment experienced a more rapid clearing of their skin condition than the control subjects. Another study found that meditation moderated the symptoms of fibromyalgia (a chronic condition where people suffer diffuse muscular pain at several sites on the body); over half of the patients reported significant improvement. Meditation was one of several stress management techniques used in a small study of HIV-positive men. The study showed improvements in immune function and psychological well-being.

In sum, holistic practitioners speak about the body's capacity for healing itself; since meditation leads to a peaceful, relaxed state with measurable physiological benefits. Healing is facilitated presumably by moderating the state of arousal generated by chronic stress. There is a variety of stress-reducing techniques available, such as hypnosis, progressive relaxation, **biofeedback**, guided imagery, and aerobic exercise. Health consumers are encouraged to investigate the various techniques and seek referrals to good physicians, therapists, or stress counselors who are willing to design a flexible program that meets their needs.

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Insight Meditation Society. 1230 Pleasant, St. Barre, MA 01005. (978) 355-4378. <http://www.dharma.org>.

Mind/Body Medical Institute. 110 Francis Street, Boston, MA 02215. (617) 632-9530. <http://www.mbmi.org>.

National Center for Complementary and Alternative Medicine. NCCAM Clearinghouse, P.O. Box 7923, Gaithersburg, MD 20898. (888) 644-6226. <http://www.nccam.nih.gov>.

Linda Chrisman
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Mellaril *see* **Thioridazine**

Mental retardation

Definition

Mental retardation (MR) is a developmental disability that first appears in children under the age of 18. It is defined as a level of intellectual functioning (as measured by standard **intelligence tests**) that is well below average and results in significant limitations in the person's daily living skills (adaptive functioning).

KEY TERMS

Amniocentesis—A test usually done between 16 and 20 weeks of pregnancy to detect any abnormalities in the development of the fetus. A small amount of the fluid surrounding the fetus (amniotic fluid) is drawn out through a needle inserted into the mother's womb. Laboratory analysis of this fluid can detect various genetic defects, such as Down syndrome, or neural tube defects.

Developmental delay—The failure to meet certain developmental milestones, such as sitting, walking, and talking, at the average age. Developmental delay may indicate a problem in development of the central nervous system.

Down syndrome—A genetic disorder characterized by an extra chromosome 21 (trisomy 21), mental retardation, and susceptibility to early-onset Alzheimer's disease.

Extensive support—Ongoing daily support required to assist an individual in a specific adaptive area, such as daily help with preparing meals.

Hib disease—An infection caused by *Haemophilus influenza*, type b (Hib). This disease mainly affects children under the age of five. In that age group, it is the leading cause of bacterial meningitis, pneumonia, joint and bone infections, and throat inflammations.

Inborn error of metabolism—A rare enzyme deficiency; children with inborn errors of metabolism do not have certain enzymes that the body requires to maintain organ functions. Inborn errors of metabolism can cause brain damage and mental retardation if left untreated. Phenylketonuria is an inborn error of metabolism.

Limited support—A predetermined period of assistance required to deal with a specific event, such as training for a new job.

Phenylketonuria—(PKU) An inherited disease in which the body cannot metabolize the amino acid phenylalanine properly. If untreated, phenylketonuria can cause mental retardation.

Trisomy—An abnormality in chromosomal development. Chromosomes are the structures within a cell that carry its genetic information. They are organized in pairs. Humans have 23 pairs of chromosomes. In a trisomy syndrome, an extra chromosome is present so that the individual has three of a particular chromosome instead of the normal pair. An extra chromosome 18 (trisomy 18) causes mental retardation.

Ultrasonography—A process that uses the reflection of high-frequency sound waves to make an image of structures deep within the body. Ultrasonography is routinely used to detect fetal abnormalities.

Description

Mental retardation begins in childhood or adolescence before the age of 18. In most cases, it persists throughout adult life. A **diagnosis** of mental retardation is made if an individual has an intellectual functioning level well below average, as well as significant limitations in two or more adaptive skill areas. Intellectual functioning level is defined by standardized tests that measure the ability to reason in terms of mental age (intelligence quotient or IQ). Mental retardation is defined as an IQ score below 70–75. Adaptive skills is a term that refers to skills needed for daily life. Such skills include the ability to produce and understand language (communication); home-living skills; use of community resources; health, safety, leisure, self-care, and social skills; self-direction; functional academic skills (reading, writing, and arithmetic); and job-related skills.

In general, mentally retarded children reach such developmental milestones as walking and talking much later than children in the general population. Symptoms of mental retardation may appear at birth or later in childhood. The child's age at onset depends on the suspected cause of the disability. Some cases of mild mental retardation are not diagnosed before the child enters preschool or kindergarten. These children typically have difficulties with social, communication, and functional academic skills. Children who have a neurological disorder or illness such as encephalitis or meningitis may suddenly show signs of cognitive impairment and adaptive difficulties.

Mental retardation varies in severity. The ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (*DSM-IV-TR*), which is the diagnostic standard for mental health care professionals in the United States, classifies four different degrees of mental

retardation: *mild, moderate, severe*, and *profound*. These categories are based on the person's level of functioning.

Mild mental retardation

Approximately 85% of the mentally retarded population is in the mildly retarded category. Their IQ score ranges from 50–70, and they can often acquire academic skills up to about the sixth-grade level. They can become fairly self-sufficient and in some cases live independently, with community and social support.

Moderate mental retardation

About 10% of the mentally retarded population is considered moderately retarded. Moderately retarded persons have IQ scores ranging from 35–55. They can carry out work and self-care tasks with moderate supervision. They typically acquire communication skills in childhood and are able to live and function successfully within the community in such supervised environments as **group homes**.

Severe mental retardation

About 3–4% of the mentally retarded population is severely retarded. Severely retarded persons have IQ scores of 20–40. They may master very basic self-care skills and some communication skills. Many severely retarded individuals are able to live in a group home.

Profound mental retardation

Only 1–2% of the mentally retarded population is classified as profoundly retarded. Profoundly retarded individuals have IQ scores under 20–25. They may be able to develop basic self-care and communication skills with appropriate support and training. Their retardation is often caused by an accompanying neurological disorder. Profoundly retarded people need a high level of structure and supervision.

The American Association on Mental Retardation (AAMR) has developed another widely accepted diagnostic classification system for mental retardation. The AAMR classification system focuses on the capabilities of the retarded individual rather than on his or her limitations. The categories describe the level of support required. They are: *intermittent support; limited support; extensive support*, and *pervasive support*. To some extent, the AAMR classification mirrors the *DSM-IV-TR* classification. Intermittent support, for example, is support that is needed only occasionally, perhaps during times of **stress** or crisis for the retarded person. It is the type of support typically required for most mildly retarded people. At the other end of the spectrum, pervasive support, or life-long, daily support for most adaptive areas, would be required for profoundly retarded persons. The AAMR classification system refers to the "below-average intellectual function" as an IQ of 70–75 or below.

Demographics

The prevalence of mental retardation in North America is a subject of heated debate. It is thought to be between 1%–3% depending upon the population, methods of assessment, and criteria of assessment that are used. Many people believe that the actual prevalence is probably closer to 1%, and that the 3% figure is based on misleading mortality rates; cases that are diagnosed in early infancy; and the instability of the diagnosis across the age span. If the 1% figure is accepted, however, it means that 2.5 million mentally retarded people reside in the United States. The three most common causes of mental retardation, accounting for about 30% of cases, are Down syndrome, fragile X, and fetal alcohol syndrome. Males are more likely than females to have MR in a 1.5:1 ratio.

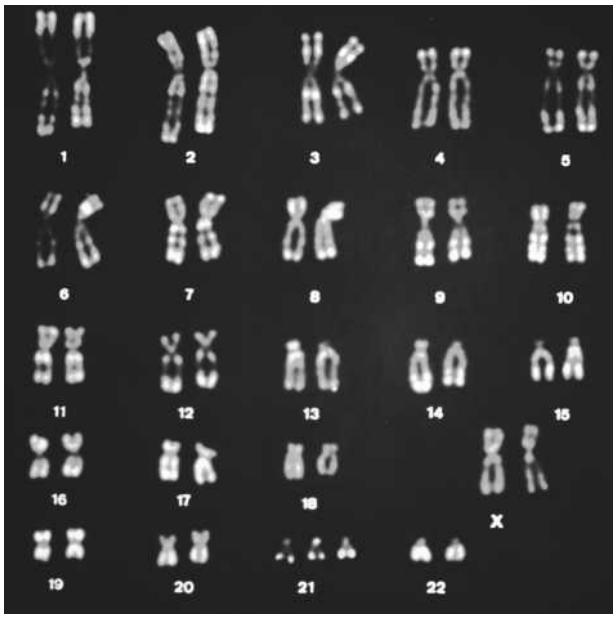
Causes and symptoms

Low IQ scores and limitations in adaptive skills are the hallmarks of mental retardation. Aggression, self-injury, and mood disorders are sometimes associated with the disability. The severity of the symptoms and the age at which they first appear depend on the cause. Children who are mentally retarded reach developmental milestones significantly later than expected, if at all. If retardation is caused by chromosomal or other genetic disorders, it is often apparent from infancy. If retardation is caused by childhood illnesses or injuries, learning and adaptive skills that were once easy may suddenly become difficult or impossible to master.

In about 40% of cases, the cause of mental retardation cannot be found. Biological and environmental factors that can cause mental retardation include:

Genetic factors

About 30% of cases of mental retardation is caused by hereditary factors. Mental retardation may be caused by an inherited genetic abnormality, such as fragile X syndrome. Fragile X, a defect in the chromosome that determines sex, is the most common inherited cause of mental retardation. Single-gene defects such as phenylketonuria (PKU) and other inborn errors of metabolism may also cause mental retardation if they are not discovered and treated early. An accident or mutation in genetic development may also cause retardation. Examples of such accidents are development of an extra



An accident or mutation in genetic development may cause retardation. An example of such a mutation is the development of an extra chromosome 21 that causes Down syndrome. Shown here is a chart (karyotype) showing the 22 chromosome pairs, and in pair 21, three chromosomes (instead of two) are shown. *(Phototake/NYC. Reproduced by permission.) See color insert for color version of photo.*

chromosome 18 (trisomy 18) and Down syndrome. Down syndrome, also called mongolism or trisomy 21, is caused by an abnormality in the development of chromosome 21. It is the most common genetic cause of mental retardation.

Prenatal illnesses and issues

Fetal alcohol syndrome (FAS) affects one in 3,000 children in Western countries. It is caused by the mother's heavy drinking during the first twelve weeks (trimester) of pregnancy. Some studies have shown that even moderate alcohol use during pregnancy may cause learning disabilities in children. Drug abuse and cigarette smoking during pregnancy have also been linked to mental retardation.

Maternal infections and such illnesses as glandular disorders, rubella, toxoplasmosis, and cytomegalovirus (CMV) infection may cause mental retardation. When the mother has high blood pressure (hypertension) or blood poisoning (toxemia), the flow of oxygen to the fetus may be reduced, causing **brain** damage and mental retardation.

Birth defects that cause physical deformities of the head, brain, and central nervous system frequently cause mental retardation. Neural tube defect, for example, is a birth defect in which the neural tube that forms the spinal cord does not close completely. This defect may cause children to develop an accumulation of cerebrospinal fluid inside the skull (hydrocephalus). Hydrocephalus can cause learning impairment by putting pressure on the brain.

Childhood illnesses and injuries

Hyperthyroidism, whooping cough, chickenpox, measles, and Hib disease (a bacterial infection) may cause mental retardation if they are not treated adequately. An infection of the membrane covering the brain (meningitis) or an inflammation of the brain itself (encephalitis) can cause swelling that in turn may cause brain damage and mental retardation. Traumatic brain injury caused by a blow to the head or by violent shaking of the upper body may also cause brain damage and mental retardation in children.

Environmental factors

Ignored or neglected infants who are not provided with the mental and physical stimulation required for normal development may suffer irreversible learning impairment. Children who live in poverty and suffer from malnutrition, unhealthy living conditions, **abuse,** and improper or inadequate medical care are at a higher risk. Exposure to lead or mercury can also cause mental retardation. Many children have developed lead poisoning from eating the flaking lead-based paint often found in older buildings.

Diagnosis

If mental retardation is suspected, a comprehensive physical examination and medical history should be done immediately to discover any organic cause of symptoms. Such conditions as hyperthyroidism and PKU are treatable. If these conditions are discovered early, the progression of retardation can be stopped and, in some cases, partially reversed. If a neurological cause such as brain injury is suspected, the child may be referred to a neurologist or neuropsychologist for testing.

A complete medical, family, social, and educational history is compiled from existing medical and school records (if applicable) and from interviews with parents. Children are given intelligence tests to measure their learning abilities and intellectual functioning. Such tests include the **Stanford-Binet Intelligence Scale**, the Wechsler Intelligence Scales, the Wechsler Preschool and Primary Scale of Intelligence, and the **Kaufman Assessment Battery for Children**. For infants, the Bayley Scales of Infant Development may be used to assess motor, language, and problem-solving skills. Interviews with parents or other caregivers are used to assess the child's daily liv-

ing, muscle control, communication, and social skills. The Woodcock-Johnson Scales of Independent Behavior and the Vineland Adaptive Behavior Scale (VABS) are frequently used to evaluate these skills.

Treatment

Federal legislation entitles mentally retarded children to free testing and appropriate, individualized education and skills training within the school system from ages three to 21. For children under the age of three, many states have established early **intervention** programs that assess children, make recommendations, and begin treatment programs. Many day schools are available to help train retarded children in such basic skills as bathing and feeding themselves. Extracurricular activities and social programs are also important in helping retarded children and adolescents gain self-esteem.

Training in independent living and job skills is often begun in early adulthood. The level of training depends on the degree of retardation. Mildly retarded people can often acquire the skills needed to live independently and hold an outside job. Moderate to profoundly retarded persons usually require supervised community living in a group home or other residential setting.

Family therapy can help relatives of the mentally retarded develop coping skills. It can also help parents deal with feelings of guilt or anger. A supportive, warm home environment is essential to help the mentally retarded reach their full potential.

Prognosis

People with mild to moderate mental retardation are frequently able to achieve some self-sufficiency and to lead happy and fulfilling lives. To reach these goals, they need appropriate and consistent educational, community, social, family, and vocational supports. The outlook is less promising for those with severe to profound retardation. Studies have shown that these persons have a shortened life expectancy. The diseases that are usually associated with severe retardation may cause the shorter life span. People with Down syndrome will develop the brain changes that characterize **Alzheimer's disease** in later life and may develop the clinical symptoms of this disease as well.

Prevention

Immunization against diseases such as measles and Hib prevents many of the illnesses that can cause mental retardation. In addition, all children should undergo routine developmental screening as part of their pediatric care. Screening is particularly critical for those children who may be neglected or undernourished or may live in disease-producing conditions. Newborn screening and immediate treatment for PKU and hyperthyroidism can usually catch these disorders early enough to prevent retardation.

Good prenatal care can also help prevent retardation. Pregnant women should be educated about the risks of alcohol consumption and the need to maintain good nutrition during pregnancy. Such tests as amniocentesis and ultrasonography can determine whether a fetus is developing normally in the womb.

See also Childhood disintegrative disorder; Pica

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The Arc of the United States (formerly Association of Retarded Citizens of the United States). 1010 Wayne Avenue, Silver Spring, M.D. 20910. (301) 565-3842. <http://thearc.org>.

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National Information Center for Children and Youth and Disabilities. P.O. Box 1492,Washington, D.C. 20013. (800) 695-0285. <http://www.nichcy.org>.

Paula Anne Ford-Martin, M.A.

Mesoridazine

Definition

Mesoridazine is a member of the phenothiazine family of drugs (drugs that reduce the action of the neurotransmitter, dopamine, in the **brain**) and sold under the brand name Serentil in the United States.

Purpose

Mesoridazine is effective in the treatment of **schizophrenia**, alcoholism, psychoneuroses (disorders of the brain), and organic brain disorders (disorders caused by temporary brain dysfunction or permanent brain damage).

Description

When used for the treatment of schizophrenia, mesoridazine reduces symptoms of emotional withdrawal, anxiety, tension, **hallucinations**, reduced **affect**, and **paranoia** (suspiciousness). It is often useful in persons for whom other tranquilizers are ineffective. In treating organic brain syndrome, mesoridazine effectively manages hyperactivity and difficult behaviors associated with mental deficiency. Mesoridazine relieves anxiety, nausea, vomiting, tension, and depression when used to treat alcoholism. It does not have side effects that affect liver function. It relieves similar symptoms when used to treat persons with psychoneurotic disorders.

Mesoridazine can be taken by mouth or given by intramuscular injection. It is supplied as 25 mg/mL in injection form, and in 10-, 25-, 50-, and 100-mg tablets.

Recommended dosage

The usual dosage used for treating schizophrenia is 50–400 mg per day and is usually administered three times per day. It is begun at a low level and slowly increased until an adequate therapeutic effect is achieved. For persons with organic brain syndrome, an optimum dosage is 75–300 mg per day, administered in three equal amounts. The optimum dosage for persons being treated for alcoholism is 50–300 mg per day, administered in three doses. The usual dosage range for persons with psychoneuroses is 30–150 mg per day, administered in three equal amounts.

Precautions

Mesoridazine has the potential to produce a serious syndrome called **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements (especially of the tongue, jaw, mouth, or face). It usually develops either late in the course of treatment or after medication has been discontinued and is potentially irreversible. Symptoms similar to those experienced by people with Parkinson's disease have been linked with the

KEY TERMS

Akathisia—Agitated or restless movement, usually affecting the legs. Movement is accompanied by a sense of discomfort and an inability to sit, stand still, or remain inactive for periods of time. Akathisia is a common side effect of some neuroleptic (antipsychotic) medications.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Organic brain syndrome—A class of disorders characterized by progressive deterioration of mental processes caused by temporary brain dysfunction or permanent brain damage. Symptoms include delusions, dementia, amnesia, and delirium that are not caused by drugs, alcohol, or as a side effect of medication.

Psychoneurotic—Pertaining to a neurosis or disorder of the brain. Informally, refers to a person with unstable emotions.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tachycardia—A pulse rate above 100 beats per minute.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

administration of mesoridazine. Mesoridazine is inappropriate for use with central nervous system depression, nor should it be administered to persons in a coma.

Side effects

A serious and relatively common side effect of mesoridazine is tardive dyskinesia, a potentially irreversible syndrome for which there is no known effective treatment. An important feature of tardive dyskinesia is that it typically develops either late into treatment or after treatment has ceased. Tardive dyskinesia consists of involuntary, uncoordinated movements of the tongue, jaw, mouth, or face that also may be accompanied by involuntary movements of the arms, legs, and trunk. The chances of developing tardive dyskinesia increase with both increasing dosage and increasing patient age.

The most common side effects of mesoridazine are drowsiness and low blood pressure and are most frequently reported in persons given relatively high dosages. Side effects also tend to appear relatively early in treatment. Mesoridazine tends to have a remarkably low incidence of side effects compared to other phenothiazine compounds. However, as mentioned, Parkinson-like symptoms have been linked with the administration of mesoridazine. These include restlessness and agitation (akathisia) and difficulty walking or moving (dystonia). These are generally controlled with **benztropine** mesylate or **trihexyphenidyl** hydrochloride.

Other known side effects include anxiety, restlessness, agitation, **insomnia**, headache, euphoria, drowsiness, depression, confusion, and dizziness. Unwanted or unexpected effects associated with the use of mesoridazine have been reported for virtually all organ systems in the body. Although numerous, such side effects are relatively uncommon. An occasionally reported side effect is neuroleptic malignant syndrome, a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia and arrhythmias.

Interactions

Mesoridazine increases the effect of drugs and substances that depress the central nervous system. This class of drugs includes anesthetics, opiates, **barbiturates**, atropine, and alcohol.

See also Alcohol and related disorders

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981 Fax: (703) 836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

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Methadone

Definition

Methadone is classified as an opioid (an analgesic that is used for severe pain). In the United States, methadone is also known as dolophine, methenex and methadose.

Purpose

Methadone is used in the long-term maintenance treatment of narcotic **addiction**. Both heroin and methadone are opioids; as such, methadone and heroin bind to the same places in the **brain**. Methadone, however, is the opioid of choice for the treatment of narcotic addiction since it is longer lasting and patients don't experience the "high" associated with the drug of abuse. In opioid maintenance therapy, a person addicted to heroin receives methadone instead of heroin. Essentially, the person is switched from an opioid that gives a "high" to an opioid that does not. The dose of methadone may then be decreased over time so that the person can overcome his or her opioid addiction without experiencing withdrawal symptoms, or, after a person has received methadone for a period of time, he or she may choose to go through **detoxification** with **clonidine**. In the United States, methadone treatment is associated with a significant reduction in predatory crime, improvement in socially acceptable behavior, and psychological well-being.

Methadone may also be prescribed for pain relief, but in these cases, the physician must note this use on the prescription.

Description

Methadone has been used successfully to treat narcotic addiction for over twenty years in the United States. Methadone is the only FDA-approved agent in its class for the maintenance treatment of narcotic addiction.

Methadone for maintenance treatment is dispensed in methadone clinics. The program needs to be registered with the Drug Enforcement Agency. For admission to methadone treatment in clinical programs, federal standards mandate a minimum of one year of opiate addiction as well as current evidence of addiction. Pregnant, opiate-addicted females can be admitted with less than a one-year history and AIDS patients are routinely accepted. New patients must report daily, take medication under observation, and participate in recommended psychosocial treatments.

Some studies have shown that over 50% of patients in methadone clinics do not abuse drugs in the first month of treatment. After ten months, however, the success rate drops to approximately 20%. Moreover, major depression is a powerful predictor of relapse in methadone treatment. If the patient has dual addictions (alcoholism along with the heroin addiction, for example), management of the other addiction increases the success rate of the methadone therapy. Proper psychiatric and psychological treatment can considerably improve methadone treatment outcome.

In the cases of pregnant women who are addicted to heroin, detoxification (discontinuing the opioid altogether) is associated with a high rate of spontaneous abortions in the first trimester and premature delivery in the third trimester. Therefore, pregnant women can be in methadone maintenance programs if they are at risk of returning to drug dependence. These women should receive the lowest effective dose, receive appropriate prenatal care, and be warned about risks of returning to drug abuse, as well as the dangers associated with withdrawal effects of methadone. Methadone is associated with lower birth weights and smaller head circumference,

KEY TERMS

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Maintenance treatment—The period of treatment beginning after the initial introduction of the treatment medication. During this period, the dose of medication can be either increased or decreased, depending on the program and needs of the patient.

MAO inhibitors—A group of antidepressant drugs that decrease the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Naloxone—A drug that combines competitively with opiate receptors on the nerve cells and blocks or reverses the action of narcotic analgesics.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Relapse—When a person returns to a habit (such as drug use) that he or she was trying to overcome.

but it has never been shown that this has any impact on the infants' further development.

Methadone is available in 5-, 10-, and 40-mg tablets and a solution.

Recommended dosage

The initial dose of methadone is 40 mg daily administered in single or divided doses. After achieving initial dosing of about 40 mg daily, the dose should be increased since there is evidence that the relapse rate is significantly lower in patients on 80-100 mg daily rather than 40-50 mg daily. The stabilization to maintenance dosing requires one to three months.

The minimum effective dose is 60 mg daily taken at once or in divided doses. Patients on lower maintenance doses have recently been studied and have shown shorter treatment retention and have continued heroin use. If patients are stable on methadone for six months or longer, their methadone dose should not be increased by 33% or over, as this sudden increase in dose is associated with an increase in craving for the drugs that were previously abused. Some heroin patients need to be on doses up to 180 mg daily to provide adequate maintenance and to prevent relapse.

Precautions

Methadone should not be used in patients who have had hypersensitivity to methadone. Patients who experience an allergic reaction to other opioids, which may include a generalized rash or shortness of breath, such as morphine, hydromorphone, oxymorphone, or codeine may try methadone. They are less likely to develop the same reaction since methadone has a different chemical structure. Methadone should be administered carefully in patients with pre-existing respiratory problems, history of bowel obstruction, glaucoma, renal problems, and hyperthyroidism.

As stated, pregnant women can be in methadone maintenance programs if they are at risk of returning to drug dependence. Methadone is associated with smaller birth weights and smaller head circumference.

Side effects

Most adverse effects of methadone are mild and seen only in the beginning of therapy. Initially, patients may develop sedation and analgesia. It takes about four to six weeks for tolerance to these effects to develop. Tolerance to constipation and sweating may take longer to develop.

A few patients who are on larger doses of methadone may experience respiratory problems. These patients also may experience unwanted cardiac effects.

A small number of patients report a decrease in libido, impotence, and premature, delayed, or failed ejaculation. There are a few reports of occasional menstrual irregularities in female patients on methadone.

Interactions

Life-threatening interactions with other drugs have not been identified. One of the initial side effects of methadone could include dizziness and sedation, and these effects are worsened if the patient is also taking other narcotics, benzodiazepines, or is consuming alcohol.

Monoamine oxidase inhibitors (MAOIs), such as Parnate (**tranylcypromine**) and Nardil (**phenelzine**), should be avoided by people taking methadone. Medications such as **naltrexone** and naloxone should never be used concurrently with methadone. People must stop taking methadone for seven to 10 days before starting naltrexone or naloxone.

See also Alcohol and related disorders; Disease concept of chemical dependency; Opioids and related disorders

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Ajna Hamidovic, Pharm.D.

Methylphenidate

Definition

Methylphenidate is a mild, central nervous system stimulant. In the United States, the drug is sold under the brand name Ritalin.

KEY TERMS

Anticoagulant—A medication (such as warfarin, Coumadin, or Heparin) that decreases the blood's clotting ability preventing the formation of new clots. Although anticoagulants will not dissolve existing clots, they can stop them from getting larger. These drugs are commonly called blood thinners.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Opiate—A drug derived from opium.

Tachycardia—A pulse rate above 100 beats per minute.

Tourette syndrome—Neurological disorder characterized by multiple involuntary movements and uncontrollable vocalizations called tics that come and go over years, usually beginning in childhood and becoming chronic. Sometimes the tics include inappropriate language.

Purpose

Methylphenidate is used primarily in the treatment of **attention-deficit/hyperactivity disorder** (ADHD) in children and adults. It also may be used to treat the sleep disorder, **narcolepsy**. In rare cases, it is used to decrease sedation and lethargy from opioid pain medications and to help improve the mood of a terminally ill person suffering from depression.

Description

The mode of action for methylphenidate is not fully understood. It presumably activates the **brain** stem arousal system and cortex to produce a stimulant effect. The brain stem arousal system increases levels of electrical activity in the brain. The effect of methylphenidate is to produce increased alertness and, although children with ADHD are overactive and have decreased attention spans, in these children, methylphenidate actually decreases motor restlessness and increases attention span. Tablets are available in 5-, 10-, and 20-mg strengths, as well as in an extended release, 20-mg tablet.

Recommended dosage

The recommended dosage of methylphenidate is determined by trial and error based on individual responses. Methylphenidate is usually administered in two or three separate doses each day, preferably 45 minutes before a meal. For children suffering from ADHD, the initial recommended dosage is 5 mg twice daily before breakfast and lunch, increased by 5–10 mg per week to a maximum of 60 mg per day. The average total dosage is 20–30 mg per day, although 10–60 mg is not uncommon. For narcolepsy in adults, the recommended dose is 5–20 mg two to three times a day, 30–45 minutes before meals.

The drug should be taken exactly as directed. Methylphenidate can become habit forming if taken in greater amounts or for longer periods than necessary. Individuals should take the last dose of the day before 6 P.M. to decrease sleep difficulties. The tablet should not be broken or crushed, as this changes the time for absorption. If the normal time of administration is missed, people should take the drug as soon as possible. However, two tablets should *not* be taken at the same time.

Precautions

Methylphenidate has a great potential to produce physical and mental dependence. Administration should not be stopped abruptly. Such action can cause withdrawal symptoms including depression, paranoid feelings, thoughts of **suicide**, anxiety, agitation, and sleep disturbances. Methylphenidate should not be given to people with extreme anxiety, tension, agitation, severe depression, mental or emotional instability, or a history of alcohol or drug abuse. It is not indicated for use by those with Tourette's syndrome, people with **tic disorders**, glaucoma, or certain mental health conditions. The drug should be used cautiously by those with high blood pressure, those with a history of **seizures**, and women who are breast-feeding. Methylphenidate is not typically ordered for women during their childbearing years, unless the physician determines that the benefits outweigh the risks.

Methylphenidate should not be ordered for children younger than six years of age as its safety has not been determined in this age group. People should not drive or operate machinery or appliances until they understand how this drug affects them. They should not drive if they become lightheaded or dizzy. Methylphenidate may cause irregularities in the composition of the blood and produce changes in liver function. People taking methylphenidate should receive regular blood tests.

Side effects

The most common side effects are nervousness, difficulties with sleep, tachycardia, and increased blood

pressure. Reducing the dose or changing the time the drug is taken may reduce some side effects. Affected persons should discuss any adverse reactions with their health care professional. Individuals taking methylphenidate should receive regular blood pressure and pulse checks. Methylphenidate also may cause dizziness, irritability, vision changes, drowsiness, and a poor appetite. Less common side effects include chest pain, palpitations, joint pain, skin rash, and uncontrolled movements or speech. Side effects may also include a rapid or irregular heartbeat, stomach upset, nausea, headache, blood in the urine or stools, muscle cramps, red dots on the skin, or bruises. At higher dosages or with long-term use, people may experience weight loss or mental changes such as confusion, false beliefs, mood changes, **hallucinations**, or feelings that they or their environment are not real.

Interactions

Several drugs may interact adversely with methylphenidate, including anticoagulants and drugs to prevent seizures, combat depression, and treat high blood pressure. The dosages of these drugs may be reduced when taken simultaneously with methylphenidate.

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American College of Physicians. 190 N Independence Mall West, Philadelphia, PA 19106-1572. Telephone: (800) 523-1546, x2600 or (215) 351-2600. Web site: <http://www.acponline.org>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850.

American Society for Clinical Pharmacology and Therapeutics; 528 North Washington Street, Alexandria, VA 22314; Phone: (703) 836-6981. Fax: (703) 836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060.

National Institute on Drug Abuse: <http://www.nida.nih.gov/Infofax/ritalin.html>.

Nurse's PDR Resource Center. <http://www.nursespdr.com/members/database/>.

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Mini-mental state examination

Definition

The mini-mental state examination, which is also known as the MMSE, standardized MMSE, SMMSE, or the Folstein, is a brief examination consisting of eleven questions intended to evaluate an adult patient's level of cognitive functioning. It was introduced in 1975 and designed for use with elderly patients who are able to cooperate at an optimum level with an examiner for only a brief period of time—no more than a few minutes.

Purpose

The MMSE concentrates on the cognitive aspects of mental functioning, excluding questions about the patient's mood or such abnormal experiences as dissociation. It is used most often to evaluate older adults for **delirium** or **dementia**. The MMSE can be used to detect a decline in cognitive function; to follow the course of

KEY TERMS

Cognition—The act or process of knowing or perceiving.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness. It can be distinguished from dementia by its relatively sudden onset and variation in the severity of the symptoms.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Orientation—In psychiatry, the ability to locate oneself in one's environment with respect to time, place and people.

the patient's illness, and to monitor responses to treatment. Recently, it has been professionally approved as a measurement of a patient's ability to complete an advance directive, or so-called living will.

The test has also been used in research as a screener in epidemiological studies for disorders that affect cognition, and to monitor changes in subjects' cognition during clinical trials. In 2001 the MMSE was recommended by a special panel of experts for use as a screener in evaluating cognitive function in depressed patients. It has also been used recently to measure the effects of **acupuncture** in improving mood and some cognitive skills in patients with Alzheimer's.

The MMSE evaluates six areas of cognitive function: orientation, attention, immediate recall, short-term recall, language, and the ability to follow simple verbal and written commands. In addition, it provides a total score allowing the examiner to place the patient on a scale of cognitive function. It correlates well with a standard measure of cognition in adults, the **Wechsler Adult Intelligence Scale** (WAIS). In contrast to the Wechsler, which takes about an hour or more to administer, the MMSE can be completed in ten minutes or less.

Precautions

The MMSE should not be used as the sole criterion for assessment during differential **diagnosis** of psychiatric disorders, as there are many disorders and conditions that affect cognitive functioning. The results of the MMSE should be interpreted in the context of the patient's history, a full mental status examination, a physical examination, and laboratory findings, if any.

A patient's score on the MMSE must be interpreted according to his or her age and educational level. Whereas the median score is 29 for persons 18–24 years of age, it is 25 for those who are 80 or older. The median score is 22 for persons with a fourth-grade education or less; 26 for those who completed the eighth grade; and 29 for those who completed high school or college. There is a complete table available for interpreting MMSE scores according to the patient's reference groups for age and education level.

The MMSE should be administered and scored only by a qualified health care professional, such as a **psychologist**, physician, or nurse.

Description

The mini-mental state examination is divided into two sections. The first part requires vocal responses to the examiner's questions. The patient is asked to repeat a short phrase after the examiner; to count backward from 100 by 7s; to name the current President of the United States (in Great Britain, the names of the Queen and her four children); and similar brief items. It tests the patient's orientation, memory, and attention. The maximum score on this section is 21.

In the second part of the examination, the patient is asked to follow verbal and written instructions, write a sentence spontaneously, and copy a complex geometric figure similar to a Bender-Gestalt figure—a series of nine designs each on separate cards given the test taker who is asked to reproduce them on blank paper. The sentence item usually asks the patient to explain the meaning of a simple proverb such as "People who live in glass houses shouldn't throw stones." The maximum score for the second section is 9. Patients with vision problems can be assisted with large writing. The MMSE is not timed.

There is little information available on allowances made in scoring the MMSE for patients whose first language is not English or who have difficulty with standard spoken English.

Results

The maximum total score on the MMSE is 30. As a rule, scores of 20 or lower indicate delirium, dementia, **schizophrenia**, or a mood disorder. Normal subjects and those with a primary diagnosis of personality disorder score close to the median for their age and education level.

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National Institute of Neurological Disorders and Stroke (NINDS). Building 31, Room 8A06, 9000 Rockville Pike, Bethesda, MD 20892. (301) 496-5751. <www.ninds.nih.gov>.

Rebecca J. Frey, Ph.D.

Minnesota Multiphasic Personality Inventory

Definition

The Minnesota Multiphasic Personality Inventory, known as the MMPI, and its revised second edition (MMPI-2) are psychological assessment instruments completed by the person being evaluated, and scored and interpreted by the examiner. The clinician evaluates the test taker's personal characteristics by comparing the test taker's answers to those given by various psychiatric and nonpsychiatric comparison groups. By analyzing the test taker's patterns of response to the test items, the examiner is able to draw some tentative conclusions about the client's level of adaptation, behavioral characteristics, and personality traits. The MMPI-2 is preferred to the older MMPI because of its larger and more representative community comparison group (also referred to as the "normative" group). The original version of the MMPI is no longer available from the publisher, although some institutions continue to use old copies of it.

Purpose

The results of the MMPI-2 allow the test administrator to make inferences about the client's typical behaviors and way of thinking. The test outcomes help the examiner to determine the test taker's severity of impairment, outlook on life, approaches to problem solving, typical mood states, likely diagnoses, and potential problems in treatment. The MMPI-2 is used in a wide range of settings for a variety of procedures. The inventory is often used as part of inpatient psychiatric assessments, differential **diagnosis**, and outpatient evaluations. In addition, the instrument is often used by expert witnesses in forensic settings as part of an evaluation of a defendant's mental health, particularly in criminal cases. The MMPI has also been used to evaluate candidates for employment in some fields, and in educational counseling.

Precautions

Although the MMPI-2 may be administered by trained clerical staff or by computer, for best results the examiner should meet the test taker before giving the test in order to establish the context and reassure the client. Most importantly, the test responses should be interpreted only by a qualified mental health professional with postgraduate education in psychological assessment and specialized training in the use of the MMPI-2. While computer-generated narrative reports are available and can be a useful tool, they should be evaluated (and edited if needed) by the on-site professional to individualize the reported results. Computer scoring and hypothesis generation is complex, and only reputable software programs should be used.

Although the MMPI-2 may yield extensive information about the client, it is not a replacement for a clinical

KEY TERMS

Battery—A number of separate items (such as tests) used together. In psychology, a group or series of tests given with a common purpose, such as personality assessment or measurement of intelligence.

Biopsychosocial history—A history of significant past and current experiences that influence client behaviors, including medical, educational, employment, and interpersonal experiences. Alcohol or drug use and involvement with the legal system are also assessed in a biopsychosocial history.

Empirical—Verified by actual experience or by scientific experimentation.

Forensic—Pertaining to courtroom procedure or evidence used in courts of law.

Hypothesis—An assumption, proposition, or educated guess that can be tested empirically.

Personality inventory—A type of psychological test that is designed to assess a client's major personality traits, behavioral patterns, coping styles, and similar characteristics. The MMPI-2 is an example of a personality inventory.

Psychological assessment—A process of gathering and synthesizing information about a person's psychological makeup and history for a specific purpose, which may be educational, diagnostic, or forensic.

Scale—A subset of test items from a multi-item test.

interview. The clinical interview helps the test administrator to develop conclusions that best apply to the client from the many hypotheses generated from test results. Furthermore, important aspects of the client's behaviors may emerge in an interview that were not reflected in the test results. For similar reasons, the test results should not be interpreted until the clinician has obtained a biopsychosocial history from the client.

The MMPI-2 should be administered as part of a battery, or group, of tests rather than as an isolated assessment measure. A comprehensive assessment of a person will typically include the **Rorschach technique**, the **Thematic Apperception Test** (TAT) or the Sentence Completion Test, and the **Wechsler Adult Intelligence Scale**, Revised (WAIS-R) or similar test of cognitive functioning as well as the MMPI-2.

Description

The MMPI-2 is composed of 567 true/false items. It can be administered using a printed test booklet and an answer sheet filled in by hand, or by responding to the items on a computer. For the person with limited reading skills or the visually impaired respondent, the MMPI-2 items are available on audiotape. Although the MMPI-2 is frequently referred to as a test, it is not an academic test with "right" and "wrong" answers. Personality inventories like the MMPI-2 are intended to discover what the respondent is like as a person. A number of areas are "tapped into" by the MMPI-2 to answer such questions as: "Who is this person and how would he or she typically feel, think and behave? What psychological problems and issues are relevant to this person?" Associations between patterns of answers to test items and particular traits or behaviors have been discovered through personality research conducted with the MMPI-2. The inventory items are not arranged into topics or areas on the test. The areas of personality that are measured are interspersed in a somewhat random fashion throughout the MMPI-2 booklet. Some examples of true-or-false statements similar to those on the MMPI-2 are: "I wake up with a headache almost every day"; "I certainly feel worthless sometimes"; "I have had peculiar and disturbing experiences that most other people have not had"; "I would like to do the work of a choir director."

The MMPI-2 is intended for use with adults over age 18; a similar test, the MMPI-A, is designed for use with adolescents. The publisher produces the MMPI-2 in English and Spanish versions. The test has also been translated into Dutch-Flemish, two French dialects (France and Canada), German, Hebrew, Hmong, Italian, and three Spanish dialects (for Spain, Mexico or United States).

From the 1940s to the 1980s, the original MMPI was the most widely used and most intensely researched psychological assessment instrument in the United States and worldwide. The test was originally developed in 1943 using a process called empirical keying, which was an innovation. Most assessment tools prior to the MMPI used questions or tasks that were merely assumed by the test designer to realistically assess the behaviors under question. The empirical keying process was radically different. To develop empirical keying, the creators of the original MMPI wrote a wide range of true-or-false statements, many of which did not directly target typical psychiatric topics. Research was then conducted with groups of psychiatric inpatients, hospital visitors, college students and medical inpatients, who took the MMPI in order to determine which test items reliably differentiated the psychiatric patients from the others. The test developers also evaluated the items that reliably distin-

guished groups of patients with a particular diagnosis from the remaining pool of psychiatric patient respondents; these items were grouped into subsets referred to as clinical scales.

An additional innovation in the original MMPI was the presence of validity scales embedded in the test questions. These sets of items, scattered randomly throughout the MMPI-2, allow the examiner to assess whether the respondent answered questions in an open and honest manner, or tried to exaggerate or conceal information. One means of checking for distortions in responding to the instrument is asking whether the test taker refused to admit to some less-than-ideal actions that most people probably engage in and will admit to doing. An example of this type of question would be (true or false) "If I could sneak into the county fair or an amusement park without paying, I would." Another type of validity check that assesses honesty in responses is whether the client admits to participating in far more unusual behaviors and actions than were admitted to by both the psychiatric comparison group and the general community sample. The validity scales also identify whether the test taker responded inconsistently or randomly.

The MMPI-2, which has demonstrated continuity and comparability with its predecessor, was published in 1989. The revised version was based on a much larger and more racially and culturally diverse normative community comparison group than the original version. Also, more in-depth and stringent research on the qualities and behaviors associated with different patterns of scores allows improved accuracy in predicting test-respondents' traits and behaviors from their test results.

Results

The true/false items are organized after scoring into validity, clinical, and content scales. The inventory may be scored manually or by computer. After scoring, the configuration of the test taker's scale scores is marked on a profile form that contrasts each client's responses to results obtained by the representative community comparison group. The clinician is able to compare a respondent's choices to those of a large normative comparison group as well as to the results derived from earlier MMPI and MMPI-2 studies. The clinician forms inferences about the client by analyzing his or her response patterns on the validity, clinical and content scales, using published guidebooks to the MMPI-2. These texts are based on results obtained from over 10,000 MMPI/MMPI-2 research studies.

In addition to the standard validity, clinical, and content scales, numerous additional scales for the MMPI have been created for special purposes over the years by researchers. These special supplementary scale scores are often incorporated into the examiner's interpretation of the test results. Commonly used supplementary scales include the MacAndrews Revised Alcoholism Scale, the **Addiction** Potential Scale, and the Anxiety Scale. The clinician may also choose to obtain computerized reporting, which yields behavioral hypotheses about the respondent, using scoring and interpretation algorithms applied to a commercial database.

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Deborah Rosch Eifert, Ph.D.

Mirtazapine

Definition

Mirtazapine is most commonly used to treat depression. Mirtazapine is available in the United States under the trade names of Remeron and Remeron SolTab.

Mirtazapine, sold under the trade name Remeron, is taken by mouth and swallowed whole. Remeron SolTabs should be allowed to dissolve in the mouth. No water is needed when taking the SolTabs, since these tablets disintegrate in saliva and are not swallowed whole.

KEY TERMS

Anti-anxiety agent—A medication that is used to treat symptoms of generalized fear that dominates a person's life.

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Antipsychotic—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Depression—A mental state characterized by excessive sadness. Other symptoms include altered sleep patterns, thoughts of suicide, difficulty concentrating, agitation, lack of energy, and loss of enjoyment in activities that are usually pleasurable.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Purpose

Mirtazapine is best known for treating depression. However, it may also be used for treating anxiety or to make people drowsy just before surgery.

Description

Mirtazapine is usually thought of as an antidepressant, or a drug that alleviates symptoms of depression. Approved by the Federal Drug Administration (FDA) in 1996, it is believed to alter the activities of some chemicals in the **brain** and, in this way, reduce chemical imbalances responsible for causing depression and anxiety. As with all antidepressants, it may take several weeks of treatment before full beneficial effects are seen. Mirtazapine is broken down by the liver and eliminated from the body mostly by the kidneys. It is supplied in 15-, 30-, and 45-mg tablets.

Recommended dosage

The recommended initial dose of mirtazapine in 15 mg taken at bedtime. The dose may be increased in 15-mg increments every one or two weeks as needed until symptoms of depression or anxiety resolve. Typical doses range between 15 and 45 mg. Dosages above 45 mg per day are not recommended. Elderly people or those with liver or kidney disease should use mirtazapine carefully, since they may be more sensitive to some of the drug's side effects.

Precautions

Mirtazapine may cause weight gain and may increase cholesterol levels and should be used carefully in overweight individuals and those with high cholesterol levels. If symptoms of fever, sore throat, or irritation in the mouth occur, a health care provider should be notified. Rarely, mirtazapine may lower blood counts, causing people to be at an increased risk of serious complications, including infections. Mirtazapine may increase the tendency for **seizures**. As a result, it should be used carefully in people with epilepsy or other seizure disorders. Mirtazapine may alter moods or cause mania. It should be used carefully in people with a history of mania. Mirtazapine may alter liver function and should be used cautiously by those with a history of liver disease. If abdominal pain, yellowing of the skin or eyes, darkening of urine, or itching occurs, a health care provider should be notified immediately.

More than 50% of individuals using mirtazapine report feeling sleepier than normal and 7% feel dizzy. As a result, people taking mirtazapine should not participate in activities that require mental alertness—like driving—until they know how the drug will affect them. Because there is an increased likelihood of **suicide** in depressed individuals, close supervision of those at high risk for suicide attempts using this drug is recommended. Mirtazapine is not recommended in pregnant or breast-feeding women.

Side effects

The most common side effects that cause people to stop taking mirtazapine are sleepiness and nausea. Other common side effects are dizziness, increased appetite and weight gain. Less common adverse effects include weakness and muscle aches, flu-like symptoms, low blood-cell counts, high cholesterol, back pain, chest pain, rapid heartbeats, dry mouth, constipation, water retention, difficulty sleeping, nightmares, abnormal thoughts, vision disturbances, ringing in the ears, abnormal taste in the mouth, tremor, confusion, upset stomach, and increased urination.

Interactions

Use of mirtazapine with antidepressants referred to as monoamine oxidase inhibitors (MAOIs) such as Parnate (**tranylcypromine**) and Nardil (**phenelzine**), is

strongly prohibited due to the potential for high fever, muscle stiffness, sudden muscle spasms, rapid changes in heart rate and blood pressure, and the possibility of death. In fact, there should be a lapse of at least 14 days between taking an MAOI and mirtazapine.

Because mirtazapine may cause drowsiness, it should be used carefully with other medications that also make people prone to sleepiness, such as antidepressants, antipsychotics, antihistamines, anti-anxiety agents, and alcohol. Increased sleepiness has been reported when mirtazapine was used with both alcohol and the anti-anxiety drug **diazepam**.

See also Depression and depressive disorders

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Mixed episode

Definition

A mixed episode is a discrete period during which a person experiences nearly daily fluctuations in mood that qualify for diagnoses of **manic episode** and major depressive episode. Over the course of at least one week, the mood of a person experiencing a mixed episode will rapidly change between abnormal happiness or euphoria and sadness or irritability.

Description

To qualify for a **diagnosis** of mixed episode, symptoms must be severe enough to interfere with an individual's ability to carry out daily routines at work or home, or to require **hospitalization**. Males may be more susceptible to this condition than females. Young people and those more than 60 years of age with **bipolar disorder** may be more prone to mixed episodes than others. A manic episode or a major depressive episode is more likely to turn into a mixed episode than vice versa. Manic episodes can also appear in an individual who does not suffer from these or other disturbances. If the episode can be attributed to side effects related to any medical treatment, medical condition, medication, or drugs of abuse, it is not classified as a mixed episode.

See also Bipolar disorder ; Depression and depressive disorders; Major depressive disorder

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Mixed receptive-expressive language disorder

Definition

Mixed receptive-expressive language disorder is diagnosed when a child has problems expressing him- or herself using spoken language, and also has problems understanding what people say to him or her.

Description

Mixed receptive-expressive language disorder is generally a disorder of childhood. There are two types of mixed receptive-expressive language disorder: developmental and acquired. Developmental mixed receptive-expressive language disorder does not have a known cause and normally appears at the time that a child is learning to talk. Acquired mixed receptive-expressive language disorder is caused by direct damage to the **brain**. It occurs suddenly after such events as a **stroke** or traumatic head injury. The acquired type can occur at any age.

Causes and symptoms

Causes

There is no known cause of developmental mixed receptive-expressive language disorder. Researchers are conducting ongoing studies to determine whether biological or environmental factors may be involved. The acquired form of the disorder results from direct damage to the brain. Damage can be sustained during a stroke, or as the result of traumatic head injury, **seizures**, or other medical conditions. The specific symptoms of the acquired form of the disorder generally depend on the parts of the patient's brain that have been injured and the severity of the damage.

Symptoms

The signs and symptoms of mixed receptive-expressive language disorder are for the most part the same as

KEY TERMS

Phonological disorder—A developmental disorder of childhood in which the child fails to use speech sounds that are appropriate for his or her age level and native language or dialect.

the symptoms of **expressive language disorder**. The disorder has signs and symptoms that vary considerably from child to child. In general, mixed receptive-expressive language disorder is characterized by a child's difficulty with spoken communication. The child does not have problems with the pronunciation of words, which is found in **phonological disorder**. The child does, however, have problems constructing coherent sentences, using proper grammar, recalling words, or similar communication problems. A child with mixed receptive-expressive language disorder is not able to communicate thoughts, needs, or wants at the same level or with the same complexity as his or her peers. In addition, the child often has a smaller vocabulary than his or her peers.

Children with mixed receptive-expressive language disorder also have significant problems understanding what other people are saying to them. This lack of comprehension may result in inappropriate responses or failure to follow directions. Some people think these children are being deliberately stubborn or obnoxious, but this is not the case. They simply do not understand what is being said. Some children with this disorder have problems understanding such specific types of terms as abstract nouns, complex sentences, or spatial terms.

Diagnosis

The ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revised (*DSM-IV-TR*), which is the standard reference work consulted by mental health professionals, specifies four general criteria for diagnosing mixed receptive-expressive language disorder. The first criterion states that the child communicates using speech and appears to understand spoken language at a level that is lower than expected for the child's general level of intelligence. Second, the child's problems with self-expression and comprehension must create difficulties for him or her in everyday life or in achieving his or her academic goals. If the child understands what is being said at a level that is normal for his or her age or stage of development, then the **diagnosis** would be expressive language disorder. If the child is mentally retarded, hard of hearing, or has other physical problems, the difficulties with speech must be greater than generally occurs with the other handicaps the child may have in order for the child to be diagnosed with this disorder.

The disorder is usually diagnosed in children because a parent or teacher expresses concern about the child's problems with spoken communication. The child's pediatrician may give the child a physical examination to rule out such medical problems as hearing loss. Specific testing for mixed expressive-receptive language disorder requires the examiner to demonstrate that the child not only communicates less well than expected, but also understands speech less well. It can be hard, however, to determine what a child understands. As a result, most examiners will use non-verbal tests in addition to tests that require spoken questions and answers in order to assess the child's condition as accurately as possible. In children who are mildly hearing-impaired, the problem can often be corrected by using hearing aids. Children who speak a language other than English (or the dominant language of their society) at home should be tested in that language if possible. In some cases, the child's ability to understand and communicate in English is the problem, not his or her competence with spoken language in general.

Demographics

Mixed receptive-expressive language disorder is diagnosed in about 5% of preschool-age children, and 3% of children in school. It is less common than expressive language disorder. Children who have mixed receptive-expressive language disorder are more likely to have other disorders as well. Between 40%–60% of preschoolers who have this disorder may also have phonological disorder (difficulty forming sounds). **Reading disorder** is linked to as many as half the children with mixed receptive-expressive language disorder who are of school age. Children with mixed receptive-expressive language disorder are also more likely to have psychiatric disorders, especially attention-deficit disorder (ADD); it is estimated that 30–60 percent of children with mixed receptive-expressive language disorder also have ADD. Children from families with a history of language disorders are more likely to have this or other language disorders.

Treatment

Mixed receptive-expressive language disorder should be treated as soon as it is identified. Early **intervention** is the key to a successful outcome. Treatment involves teachers, siblings, parents, and anyone else who interacts regularly with the child. Regularly scheduled one-on-one treatment that focuses on specific language skills can also be effective, especially when combined

with a more general approach involving family members and caregivers. Teaching children with this disorder specific communication skills so that they can interact with their peers is important, as problems in this area may lead to later social isolation, depression, or behavioral problems. Children who are diagnosed early and taught reading skills may benefit especially, because problems with reading are often associated with mixed receptive-expressive language disorder and can cause serious long-term academic problems. There is little information comparing different treatment methods; often several are tried in combination.

Prognosis

The developmental form of mixed receptive-expressive language disorder is less likely to resolve well than the developmental form of expressive language disorder. Most children with the disorder continue to have problems with language skills. They develop them at a much slower rate than their peers, which puts them at a growing disadvantage throughout their educational career. Some persons diagnosed with the disorder as children have significant problems with expressing themselves and understanding others in adult life.

The prognosis of the acquired type of mixed receptive-expressive language disorder depends on the nature and location of the brain injury. Some people get their language skills back over days or months. For others it takes years, and some people never fully recover expressive language function or the ability to understand speech.

Prevention

Because the causes of developmental mixed receptive-expressive language disorder are unclear, there are no specific ways to prevent it. A healthy diet during pregnancy and regular prenatal care are always recommended. Because the acquired form of the disorder is caused by damage to the brain, anything that helps to prevent brain damage may offer protection against that form of the disorder. Preventive measures include such precautions as lowering blood cholesterol levels, which may help to prevent stroke; or wearing bicycle helmets or automobile seat belts to prevent traumatic head injury.

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American Speech-Language-Hearing Association. 10801 Rockville Pike, Rockville, MD 20852. (800) 638-8355. <http://www.asha.org>.

See also Attention-deficit/hyperactivity disorder

Tish Davidson, A.M.

MMPI *see* **Minnesota Multiphasic Personality Inventory**

Moban *see* **Molindone**

Modeling

Definition

Modeling, which is also called observational learning or imitation, is a behaviorally based procedure that involves the use of live or symbolic models to demonstrate a particular behavior, thought, or attitude that a client may want to acquire or change. Modeling is sometimes called vicarious learning, because the client need not actually perform the behavior in order to learn it.

Purpose

Modeling therapy is based on social learning theory. This theory emphasizes the importance of learning from observing and imitating role models, and learning about rewards and punishments that follow behavior. The technique has been used to eliminate unwanted behaviors, reduce excessive fears, facilitate learning of social behaviors, and many more. Modeling may be used either to strengthen or to weaken previously learned behaviors.

Modeling has been used effectively to treat individuals with anxiety disorders, **post-traumatic stress disorder**, **specific phobias**, **obsessive-compulsive disorder**, eating disorders, **attention-deficit/hyperactivity disorder**, and **conduct disorder**. It has also been used

KEY TERMS

Generalization—A person's ongoing use of new behaviors that were previously modeled for him or her. Generalization is also called transfer of training or maintenance.

In vivo—A Latin phrase that means "in life." In modeling and exposure therapies, it refers to practicing new behaviors in a real setting, as distinct from using imagery or imagined settings.

Reinforcement—In behavioral therapy, the ability of a behavior to produce effects that will make the user want to perform the behavior again. In modeling, reinforcement refers to rewarding the model's demonstration of a skill or the client's performance of the newly acquired skill in practice or in real-life situations.

Role-playing—A technique used in therapy in which participants act out roles relevant to real-life situations in order to change their attitudes and behaviors.

Vicarious—Acquired through imagined participation in the experience of others. Modeling is a form of vicarious learning.

successfully in helping individuals acquire such social skills as public speaking or assertiveness. The effectiveness of modeling has led to its use in behavioral treatment of persons with substance abuse disorders, who frequently lack important behavioral skills. These persons may lack assertiveness, including the ability to say "no"; in addition, they may have thought patterns that make them more susceptible to substance abuse.

Modeling when used alone has been shown to be effective for short-term learning. It is, however, insufficient for long-lasting behavior change if the target behavior does not produce rewards that sustain it. Modeling works well when it is combined with role-play and **reinforcement**. These three components are used in a sequence of modeling, role-play, and reinforcement. Role-play is defined as practice or behavioral rehearsal of a skill to be used later in real-life situations. Reinforcement is defined as rewarding the model's performance or the client's performance of the newly acquired skill in practice or in real-life situations.

Several factors increase the effectiveness of modeling therapy in changing behaviors. Modeling effects have been shown to be more powerful when:

- The model is highly skilled in enacting the behavior; is likable or admirable; is friendly; is the same sex and age; and is rewarded immediately for the performance of the particular behavior.
- The target behavior is clearly demonstrated with very few unnecessary details; is presented from the least to the most difficult level of behavior; and several different models are used to perform the same behavior(s).

Description

Types of modeling

Therapy begins with an assessment of the client's presenting problem(s). The assessment usually covers several areas of life, including developmental history (the client's family background, education, employment, social relationships); past traumatic experiences; medical and psychiatric history; and an outline of the client's goals. The client works with the therapist to list specific treatment goals; to determine the target behavior(s) to be learned or changed; and to develop a clear picture of what the behavior(s) will look like. The therapist then explains the rationale and concepts of the treatment. He or she also considers any negative consequences that may arise as the client makes changes in his or her behavior.

The client then observes the model enacting the desired behavior. Some models may demonstrate poor or inadequate behaviors as well as those that are effective. This contrast helps the client to identify ineffective behaviors as well as desired ones. Modeling can be done in several different ways, including live modeling, symbolic modeling, participant modeling, or covert modeling.

Live modeling refers to watching a real person, usually the therapist, perform the desired behavior the client has chosen to learn. For example, the therapist might model good telephone manners for a client who wants a job in a field that requires frequent telephone contact with customers.

Symbolic modeling includes filmed or videotaped models demonstrating the desired behavior. Other examples of symbolic models include photographs, picture books, and plays. A common example of symbolic modeling is a book for children about going to the hospital, intended to reduce a child's anxiety about hospitals and operations. With child clients, cartoon figures or puppets can be used as the models. Self-modeling is another form of symbolic modeling in which clients are videotaped performing the target behavior. The video is than replayed and clients can observe their behaviors and how they appear to others. For example, public speaking is one of the most common feared situations in the general adult population. A law student who is afraid of having to present arguments

Young girls in a ballet dancing class. The instructors are serving as live models, showing the girls a behavior that they are to imitate and practice. This is an example of learning through modeling. *(Bob Krist/CORBIS. Photo reproduced by permission.)*

in a courtroom might be videotaped speaking to classmates who are role-playing the judge and members of the jury. The student can then review the videotape and work on his or her speech problems or other aspects of the performance that he or she would like to change.

In participant modeling, the therapist models anxiety-evoking behaviors for the client, and then prompts the client to engage in the behavior. The client first watches as the therapist approaches the feared object, and then approaches the object in steps or stages with the therapist'sencouragement and support. This type of modeling is often used in the treatment of specific phobias. For example, a person who is afraid of dogs might be asked to watch the therapist touch or pet a dog, or perhaps accompany the therapist on a brief walk with a dog. Then, with the therapist's encouragement, the client might begin by touching or holding a stuffed dog, then watching a live dog from a distance, then perhaps walking a small dog on a leash, and eventually by degrees touching and petting a live dog.

In covert modeling, clients are asked to use their imagination, visualizing a particular behavior as the therapist describes the imaginary situation in detail. For example, a child may be asked to imagine one of his or her favorite cartoon characters interacting appropriately with other characters. An adult client is asked to imagine an admired person in his or her life performing a behavior that the client wishes to learn. For example, a person may greatly admire his or her mother for the way she handled the challenges of coming to the United States from another country. If the client is worried about the challenge of a new situation (changing careers, having their first child, etc.), the therapist may ask him or her to imagine how their mother would approach the new situation, and then imagine themselves acting with her courage and wisdom.

Models in any of these forms may be presented as either a coping or a mastery model. The coping model is shown as initially fearful or incompetent and then is shown as gradually becoming comfortable and competent performing the feared behavior. A coping model might show a small child who is afraid of swimming in the ocean, for example. The little boy or girl watches smaller children having fun playing in the waves along the edge of the shore. Gradually the child moves closer

and closer to the water and finally follows a child his or her age into the surf. The mastery model shows no fear and is competent from the beginning of the demonstration. Coping models are considered more appropriate for reducing fear because they look more like the client, who will probably make mistakes and have some setbacks when trying the new behavior.

Having the model speak his or her thoughts aloud is more effective than having a model who does not verbalize. As the models speak, they show the client how to think through a particular problem or situation. A common example of this type of modeling is sports or cooking instruction. A golf or tennis pro who is trying to teach a beginner how to hold and swing the club or racquet will often talk as they demonstrate the correct stance and body movements. Similarly, a master chef will often talk to students in a cooking class while he or she is cutting up the ingredients for a dish, preparing a sauce, kneading dough, or doing other necessary tasks. The model's talking while performing an action also engages the client's sense of hearing, taste, or smell as well as sight. Multisensory involvement enhances the client's learning.

Role-playing

Role-playing is a technique that allows the client opportunities to imitate the modeled behaviors, which strengthens what has been learned. Role-play can be defined as practice or behavior rehearsal; it allows the client to receive feedback about the practice as well as encouraging the use of the newly learned skill in real-life situations. For example, a group of people who are trying to learn social skills might practice the skills needed for a job interview or for dealing with a minor problem (returning a defective item to a store, asking someone for directions, etc.). Role-play can also be used for modeling, in that the therapist may role-play certain situations with clients. During practice, the therapist frequently coaches, prompts, and shapes the client's enactment of the behavior so that the rehearsals can come increasingly close to the desired behavior.

Feedback and social reinforcement of the client's performance in the practice phase is an important motivator for behavior change. Feedback may take the form of praise, approval, or encouragement; or it may be corrective, with concrete suggestions for improving the performance. Suggestions are followed by additional practice. Such tangible reinforcements as money, food, candy, or tokens have been used with young children and chronic psychiatric patients. The therapist may teach the client how to use self-reinforcement; that is, using self-praise after performing the desired behavior. The purpose of reinforcement is to shift the client's performance concerns from external evaluation by others to internal evaluation of their own efforts.

Modeling in group settings

Modeling has been shown to be effective in such group programs as **social skills training** and **assertiveness training** as well as in individual therapy. The general approach to both social skills training and assertiveness training is the incorporation of the modeling, role-play, and reinforcement sequence. After assessment of each group member's presenting problem, each member is asked to keep a diary of what happened when the situation occurred during the week. Group members develop goals for dealing with their individual situations, and each person determines how he or she can meet these goals. Modeling is done with either the therapist or other group members role-playing how to deal effectively with a particular problem situation.

Length of treatment

While modeling therapy is a relatively short-term approach to behavioral change, some therapeutic techniques take longer than others. Imagery, for example, requires more sessions than in vivo (real-life) treatments. In vivo work that takes place outside the therapist's office would require longer time periods for each session. Other considerations include the nature of the client's problem; the client's willingness to do homework; the client's financial resources; and the presence and extent of the client's support network. The therapist's length of experience and personal style also affect the length of therapy.

There are, however, guidelines of treatment length for some disorders. Treatment of obsessive-compulsive disorder may require five weekly sessions for approximately three weeks, with weekly follow-up sessions for several months. Depressive disorders may require three to six months, with the client experiencing short-term relief after three to four weeks of treatment. General anxiety disorder may also take several months of weekly sessions. The length of treatment depends on the ability to define and assess the target behaviors. Clients may meet with the therapist several times a week at the beginning of treatment; then weekly for several months; then monthly for follow-up sessions that may become fewer in number or spaced more widely until therapy is terminated.

Normal results

Modeling or observational learning is effective as a method of learning such behaviors as self-assertion, self-disclosure, helping others, empathic behaviors, moral

judgment, and many other interpersonal skills. Modeling is also effective in eliminating or reducing such undesirable behaviors as uncontrolled aggression, smoking, weight problems, and single phobias.

The expected outcome is that clients will be able to use their new behaviors outside the treatment setting in real-life situations. This result is called transfer of training, generalization, or maintenance. Homework is the most frequently used technique for transfer of training. Homework may represent a contractual agreement between the therapist and the client in which the client gives a report on his or her progress at each meeting.

To ensure that generalization occurs and that clients will use their new skills, several "tranfer enhancers" are used to increase the likelihood of successful transfer of training. Transfer enhancers include:

- Giving clients appropriate rationales and concepts, rules, or strategies for using skills properly.
- Giving clients ample opportunity to practice new skills correctly and successfully.
- Making the treatment setting as much like the real-life situation as possible.
- Giving clients opportunities to practice their new skills in a variety of physical and interpersonal settings.
- Giving clients adequate external social reinforcement and encouraging internal self-reinforcement as they use their skills successfully in real life.

See also Behavior modification

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American Psychological Association. 750 First St. N.E., Washington, D.C. 20002. (202) 336-5800. <http://helping.apa.org>.

Association for Advancement of Behavior Therapy. 305 Seventh Ave., 16th Floor, New York, NY 10001. (212) 647-1890. <http://www.aabt,org>.

National Institute of Mental Health. 6001 Executive Boulevard, RM8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

National Mental Health Association. 1021 Prince Street, Alexander, VA 22314-2971. (703) 684-7722. <http://www.nmha.org>.

Janice VanBuren, Ph.D.

Molindone

Definition

Molindone is an antipsychotic. It is sold in the United States under the trade name of Moban.

Purpose

Molindone is used to treat psychotic symptoms that may appear in depression, mania, or **schizophrenia**.

Description

Molindone is taken orally, and is rapidly absorbed and metabolized. Peak levels are reached within 90 minutes of taking the medication, and its effect lasts 24 to 36 hours. Molindone is available in 5-, 10-, 25-, and 100-mg tablets.

Recommended dosage

The dosage of molindone should be adjusted to the lowest level needed to control symptoms. The usual initial dosage is 50 to 75 mg per day. This may be increased to 100 mg per day three to four days after beginning treatment. A maximal dosage of up to 225 mg per day may be required.

Precautions

Prolonged or chronic administration of molindone increases the probability of developing **tardive dyskinesia**, a cluster of involuntary, uncoordinated movements that is potentially irreversible. These movements involve the head, neck, trunk, feet, and hands. Some of the movements involving the face and head include worm-like movement of the tongue, grimacing, chewing, and lip smacking. Tardive dyskinesia usually disappears once the affected person stops taking the medication, but it may not.

KEY TERMS

Akathisia—Agitated or restless movement, usually affecting the legs. Movement is accompanied by a sense of discomfort and an inability to sit, stand still, or remain inactive for periods of time. Akathisia is a common side effect of some neuroleptic (antipsychotic) medications.

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

Orthostatic hypotension—A sudden decrease in blood pressure due to a change in body position, as when moving from a sitting to standing position.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

Tranquilizer—A medication that induces a feeling of calm and relaxation.

People who are comatose or are experiencing central nervous system depression from alcohol, **barbiturates** or narcotics are not prescribed this medication.

Drowsiness is often reported by people using molindone. For that reason, people using molindone should not operate machinery or drive automobiles.

Molindone administration causes the level of prolactin (a hormone that initiates lactation) in the blood to rise. This is a potential problem for people with a personal or family history of breast cancer. The drug may lead to the initiation of breast cancer. For this reason, the benefits of the drug must be carefully evaluated before it is administered.

Side effects

As stated, molindone has the potential to produce tardive dyskinesia. This is a syndrome consisting of involuntary, uncoordinated movements that is potentially irreversible. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage of molindone. Tardive dyskinesia is more likely to occur after a long period of taking antipsychotic drugs, like molindone, but it may also appear after molindone use has been discontinued. Females are at greater risk than males for developing tardive dyskinesia. Involuntary movements of the tongue, jaw, mouth or face characterize tardive dyskinesia. These may be accompanied by involuntary movements of the arms, legs and trunk. There is no known effective treatment for tardive dyskinesia.

Parkinson-like symptoms have been linked with the administration of molindone. These include restlessness and agitation (akathisia) and difficulty walking or moving (dystonia). These are generally controlled with **benztropine** mesylate or **trihexyphenidyl** hydrochloride.

An occasionally reported side effect of molindone is neuroleptic malignant syndrome. This is a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia and arrhythmias. This condition is considered a medical emergency.

Interactions

Molindone increases the effect of drugs and substances that depress the central nervous system. This class of drugs includes anesthetics, opiates, barbiturates, atropine and alcohol.

Molindone interferes with the absorption of phenytoin and tetracyclines.

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association, 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax: (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

L. Fleming Fallon, Jr., M.D., Dr.P.H.

Mood disorders *see* **Depression and depressive disorders and Bipolar disorders**

Movement disorders

Definition

Movement disorders describe a variety of abnormal movements of the body that have a neurological basis. These abnormal movements are characterized by changes in the coordination and speed of voluntary movement. They may also involve the presence of additional movements that are not voluntary.

Description

Movement disorders are sometimes referred to by medical professionals as extrapyramidal diseases because this class of disorders is distinct from the disorders caused by disorders of the pyramidal region of the **brain**. Researchers have determined that movement disorders are caused by diseases in various parts of the brain, including the substantia nigra, the subthalamic nucleus, the globus pallidus, the striatum, and the basal ganglia.

KEY TERMS

Basal ganglia—A group of masses of gray matter located in the cerebral hemispheres of the brain that control movement as well as some aspects of emotion and cognition.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Extrapyramidal—Brain structures located outside the pyramidal tracts of the central nervous system.

Substantia nigra—Dark-colored matter located in a section of the crus cerebri area of the brain.

Movement disorders are usually broken down into two types of movement: hyperkinetic movement and hypokinetic movement. Hyperkinetic movement disorders are characterized by a significant and excessive amount of motor activity. This type also includes cases in which there is a significant amount of abnormal involuntary movement. Hypokinetic movement disorders are those in which there is an abnormally reduced amount of intentional motor activity.

Hyperkinetic movement disorders are characterized by two types of behavior: rhythmical and irregular. Tremor is a rhythmic movement that is further divided into three forms: rest, postural, and intention. Rest tremor is most prominent when an individual is at rest and decreases with voluntary activity. Postural tremor occurs when an individual attempts to support a position against gravity (such as holding an arm outstretched). Intention tremor occurs during voluntary movement toward a specific target.

Irregular involuntary movements are classified by their speed and site of occurrence. Tics are rapid irregular movements that are controlled with voluntary effort. The types of rapid irregular movements that cannot be controlled voluntarily are called chorea, hemiballismus, and myoclonus. Chorea is a rapid, jerking movement that most

often affects the face or limbs. Hemiballismus is the sudden and extreme swinging of a limb. Myoclonus is a rapid, irregular movement that usually occurs for a short period of time. It usually occurs when the person is at rest, and it often affects more than one area of the body at a time.

One of the most well-known hyperkinetic movement disorders is called Huntington's disease, characterized by chorea-type movements. This disease is inherited and usually develops between 30 and 50 years of age. Persons with this condition have progressive **dementia**, and the condition eventually causes death. Children of persons with Huntington's disease have a 50% chance of developing the condition. **Stereotypic movement disorder** is characterized by repetitive behaviors that meet no functional need such as hand waving; rocking; head banging; mouthing of objects; or biting, picking, or hitting oneself. These behaviors interfere with normal activities and are not caused by substance abuse or a general medical condition.

The symptoms of hypokinetic movement disorders include a rigid, stone-like face; decreased limb motion during walking; and stiff turning movements. These features are classified as bradykinesia, while akinesia is the absence of purposeful movement. The most common type of hypokinetic movement disorder is Parkinson's disease, caused by the loss of neurons containing dopamine in the area of the brain called the substantia nigra pars compacta. The loss of these neurons is a part of the alteration of vital motor circuits in the brain that leads to a slowing of intentional movements.

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ORGANIZATIONS

NIH Neurological Institute. P.O. Box 5801. Bethesda, MD 20824. (800) 352-9424. <http://www.ninds.nih.gov>.

Mark Mitchell, M.D.

Movement therapies *see* **Bodywork therapies**

MRI *see* **Magnetic resonance imaging**

Multiple personality disorder *see* **Dissociative identity disorder**

Multisystemic therapy

Definition

Multisystemic therapy (MST) is an intensive family- and community-based treatment program designed to make positive changes in the various social systems (home, school, community, peer relations) that contribute to the serious antisocial behaviors of children and adolescents who are at risk for out-of-home placement. These out-of-home placements might include foster care, **group homes**, residential care, correctional facilities, or **hospitalization**.

Purpose

MST is licensed by MST Services, Inc., through the Medical University of South Carolina and operates with the fundamental assumption that parents (defined as guardians), or those who have primary caregiving responsibilities to children, have the most important influence in changing problem behaviors in children and adolescents.

The primary goals of MST are to:

- develop in parents or caregivers the capacity to manage future difficulties
- reduce juvenile criminal activity
- reduce other types of antisocial behaviors, such as drug abuse
- achieve these outcomes at a cost savings by decreasing rates of incarceration and other out-of-home placements

MST was created approximately 25 years ago as an intensive family- and community-based treatment program to focus on juvenile offenders presenting with serious antisocial behaviors and who were at-risk for out-of-home placement. The program has been shown to be effective with targeted populations that include inner-city delinquents, violent and chronic juvenile offenders, juvenile offenders who abuse or are dependent on substances and also have psychiatric disorders, adolescent sex offenders, and abusive and neglectful parents. A more recent focus (1994–1999) of MST has been to treat youths with psychiatric emergencies such as suicidal ideation, homicidal ideation, **psychosis**, or threat of harm to self or others due to mental illness. The results are promising and indicate that MST is an effective alternative to psychiatric hospitalization. Some treatment conditions and interventions were modified to take care of this population, including developing a crisis plan during the initial family assessment and adding child and adolescent psychiatrists, psychiatric residents, and crisis casework-

ers to the MST treatment team. Supervision of the treatment team was initially increased from weekly to daily meetings. Caseloads of MST therapists were reduced from five to three families, increasing the intensity of the **intervention**. When some adolescents were hospitalized for safety, the MST staff maintained clinical responsibility for the adolescent who was insulated from the usual activities due to inpatient care.

Description

MST programs are usually housed in community-based mental health organizations considered to have a culture more rehabilitative than punitive. The program staff creates strong working relationships with referral sources such as juvenile justice and the family court. They work closely with deputy juvenile officers, social welfare workers, teachers, and guidance counselors, for example, to obtain the perspectives of multiple systems or "stake-holders" who have the common goal of improving children, adolescent and family treatment goals. Each youth referred to the program is assigned to an MST therapist who designs individualized interventions in accordance with the nine MST treatment principles, thereby addressing specific needs of the youth and his or her specific environment.

MST is a time-limited (four to six months) intensive therapeutic program that provides services in the family's home, at other locations (school, neighborhoods), or wherever the family feels most comfortable. After the initial sessions, family members who attend family sessions with the therapist will vary depending on the nature of the particular problem being discussed. For example, children are not included in sessions addressing intimate marital issues between parents or dealing with poor parental discipline, so as not to undermine parental authority.

Characteristics of the MST model—such as availability of the MST staff (24 hours a day, seven days per week), flexible scheduling, and delivery of services in the home—all provide safety for the family, prevent violence, develop a joint working relationship between therapist and family, provide the family with easier access to needed services, increase the likelihood that the family will stay in treatment, and help the family maintain changes in behaviors. The MST staff are full-time practitioners, wear pagers, carry cellular telephones, and work in teams of three. They can provide intensive services because of small caseloads and have multiple contacts with the family during the week (sometimes daily). They stay as long as required and at times most convenient to the family, including weekends, evenings, and holidays. Services provided by staff at unusual times (10 P.M. to 8 A.M.) are discouraged, except in emergencies. The development of an informal support system in which the family can call on a friend or family member at crucial times is part of the treatment goals. Families have less contact with the therapist as they get closer to being discharged from treatment.

KEY TERMS

Psychotropic medication—Medication that has an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

Punitive—Concerned with, or directed toward, punishment.

Rehabilitative—To restore; to put back into good condition.

MST is designed to be a flexible intervention to provide highly individualized treatment to families. Specific treatment techniques or therapies are used as a part of MST interventions. These include behavior parent training, structural **family therapy**, and strategic family and **cognitive-behavioral therapy**. In addition, some biological influences such as **depression and depressive disorders** may be identified, and psychotropic medications are integrated into treatment. This model does not support one method for obtaining successful changes in behaviors; however, there are nine guiding principles of treatment:

- The primary purpose of assessment is to understand the fit between the identified problems and their broader systemic context. At the initial visit with the family, the staff begins to assess the family's strengths; capabilities; needs; problems; environmental support systems; and transactions with social systems such as peers, extended family, friends, teachers, parental workplace, referral resources, and neighbors. The therapist and family work together to identify and prioritize problems to be targeted for change, determine interventions, and develop a treatment plan. The assessment is conducted in a manner that empowers family members by encouraging them to define their problems, needs, strengths, and—except in matters of imminent safety—set their priorities. The assessment is gradually updated until the family has reached its goals and is functioning independently.
- Therapeutic contacts emphasize the positive and should use systemic strengths as levers for change. MST is a strength-based treatment program and adherence to this principle decreases negativity among family members,

builds positive expectations and hope, identifies strengths, and decreases therapist and family frustrations by emphasizing problem-solving. It also builds the caregiver's confidence. The therapist develops and maintains the focus on the strength of families and positive thinking through the use of positive language, teaching, and the technique of reframing negative thoughts and beliefs; the liberal use of positive rewards for appropriate behaviors; using a problem-solving stance rather than one of failure and seeing barriers as challenges; and identifying and using what the family does well.

- Interventions are designed to promote responsible behavior and decrease irresponsible behavior among family members. The therapist assists parents and youths in behaving in a responsible manner across a variety of domains. Parental responsibilities include providing support, guidance, and discipline; expressing love and nurturance; protection; advocacy; and meeting basic physical needs. The child and adolescent's primary responsibilities include complying with family and societal rules, attending school and putting forth reasonable effort, helping around the house, and not harming self or others. Therapists will spend a great deal of time throughout the treatment process enhancing, developing, and maintaining the responsible behaviors of parents through praise and support. Other family members who become engaged in the treatment process are also encouraged by the therapist to reinforce responsible parental behaviors that will help maintain these behaviors when treatment ends. It has been noted that when parents increase their responsibilities, there is almost always improvement in the child's behavior. Parental abdication of responsibilities may be caused by factors such as mental illness or the lack of necessary parenting skills. Interventions would be designed to address these influences. For children and adolescents, positive **reinforcement** and discipline are used to increase responsible behaviors and decrease irresponsible behaviors. Parents are encouraged to spell out clearly expectations and punishment for **compliance** and noncompliance before putting them into action. For example, the child should know ahead of time that missing curfew will result in being grounded for a week. Parents are also taught to praise often for compliant behaviors.
- Interventions are focused on the present and are action oriented, targeting specific and well-defined problems. Due to time limitations of the MST model, family members are required to work intensely to solve often long-standing problems. Once information has been gathered and assessed, therapist and family jointly formalize problem and goals into a treatment plan. The plan specifies which changes in what behavior or skill will be achieved by whom, by what method or action, and in what period of time within the limits of the program. The treatment plan contains the family's overarching goals or ultimate aims that are to be accomplished by the end of the treatment period, and intermediate goals or incremental steps needed to reach the overarching goal. These intermediate goals are measurable and time limited and the interventions chosen are those that have been determined to have the most immediate and powerful impact on the problem behavior. The therapist assists families in meeting their specific goals by helping them focus their time, energy, and resources on their assignments. Also, the expected outcome of each intervention is described in observable and measurable terms before the treatment plan is put into action. This aids the MST staff and the family to determine whether the interventions are effective or if alternatives are needed.
- Interventions should target sequences of behavior within and between multiple systems that maintain the identified problem. As an example, an ineffective parenting style (permissive, authoritarian, neglectful) may be identified as a factor in influencing the problem behavior and is, therefore, targeted for an intervention. However, the parents are having marital difficulties that lead to disagreements in child rearing practices; these difficulties are maintaining the poor parenting style and will be the focus of an intervention as well. In addition, the family may have some practical or concrete needs (housing, heat, transportation) that are, in turn, having an impact on parental discipline and require interventions across the family—community support system that is also a factor in maintaining the identified problem.
- Interventions are developmentally appropriate and fit the developmental needs of the youth. The nature of the intervention should take into account the age and maturity of the child or adolescent and the caregiver. It is noted that, for children and young adolescents, interventions aimed at increasing parental control are the most appropriate. Such interventions might include introducing systematic monitoring, reward, and discipline systems. For an older adolescent, interventions would most likely focus on preparing the youth for entry into the adult world, such as increasing his or her social maturity. Other interventions may be needed to overcome obstacles to independent living, such as having the teenager participate in GED classes or enter a vocational training school. The developmental stage of the caregiver is also important to consider. For example, grandparents may not have the physical or emotional health to become primary caregivers but may be able to assist parents in other ways, such as helping with homework or sitting with the youth after school for a few hours.

- Interventions are designed to require daily or weekly effort by family members. This leads to a more rapid decrease in the problem behavior, and current and continuous evaluation of whether the intervention is working and producing the expected results. For example, if a parent sits near the child while he or she is doing homework, progress toward the anticipated goal of better school performance is gauged. This design also allows family members to experience immediate success and obtain positive feedback.
- Intervention effectiveness is evaluated continuously from multiple perspectives with providers assuming accountability for overcoming barriers to successful outcomes. To assess the impact of an intervention, before intervention is implemented the therapist is required to document anticipated outcomes for each intervention by describing the observable and measurable outcome that he or she is aiming for. This information is used to assess the successes made or barriers encountered during treatment. The MST staff may also be in daily contact with teachers and administrators, deputy juvenile officers, and welfare professionals who provide feedback regarding whether the interventions across systems are successful in changing behaviors.
- Interventions are designed to promote treatment generalization and long-term maintenance of therapeutic change by empowering caregivers to address family members' needs across multiple systemic contexts. The MST therapist, the MST team, and the provider agency are responsible for engaging the family in treatment, making services for the family easier to obtain, and achieving positive outcomes for the child or adolescent and the family in every case. The program's achievement of successful goals and maintenance of behavior change is due to staff adherence to the treatment model. Research has demonstrated that strong adherence correlates to strong case outcomes. The key to the success of the model is intensive and ongoing staff training. Clinical staff training includes five days of orientation training, weekly supervision with an MST expert, and quarterly booster training. On-site supervisors are also intensively trained to ensure that the MST staff adhere to the MST model.

Normal results

At the end of MST treatment, parents have been provided with the resources needed to parent effectively and maintain better family structure and cohesion. Specifically, parents:

- are able to monitor their child(ren) or adolescent's behaviors systematically
- have learned to use appropriate reward and discipline measures to maintain new behavioral changes
- can communicate more effectively with each other and their children
- can advocate for their children and themselves across social systems (school, social services)
- can problem-solve daily conflicts
- can maintain positive relations with natural social supports such as extended family, friends, and church members
- are able to maintain a positive working relationship with school personnel
- have learned strategies to monitor and promote the child's or adolescent's school performance and/or vocational functioning

Other outcomes to be expected have to do with the youth's relationships with peers and his or her performance in school. Specifically, it is expected that the child or adolescent has decreased his or her association with delinquent and/or drug-using peers; has increased his or her relationships with positive peers and engages in positive activities through after-school activities, organized athletics, or volunteer or paid activities; has better school performance; and has had no, or has decreased, days requiring out-of-home placement.

See also Antisocial personality disorder; Cognitive-behavioral therapy; Community mental health; Family education; Family psychoeducation; Family therapy

Resources

BOOKS

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Henggeler, Scott W., and others. *Multisystemic Treatment of Antisocial Behavior in children and Adolescents.* New York: The Guilford Press, 1998.

ORGANIZATIONS

National Institute of Mental Health. 6001 Executive Bloulevard, RM.8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. Fax: (301) 443-4279. TTY: (301) 443-8431. <http://www.nimh.nih.gov>.

Office of Juvenile Justice and Delinquency Prevention. 810 Seventh Street NW, Washington, D.C. 20531. (202) 307-5911. Fax: (202) 307-2093. <http://www.ojjdp.ncjrs.org>.

OTHER

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Munchausen syndrome *see* **Factitious disorder**

Music therapy *see* **Creative therapies**

Mutual support *see* **Support groups**

N

Naltrexone

Definition

Naltrexone is classified as a pure opiate antagonist. It is sold in the United States under the brand names ReVia and Depade, but is also manufactured and sold under its generic name.

Purpose

Naltrexone is used as part of medically supervised **behavior modification** programs in order to maintain a patient previously addicted to opiates in an opiate-free state following successful opiate **detoxification**. Naltrexone is also used in the management of alcohol dependence and abstinence in combination with medically supervised behavior modification programs.

Description

Opiates are a group of drugs that are either derived from opium (i.e. morphine, hydromorphone, oxymorphone, heroin, codeine, hydrocodone, oxycodone) or chemically resemble these opium derivatives (such as meperidine). They are commonly referred to as narcotics. Some opiates have medically valid uses, while others are recreational drugs of abuse. All are physically addictive.

The drug naltrexone is an opiate antagonist. This means that it blocks and reverses the physical effects of drugs such as morphine, hydromorphone, oxymorphone, heroin, meperidine, codeine, hydrocodone, oxycodone and other drugs classified as narcotics. When given to patients who have been successfully treated for opiate **addiction**, it not only decreases craving for these types of drugs, it also prevents patients who use opiates while taking naltrexone from experiencing the euphoria associated with their use. In these two ways, naltrexone helps prevent re-addiction to opiates.

Chemically, naltrexone is not an alcohol antagonist. However, when it is used in combination with behavior modification in the recovering alcoholic, naltrexone decreases the craving for alcohol. This helps to prevent a return to alcohol use, or it decreases the severity of relapse by reducing the amount of alcohol consumed during the relapse or decreasing the length of the relapse.

Naltrexone is available in 50-mg oral tablets.

Recommended dosage

After a person has been successfully detoxified from opiates, he or she will receive a test dose of 25 mg of naltrexone, then be observed for one hour for symptoms of opiate withdrawal. If no problems occur after this test dose, another 25 mg test dose is administered.

Getting a person to comply with treatment for opiate addiction is the single most important aspect in maintaining an opiate-free state. Different schedules for taking naltrexone have been developed to help meet the needs of individuals in order to make taking the drug easier. Following successful initiation of therapy, naltrexone may be administered in one of the following ways:

- 50 mg daily Monday through Friday and 100 mg Saturday
- 100 mg every other day
- 150 mg every third day
- 100 mg on Monday and Wednesday and 150 mg on Friday
- 150 mg on Monday and 200 mg on Thursday

The duration of treatment with naltrexone for opiate dependence varies with patient need, although most patients will require at least six months of treatment.

The usual dose of naltrexone for alcohol dependence is 50 mg daily, although a few patients may require only 25 mg daily. The proper duration of therapy is not known, as studies of the use of naltrexone in alcohol dependence did not go beyond 12 weeks.

KEY TERMS

Antagonist—A substance whose actions counteract the effects of or work in the opposite way from another chemical or drug.

Opiates—A class of drugs that is either derived from opium (i.e. morphine, hydromorphone, oxymorphone, heroin, codeine, hydrocodone, oxycodone) or resembles these opium derivatives (such as meperidine) and is commonly referred to as narcotics.

Precautions

In a very small number of patients, naltrexone may be toxic and cause damage to the liver. Before starting naltrexone and throughout treatment, patients should receive monthly liver function tests to assess the drug's effect on the liver.

Patients should be free of all opiates for seven to 10 days before starting naltrexone. Naltrexone may cause opiate withdrawal symptoms in people whose bodies are not free from opiates. Patients should be observed for opiate withdrawal immediately following the first dose of the drug.

Patients may have a false sense of security that the presence of naltrexone in their system makes them immune from the effects of opiates. In fact, the opiate antagonism caused by naltrexone is not absolute and patients can still experience both analgesia (suppression of pain) and euphoria by administration of larger-than-normal amounts of opiates. Consequently, patients receiving naltrexone who continue to use or receive opiates may take larger doses and should be monitored for signs and symptoms of opiate overdose.

Side effects

The following represents the most common side effects associated with naltrexone:

- nausea, vomiting, diarrhea, cramps
- headache, **insomnia**, anxiety, irritability, depression, dizziness
- joint and muscle pain
- rash

Interactions

Because naltrexone is an opiate antagonist, opiate derivatives that are used for medicinally in treating cough, diarrhea, and pain may no longer be effective.

The combination of naltrexone and **disulfiram**, a drug that is also used for alcohol abuse, may cause increased liver toxicity and liver damage when taken together. This combination should be avoided unless in consultation with a physician, it is decided that the potential benefits of this combination outweigh the risks.

Resources

BOOKS

American Society of Health-System Pharmacists. *AHFS Drug Information 2002.* Bethesda: American Society of Health-System Pharmacists, 2002.

O'Brien, Charles P. "Drug Addiction and Drug Abuse." In *Goodman & Gillman's The Pharmacological Basis of Therapeutics Tenth Edition* edited by Joel G. Hardman, Ph.D. and Lee E. Limbird, Ph.D. New York: McGraw-Hill, 2001.

Jack Raber, Pharm.D.

Narcissistic personality disorder

Definition

Narcissistic personality disorder (NPD) is defined by the Fourth Edition Text Revision of the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*, a handbook that mental health professionals use to diagnose mental disorders) as one of ten **personality disorders**. As a group, these disorders are described by *DSM-IV-TR* as "enduring pattern[s] of inner experience and behavior" that are sufficiently rigid and deep-seated to bring a person into repeated conflicts with his or her social and occupational environment. *DSM-IV-TR* specifies that these dysfunctional patterns must be regarded as nonconforming or deviant by the person's culture, and cause significant emotional pain and/or difficulties in relationships and occupational performance.

To meet the **diagnosis** of a personality disorder, the patient's problematic behaviors must appear in two or more of the following areas:

- perception and interpretation of the self and other people
- intensity and duration of feelings and their appropriateness to situations
- relationships with others
- ability to control impulses

It is important to note that all the personality disorders are considered to have their onset in late adolescence or early adulthood. Doctors rarely give a diagnosis of personality disorder to children on the grounds that children's personalities are still in process of formation and may change considerably by the time they are in their late teens.

NPD is defined more specifically as a pattern of grandiosity (exaggerated claims to talents, importance, or specialness) in the patient's private fantasies or outward behavior; a need for constant admiration from others; and a lack of empathy for others. The term *narcissistic* is derived from an ancient Greek legend, the story of Echo and Narcissus. According to the legend, Echo was a woodland nymph who fell in love with Narcissus, who was an uncommonly handsome but also uncommonly vain young man. He contemptuously rejected her expressions of love. She pined away and died. The god Apollo was angered by Narcissus' pride and self-satisfaction, and condemned him to die without ever knowing human love. One day, Narcissus was feeling thirsty, saw a pool of clear water nearby, and knelt beside it in order to dip his hands in the water and drink. He saw his face reflected on the surface of the water and fell in love with the reflection. Unable to win a response from the image in the water, Narcissus eventually died beside the pool.

Havelock Ellis, a British **psychologist**, first used the story of Echo and Narcissus in 1898 as a capsule summary of pathological self-absorption. The words *narcissist* and *narcissistic* have been part of the vocabulary of psychology and psychiatry ever since. They have, however, been the subjects of several controversies. In order to understand NPD, the reader may find it helpful to have an outline of the different theories about narcissism in human beings, its relation to other psychiatric disorders, and its connections to the wider culture. NPD is unique among the *DSM-IV-TR* personality disorders in that it has been made into a symbol of the problems and discontents of contemporary Western culture as a whole.

KEY TERMS

Grandiosity—Exaggerated and unrealistic self-importance; inflated self-assessment. Grandiosity is considered one of the core characteristics of persons diagnosed with NPD.

Macrosocial—Pertaining to the wider society, as distinct from such smaller social groupings as families, neighborhoods, etc.

Narcissistic Personality Inventory (NPI)—The most widely used English-language diagnostic instrument for narcissistic personality disorder. Based on the *DSM-III* criteria for NPD, the NPI is frequently used in research studies as well as patient assessment.

Primary narcissism—Sigmund Freud's term for a normal phase in early childhood development in which the infant has not yet learned to distinguish between itself and its world, and sees other people and things in its environment as extensions of itself.

Projection—A psychological process in which a person unconsciously attributes unacceptable feelings to someone else. Narcissists often project their envy onto other people, claiming that the person in question is envious of them.

Splitting—A psychological process that occurs during the childhood of a person with NPD, in which the child separates aspects of him- or herself that the parents value from those that they disregard.

Superego—According to Freud, the part of the mind that represents traditional parental and societal values. The superego is the source of guilt feelings.

Description

A good place to begin a discussion of the different theories about narcissism is with the observation that NPD exists as a diagnostic category only in *DSM-IV-TR*, which is an American diagnostic manual. The *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision* (*ICD-10*, the European equivalent of *DSM*) lists only eight personality disorders. What *DSM-IV-TR* defines as narcissistic personality disorder, *ICD-10* lumps together with "eccentric, impulsive-type, immature, passive-aggressive, and psychoneurotic personality disorders."

DSM-IV-TR specifies nine diagnostic criteria for NPD. For the clinician to make the diagnosis, an individual must fit five or more of the following descriptions:

- He or she has a grandiose sense of self-importance (exaggerates accomplishments and demands to be considered superior without real evidence of achievement).
- He or she lives in a dream world of exceptional success, power, beauty, genius, or "perfect" love.
- He or she thinks of him- or herself as "special" or privileged, and that he or she can only be understood by other special or high-status people.

- He or she demands excessive amounts of praise or admiration from others.
- He or she feels entitled to automatic deference, **compliance**, or favorable treatment from others.
- He or she is exploitative towards others and takes advantage of them.
- He or she lacks empathy and does not recognize or identify with others' feelings.
- He or she is frequently envious of others or thinks that they are envious of him or her.
- He or she "has an attitude" or frequently acts in haughty or arrogant ways.

In addition to these criteria, *DSM-IV-TR* groups NPD together with three other personality disorders in its so-called Cluster B. These four disorders are grouped together on the basis of symptom similarities, insofar as patients with these disorders appear to others as overly emotional, unstable, or self-dramatizing. The other three disorders in Cluster B are antisocial, borderline, and histrionic personality disorders.

The *DSM-IV-TR* clustering system does not mean that all patients can be fitted neatly into one of the three clusters. It is possible for patients to have symptoms of more than one personality disorder or to have symptoms from different clusters. In addition, patients diagnosed with any personality disorder may also meet the criteria for mood, substance abuse, or other disorders.

Subtypes of NPD

AGE GROUP SUBTYPES. Ever since the 1950s, when psychiatrists began to notice an increase in the number of their patients that had narcissistic disorders, they have made attempts to define these disorders more precisely. NPD was introduced as a new diagnostic category in *DSM-III*, which was published in 1980. Prior to *DSM-III*, narcissism was a recognized phenomenon but not an official diagnosis. At that time, NPD was considered virtually untreatable because people who suffer from it rarely enter or remain in treatment; typically, they regard themselves as superior to their therapist, and they see their problems as caused by other people's "stupidity" or "lack of appreciation." More recently, however, some psychiatrists have proposed dividing narcissistic patients into two subcategories based roughly on age: those who suffer from the stable form of NPD described by *DSM-IV-TR*, and younger adults whose narcissism is often corrected by life experiences.

This age group distinction represents an ongoing controversy about the nature of NPD—whether it is fundamentally a character disorder, or whether it is a matter of learned behavior that can be unlearned. Therapists who incline toward the first viewpoint are usually pessimistic about the results of treatment for patients with NPD.

PERSONALITY SUBTYPES. Other psychiatrists have noted that patients who meet the *DSM-IV-TR* criteria for NPD reflect different clusters of traits within the *DSM-IV-TR* list. One expert in the field of NPD has suggested the following subcategories of narcissistic personalities:

- Craving narcissists. These are people who feel emotionally needy and undernourished, and may well appear clingy or demanding to those around them.
- Paranoid narcissists. This type of narcissist feels intense contempt for him- or herself, but projects it outward onto others. Paranoid narcissists frequently drive other people away from them by hypercritical and jealous comments and behaviors.
- Manipulative narcissists. These people enjoy "putting something over" on others, obtaining their feelings of superiority by lying to and manipulating them.
- Phallic narcissists. Almost all narcissists in this subgroup are male. They tend to be aggressive, athletic, and exhibitionistic; they enjoy showing off their bodies, clothes, and overall "manliness."

Causes and symptoms

Causes

At present there are two major theories about the origin and nature of NPD. One theory regards NPD as a form of arrested psychological development while the other regards it as a young child's defense against psychological pain. The two perspectives have been identified with two major figures in psychoanalytic thought, Heinz Kohut and Otto Kernberg respectively.

Both theories about NPD go back to Sigmund Freud's pioneering work *On Narcissism,* published in 1914. In this essay, Freud introduced a distinction which has been retained by almost all later writers—namely, the distinction between primary and secondary narcissism. Freud thought that all human infants pass through a phase of primary narcissism, in which they assume they are the center of their universe. This phase ends when the baby is forced by the realities of life to recognize that it does not control its parents (or other caregivers) but is in fact entirely dependent on them. In normal circumstances, the baby gives up its fantasy of being all-powerful and becomes emotionally attached to its parents rather than itself. What Freud defined as secondary narcissism is a pathological condition in which the infant does not invest its emotions in its parents but rather redirects them back to itself. He thought that secondary narcissism developed

in what he termed the pre-Oedipal phase of childhood; that is, before the age of three. From a Freudian perspective, then, narcissistic disorders originate in very early childhood development, and this early origin is thought to explain why they are so difficult to treat in later life.

CAUSES IN THE FAMILY OF ORIGIN. Kohut and Kernberg agree with Freud in tracing the roots of NPD to disturbances in the patient's family of origin—specifically, to problems in the parent-child relationship before the child turned three. Where they disagree is in their accounts of the nature of these problems. According to Kohut, the child grows out of primary narcissism through opportunities to be mirrored by (i.e., gain approval from) his or her parents and to idealize them, acquiring a more realistic sense of self and a set of personal ideals and values through these two processes. On the other hand, if the parents fail to provide appropriate opportunities for idealization and mirroring, the child remains "stuck" at a developmental stage in which his or her sense of self remains grandiose and unrealistic while at the same time he or she remains dependent on approval from others for self-esteem.

In contrast, Kernberg views NPD as rooted in the child's defense against a cold and unempathetic parent, usually the mother. Emotionally hungry and angry at the depriving parents, the child withdraws into a part of the self that the parents value, whether looks, intellectual ability, or some other skill or talent. This part of the self becomes hyperinflated and grandiose. Any perceived weaknesses are "split off" into a hidden part of the self. Splitting gives rise to a lifelong tendency to swing between extremes of grandiosity and feelings of emptiness and worthlessness.

In both accounts, the child emerges into adult life with a history of unsatisfactory relationships with others. The adult narcissist possesses a grandiose view of the self but has a conflict-ridden psychological dependence on others. At present, however, psychiatrists do not agree in their description of the central defect in NPD; some think that the problem is primarily emotional while others regard it as the result of distorted cognition, or knowing. Some maintain that the person with NPD has an "empty" or hungry sense of self while others argue that the narcissist has a "disorganized" self. Still others regard the core problem as the narcissist's inability to test reality and construct an accurate view of him- or herself.

MACROSOCIAL CAUSES. One dimension of NPD that must be taken into account is its social and historical context. Psychiatrists became interested in narcissism shortly after World War II (1939–45), when the older practitioners in the field noticed that their patient population had changed. Instead of seeing patients who suffered from obsessions and compulsions related to a harsh and punishing superego (the part of the psyche that internalizes the standards and moral demands of one's parents and culture), the psychiatrists were treating more patients with character disorders related to a weak sense of self. Instead of having a judgmental and overactive conscience, these patients had a weak or nonexistent code of morals. They were very different from the patients that Freud had treated, described, and analyzed. The younger generation of psychiatrists then began to interpret their patients' character disorders in terms of narcissism.

in the 1960s historians and social critics drew the attention of the general public to narcissism as a metaphorical description of Western culture in general. These writers saw several parallels between trends in the larger society and the personality traits of people diagnosed with narcissistic disorders. In short, they argued that the advanced industrial societies of Europe and the United States were contributing to the development of narcissistic disorders in individuals in a number of respects. Some of the trends they noted include the following:

- The mass media's preoccupation with "lifestyles of the rich and famous" rather than with ordinary or average people.
- Social approval of open displays of money, status, or accomplishments ("if you've got it, flaunt it") rather than modesty and self-restraint.
- Preference for a leadership style that emphasizes the leader's outward appearance and personality rather than his or her inner beliefs and values.
- The growth of large corporations and government bureaucracies that favor a managerial style based on "impression management" rather than objective measurements of performance.
- Social trends that encourage parents to be self-centered and to resent their children's legitimate needs.
- The weakening of churches, synagogues, and other religious or social institutions that traditionally helped children to see themselves as members of a community rather than as isolated individuals.

Although discussion continues about the location and forms of narcissism in the larger society, no one now denies that personality disorders both reflect and influence the culture in which they arise. Family therapists are now reporting on the treatment of families in which the children are replicating the narcissistic disorders of their parents.

Symptoms

Most observers regard grandiosity as the most important single trait of a narcissistic personality. It is

important to note that grandiosity implies more than boasting or prideful display as such—it signifies self-aggrandizement that is not borne out by reality. For example, a person who claims that he or she was the most valuable player on a college athletic team may be telling the truth about their undergraduate sports record. Their claim may be bad manners but is not grandiosity. On the other hand, someone who makes the same claim but had an undistinguished record or never even made the team is being grandiose. Grandiosity in NPD is related to some of the diagnostic criteria listed by *DSM-IV-TR*, such as demanding special favors from others or choosing friends and associates on the basis of prestige and high status rather than personal qualities. In addition, grandiosity complicates diagnostic assessment of narcissists because it frequently leads to lying and misrepresentation of one's past history and present accomplishments.

Other symptoms of NPD include:

- a history of intense but short-term relationships with others; inability to make or sustain genuinely intimate relationships
- a tendency to be attracted to leadership or high-profile positions or occupations
- a pattern of alternating between unrealistic idealization of others and equally unrealistic devaluation of them
- assessment of others in terms of usefulness
- a need to be the center of attention or admiration in a working group or social situation
- hypersensitivity to criticism, however mild, or rejection from others
- an unstable view of the self that fluctuates between extremes of self-praise and self-contempt
- preoccupation with outward appearance, "image," or public opinion rather than inner reality
- painful emotions based on shame (dislike of who one is) rather than guilt (regret for what one has done)

People diagnosed with NPD represent a range of levels of functioning. Otto Kernberg has described three levels of narcissistic impairment. At the top are those who are talented or gifted enough to attract all the admiration and attention that they want; these people may never enter therapy because they don't feel the need. On the second level are those who function satisfactorily in their jobs but seek professional help because they cannot form healthy relationships or because they feel generally bored and aimless. Narcissists on the lowest level have frequently been diagnosed with another mental disorder and/or have gotten into trouble with the law. They often have severe difficulties with anxiety and with controlling their impulses.

Demographics

DSM-IV-TR states that 2% to 16% of the clinical population and slightly less than 1% of the general population of the United States suffers from NPD. Between 50% and 75% of those diagnosed with NPD are males. Little is known about the prevalence of NPD across racial and ethnic groups.

Gender issues

The high preponderance of male patients in studies of narcissism has prompted researchers to explore the effects of gender roles on this particular personality disorder. Some have speculated that the gender imbalance in NPD results from society's disapproval of self-centered and exploitative behavior in women, who are typically socialized to nurture, please, and generally focus their attention on others. Others have remarked that the imbalance is more apparent than real, and that it reflects a basically sexist definition of narcissism. These researchers suggest that definitions of the disorder should be rewritten in future editions of *DSM* to account for ways in which narcissistic personality traits manifest differently in men and in women.

Professional and leadership positions

One important aspect of NPD that should be noted is that it does not prevent people from occupying, as well as aspiring to, positions of power, wealth, and prestige. Many people with NPD, as Kernberg's classification makes clear, are sufficiently talented to secure the credentials of success. In addition, narcissists' preoccupation with a well-packaged exterior means that they often develop an attractive and persuasive social manner. Many high-functioning narcissists are well liked by casual acquaintances and business associates who never get close enough to notice the emptiness or anger underneath the polished surface.

Unfortunately, narcissists in positions of high visibility or power—particularly in the so-called helping professions (medicine, education, and the ministry)—often do great harm to others. In recent years a number of books and articles have been published within the religious, medical, and business communities regarding the problems caused by professionals with NPD. One **psychiatrist** noted in a lecture on substance abuse among physicians that NPD is one of the three most common psychiatric diagnoses among physicians in court-mandated substance abuse programs. A psychologist who serves as a consultant in the evaluation of seminary students and ordained clergy has remarked that the proportion of narcissists in the clergy has risen dramatically since the 1960s. Researchers in the field of business organization and man-

agement styles have compiled data on the human and economic costs of executives with undiagnosed NPD.

Diagnosis

The diagnosis of NPD is complicated by a number of factors.

Complications of diagnosis

NPD is difficult to diagnose for several reasons. First, some people with NPD function sufficiently well that they do not come to the attention of therapists. Second, narcissists are prone to lie about themselves; thus it may take a long time for a therapist to notice discrepancies between a patient's version of his or her life and information gained from others or from public records. Third, many traits and behaviors associated with NPD may be attributed to other mental disorders. Low-functioning narcissists are often diagnosed as having **borderline personality disorder** (BPD), particularly if they are female; if they are male, they may be diagnosed as having **antisocial personality disorder** (ASPD). If the person with NPD has a substance abuse disorder, some of their narcissistic behaviors may be written off to the mood-altering substance. More recently, some psychiatrists have pointed to a tendency to confuse narcissistic behaviors in people with NPD who have had a traumatic experience with full-blown **post-traumatic stress disorder** (PTSD). Given the lack of clarity in the differential diagnosis of NPD, some therapists are calling for a fundamental revision of *DSM-IV-TR* definitions of the personality disorders.

An additional complication is posed by economic considerations. The coming of **managed care** has meant that third-party payers (insurance companies) prefer short-term **psychotherapy** that concentrates on a patient's acute problems rather than on underlying chronic issues. Since narcissists are reluctant to trust others or form genuine interpersonal bonds, there is a strong possibility that many therapists do not recognize NPD in patients that they are treating for only a few weeks or months.

Diagnostic interviews

Diagnosis of NPD is usually made on the basis of several sources of information: the patient's history and self-description, information from family members and others, and the results of diagnostic questionnaires. One questionnaire that is often used in the process of differential diagnosis is the Structured Clinical Interview for *DSM-III-R* Disorders, known as the SCID-II.

The most common diagnostic instrument used for narcissistic NPD is the Narcissistic Personality Inventory (NPI). First published by Robert R. Raskin and Calvin S. Hall in 1979, the NPI consists of 223 items consisting of paired statements, one reflecting narcissistic traits and the other nonnarcissistic. Subjects are required to choose one of the two items. The NPI is widely used in research as well as diagnostic assessment.

Treatments

Treatments for NPD include a variety of pharmacologic, individual, and group approaches; none, however, have been shown to be particularly effective as of 2002.

Medication

As of 2002, there are no medications that have been developed specifically for the treatment of NPD. Patients with NPD who are also depressed or anxious may be given drugs for relief of those symptoms. There are anecdotal reports in the medical literature that the selective serotonin reuptake inhibitors, or SSRIs, which are frequently prescribed for depression, reinforce narcissistic grandiosity and lack of empathy with others.

Psychotherapy

Several different approaches to individual therapy have been tried with NPD patients, ranging from classical **psychoanalysis** and Adlerian therapy to rational-emotive approaches and **Gestalt therapy**. The consensus that has emerged is that therapists should set modest goals for treatment with NPD patients. Most of them cannot form a sufficiently deep bond with a therapist to allow healing of early-childhood injuries. In addition, the tendency of these patients to criticize and devalue their therapists (as well as other authority figures) makes it difficult for therapists to work with them.

An additional factor that complicates psychotherapy with NPD patients is the lack of agreement among psychiatrists about the causes and course of the disorder. One researcher has commented that much more research is necessary to validate *DSM-IV-TR*'s description of NPD before outcome studies can be done comparing different techniques of treatment.

Hospitalization

Low-functioning patients with NPD may require inpatient treatment, particularly those with severe self-harming behaviors or lack of impulse control. Hospital treatment, however, appears to be most helpful when it is focused on the immediate crisis and its symptoms rather than the patient's underlying long-term difficulties.

Prognosis

The prognosis for younger persons with narcissistic disorders is hopeful to the extent that the disturbances reflect a simple lack of life experience. The outlook for long-standing NPD, however, is largely negative. Some narcissists are able, particularly as they approach their midlife years, to accept their own limitations and those of others, to resolve their problems with envy, and to accept their own mortality. Most patients with NPD, on the other hand, become increasingly depressed as they grow older within a youth-oriented culture and lose their looks and overall vitality. The retirement years are especially painful for patients with NPD because they must yield their positions in the working world to the next generation. In addition, they do not have the network of intimate family ties and friendships that sustain most older people.

Prevention

The best hope for prevention of NPD lies with parents and other caregivers who are close to children during the early preschool years. Parents must be able to demonstrate empathy in their interactions with the child and with each other. They must also be able to show that they love their children for who they are, not for their appearance or their achievements. And they must focus their parenting efforts on meeting the child's changing needs as he or she matures, rather than demanding that the child meet their needs for status, comfort, or convenience.

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Narcolepsy

Definition

Narcolepsy is a disorder marked by excessive daytime sleepiness, uncontrollable sleep attacks, and cataplexy (a sudden loss of muscle tone, usually lasting up to half an hour).

Description

Narcolepsy is the second-leading cause of excessive daytime sleepiness (after obstructive sleep apnea). Persistent sleepiness and sleep attacks are the hallmarks of this condition. The sleepiness has been compared to the feeling of trying to stay awake after not sleeping for two or three days.

People with narcolepsy fall asleep suddenly—anywhere, at any time, even in the middle of a conversation. These sleep attacks can last from a few seconds to more than an hour. Depending on where the sleep attacks occur, they may be mildly inconvenient or even dangerous to the person, particularly if they occur while he or she is driving. Some people continue to function outwardly during the sleep episodes, such as continuing a conversation or putting things away. But when they wake up, they have no memory of the event.

Sleep researchers have identified several different types of sleep in humans. One type of sleep is called rapid eye movement (REM) sleep, because the person's eyes move rapidly back and forth underneath the closed eyelids. REM sleep is associated with dreaming. Normally, when people fall asleep, they experience 90 minutes of non-REM sleep, which is then followed by a phase of REM sleep. People with narcolepsy, however, enter REM sleep immediately. In addition, REM sleep occurs inappropriately in patients with narcolepsy throughout the day.

KEY TERMS

Cataplexy—A symptom of narcolepsy marked by a sudden episode of muscle weakness triggered by strong emotions. The muscle weakness may cause the person's knees to buckle, or the head to drop. In severe cases, the patient may become paralyzed for a few seconds to minutes.

Hypnagogic hallucinations—Dream-like auditory or visual hallucinations that occur while a person is falling asleep.

Hypothalamus—A part of the forebrain that controls heartbeat, body temperature, thirst, hunger, blood pressure, blood sugar levels, and other functions.

Polysomnogram—A machine that is used to diagnose sleep disorders by measuring and recording a variety of body functions related to sleep, including heart rate, eye movements, brain waves, muscle activity, breathing, changes in blood oxygen concentration, and body position.

Rapid eye movement (REM) sleep—A type of sleep during which the person's eyes move back and forth rapidly underneath their closed eyelids. REM sleep is associated with dreaming.

Sleep paralysis—An abnormal episode of sleep in which the patient cannot move for a few minutes, usually occurring while falling asleep or waking up. Sleep paralysis is often found in patients with narcolepsy.

Causes and symptoms

Causes

One of the causes of narcolepsy is a genetic mutation. In 1999 researchers identified the gene that causes the disorder. The narcolepsy gene allows cells in the hypothalamus (the part of the **brain** that regulates sleep behavior) to receive messages from other cells. As a result of the mutation, the cells cannot communicate properly, and abnormal sleeping patterns develop.

Other researchers are also looking into the possibility that narcolepsy may be caused by some kind of autoimmune disorder. This theory suggests that the person's immune system accidentally turns against the specific area of the brain that controls alertness and sleep, injuring or destroying it.

The disorder sometimes runs in families, but most people with narcolepsy have no relatives with the disorder. Researchers believe that the inheritance of narcolepsy is similar to that of heart disease. In heart disease, several genes play a role in being susceptible to the disorder, but it does not usually develop without an environmental trigger of some sort.

Symptoms

While the symptoms of narcolepsy usually appear during a person's late teens or early 20s, the disease may not be diagnosed for many years. Most often, the first symptom is an overwhelming feeling of **fatigue**. After several months or years, cataplexy and other symptoms of the disorder appear.

Cataplexy is the most dramatic symptom of narcolepsy, affecting 75% of people with the disorder. During attacks, the knees buckle and the neck muscles go slack. In extreme cases, the person may become paralyzed and fall to the floor. This loss of muscle tone is temporary, lasting from a few seconds to half an hour, but it is frightening. The attacks can occur at any time but are often triggered by such strong emotions as anger, joy, or surprise.

Other symptoms of narcolepsy include:

- sleep attacks: short, uncontrollable sleep episodes throughout the day
- sleep paralysis: a frightening inability to move shortly after awakening or dozing off

- auditory or visual **hallucinations**: intense, sometimes terrifying experiences at the beginning or end of a sleep period
- disturbed nighttime sleep: tossing and turning, nightmares, and frequent awakenings during the night

Demographics

There has been debate over the incidence of narcolepsy. It is thought to affect between one in every 1,000 to 2,000 Americans. The known prevalence in other countries varies, from one in 600 in Japan to one in 500,000 in Israel. The reasons for these demographic differences are not clear. In about 8–12% of cases, people diagnosed with narcolepsy know of other family members with similar symptoms.

Diagnosis

The **diagnosis** of narcolepsy can be made by a general practitioner familiar with the disorder as well as by a **psychiatrist**. If a person comes to the doctor with reports of both excessive daytime sleepiness and cataplexy, a diagnosis may be made on the patient's history alone. Laboratory tests, however, can confirm a diagnosis of narcolepsy. These tests may include an overnight polysomnogram— a test in which sleep is monitored with a variety of electrodes that record information about heart rate, eye movements, brain waves, muscle activity, breathing, changes in blood oxygen concentration, and body position. A Multiple Sleep Latency Test, which measures sleep latency (onset) and how quickly REM sleep occurs, may also be used. People who have narcolepsy usually fall asleep in less than five minutes.

If the diagnosis is still open to question, a genetic blood test can reveal the existence of certain substances in people who have a tendency to develop narcolepsy. Positive test results suggest, but do not prove, that the patient has narcolepsy.

Narcolepsy is a complex disorder, and it is often misdiagnosed. Many people with the disorder struggle with symptoms for an average of 14 years before being correctly diagnosed.

Treatment

There is no cure for narcolepsy. It is not progressive, and it is not fatal, but it is a chronic disorder. The symptoms can be managed with lifestyle adjustments and/or medication.

People with narcolepsy must plan their days carefully. Scheduling regular naps (either several short, fifteen-minute naps or one long nap in the afternoon) can help boost alertness and awakeness. A full eight hours of nighttime sleep should also be a goal. Exercise can often help people with narcolepsy feel more alert and energetic, although they should avoid exercising within a few hours of bedtime. Substances that contain alcohol, nicotine, and caffeine should be avoided because they can interfere with refreshing sleep and with daytime alertness.

Medications for narcolepsy may include the use of antidepressants (tricyclic antidepressants or selective serotonin-reuptake inhibitors) to treat such symptoms of the disorder as cataplexy, hypnagogic hallucinations, and/or sleep paralysis.

Stimulants (**amphetamines**) may also be used to help individuals with narcolepsy stay awake and alert.

With the recent discovery of the gene that causes narcolepsy, researchers are hopeful that other treatments can be designed to relieve the symptoms of the disorder.

Prognosis

Narcolepsy is not a degenerative disease, and patients do not develop other neurologic symptoms. Narcolepsy can, however, interfere with a person's ability to work, play, drive, socialize, and perform other daily activities. In severe cases, the disorder prevents people from living a normal life, leading to depression and a loss of independence.

Prevention

As of 2002, narcolepsy is not a preventable disorder.

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Nardil *see* **Phenelzine**

Navane *see* **Thiothixene**

Nefazodone

Definition

Nefazodone is a prescription antidepressant. Nefazodone is available in the United States under the trade name of Serzone.

Purpose

Nefazodone is used to treat depression. It may be used to treat **major depressive disorder**, **dysthymic disorder**, and the depressed phase of **bipolar disorder**. As with all antidepressants, it may take several weeks before full beneficial effects are seen.

Description

Nefazodone was approved by the FDA in 1994. It is believed to increase the activities of some chemicals in the **brain**. By altering the activities of specific brain chemicals, nefazodone may reduce the chemical imbalances responsible for causing depression.

The drug is available as tablets in several different strengths, including 50-, 100-, 150-, 200-, and 250-mg tablets.

Nefazodone is broken down by the liver.

Recommended dosage

For most people, the recommended initial dose of nefazodone is 100 mg taken by mouth twice daily. The dose may be increased in 100 or 200 mg increments once a week. Most commonly, final dosages range between 300-600 mg taken by mouth each day.

It is recommended that the initial dose of nefazodone be lowered to 50 mg twice daily for elderly or debilitated individuals, because these individuals may be more sensitive to some of the drug's side effects.

Precautions

People who have a history of epilepsy or other seizure disorders, heart attack, **stroke**, high blood pressure, or mania may require close physician supervision while taking nefazodone. Nefazodone may increase the tendency to have **seizures**, and should be used carefully by people with epilepsy or other seizure disorders. Nefazodone may lower blood pressure. This effect may be most noticeable when rising suddenly from a lying or sitting position. People with a history of heart attack or stroke, those taking medications for high blood pressure, or people who are dehydrated may be most sensitive to this effect and may feel dizzy or faint when standing up suddenly. Nefazodone may alter moods or cause mania, so patients with a history of mania should use nefazodone with caution.

KEY TERMS

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Antipsychotic—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Depression—A mental state characterized by excessive sadness. Other symptoms include altered sleep patterns, thoughts of suicide, difficulty concentrating, agitation, lack of energy, and loss of enjoyment in activities that are usually pleasurable.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

In rare situations, men taking nefazodone may experience long, painful erections. If this occurs, a health care provider should be notified immediately.

Because there is an increased likelihood of **suicide** in depressed individuals, close supervision of those at high risk for suicide attempts is recommended. Nefazodone is not recommended for pregnant or breast-feeding women.

Side effects

The most common side effects that cause people to stop taking nefazodone are dizziness, difficulty sleeping, weakness, or agitation. Other common adverse effects are sleepiness, dry mouth, nausea, constipation, blurred vision, and confusion.

Other, less common adverse effects associated with nefazodone are headache, flu-like symptoms, low blood pressure, itching, rash, upset stomach, fluid retention, muscle aches, thirst, memory impairment, nerve pain, nightmares, difficulty walking, ringing in the ears, urinary difficulties, breast pain, or vaginal irritation.

It has recently been discovered that in rare situations, nefazodone causes liver failure. If nausea, stomach pains, yellowing of the skin or eyes, itching, or darkening of urine occurs while taking nefazodone, a health care professional should be consulted immediately.

Interactions

Use of nefazodone with antidepressants referred to as monoamine oxidase inhibitors (MAOIs) is strongly discouraged due to the potential for high fever, muscle stiffness, sudden muscle spasms, rapid changes in heart rate and blood pressure, and the possibility of death. In fact, there should be a lapse of at least 14 days between taking a monoamine oxidase inhibitor and nefazodone or at least seven days should pass if switching from nefazodone to a monoamine oxidase inhibitor. Some examples of MAOIs include **phenelzine** (Nardil) and **tranylcypromine** (Parnate).

Some other drugs such as **trazodone** (Desyrel) and sibutramine may also interact with nefazodone and cause a syndrome characterized by irritability, muscle stiffness, shivering, muscle spasms, and altered consciousness. If nefazodone is used with **buspirone** (BuSpar), the dosage of buspirone should be lowered to prevent adverse effects. Additionally, when nefazodone is used in combination with digoxin (Lanoxin), frequent monitoring of blood levels of digoxin is recommended to prevent toxicity.

Nefazodone should not be used with the drugs **triazolam** (Halcion) and **alprazolam** (Xanax) because the side effects of these drugs are likely to increase. Use of nefazodone should also be avoided with **carbamazepine** (Tegretol), because nefazodone is likely to lose its effectiveness.

It is best to avoid using nefazodone with **pimozide** (Orap) due to an increased tendency for severe and potentially life-threatening irregular heartbeats.

When used with gemfibrozil or other drugs that lower cholesterol levels, the risk of muscle pain and weakness may be increased.

Because nefazodone may cause drowsiness, it should be used carefully with other medications that also make people prone to sleepiness such as antidepressants, antipsychotics, antihistamines, and alcohol.

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Negative symptoms

Definition

Negative symptoms are thoughts, feelings, or behaviors normally present that are absent or diminished in a person with a mental disorder.

Description

Examples of negative symptoms are social withdrawal, **apathy** (decreased motivation), poverty of speech (brief replies), inability to experience pleasure (anhedonia), limited emotional expression, or defects in attention control. The term "negative symptoms" is specifically used for describing **schizophrenia**, but sometimes used more generally in reference to disorders such as depression or **dementia**. These symptoms may be associated with altered brainwave activity or **brain** damage. They can be more difficult to diagnose than **positive symptoms** (**hallucinations**, **delusions**, bizarre behavior, or formal thought disorder) because they represent a lesser degree of normal, desirable activity rather than the presence of undesirable or bizarre behavior. Side

effects of certain medications, demoralization (loss of positive emotions like hope or confidence usually as the result of situations in which one feels powerless), or a lack of stimulation in one's environment can also cause negative symptoms, so these possibilities must be ruled out before attributing the symptoms to a disorder.

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Neglect

Definition

Neglect occurs when a parent or other primary caretaker chooses not to fulfill their obligations to care for, provide for, or adequately supervise and monitor the activities of their child. Parental and caregiving obligations include the physical, emotional, and educational well-being of the child. Thus, neglect can also occur when the parent or caretaker does not seek adequate medical or dental care for the child. Another definition of neglect is when the parental figure does not provide sufficient food, clothing, or shelter.

Parents are also expected to provide for the emotional needs of the child. Thus, neglect can occur when parents abandon the child, or simply have no time to spend with the child, in essence leaving the child to raise himself. If the child is actually left without supervision, this certainly constitutes neglect as well.

The final feature of neglect includes educational neglect, which often occurs when one child is responsible for other children in the family. Shifting the responsibility of caring for younger children to another child in the family prevents the caregiving child from participating in age-appropriate activities for themselves, such as attending school. This is a relatively common situation that makes it difficult for the oldest—and perhaps all of the children—to attend school. Parental responsibility includes providing adequate guidance and supervision for the children to regularly attend school. Truancy is not only a problem for children, but may be part of the picture of neglect as well.

Effects of neglect

Consequences of neglect are generally cumulative, and often negatively affect the child's development. For example, poor nutrition has negative consequences on the child's physical and psychological development. If proper nutrients are not available at critical growth periods, the child's development will not follow the normal and usual pattern. Common physical and psychological reactions to neglect include stunted growth, chronic medical problems, inadequate bone and muscle growth, and lack of neurological development that negatively affects normal **brain** functioning and information processing. Processing problems may often make it difficult for children to understand directions, may negatively impact the child's ability to understand social relationships, or may make completion of some academic tasks impossible without assistance or **intervention** from others. Lack of adequate medical care may result in long-term health problems or impairments such as hearing loss from untreated ear infections.

Long-term mental health effects of neglect are inconsistent. Effects of neglect can range from chronic depression to difficulty with relationships; however, not all adults neglected as children will suffer from these results. Some individuals are more resilient than others and are able to move beyond the emotional neglect they may have experienced. Characteristics of resilient individuals include an optimistic or hopeful outlook on life, and feeling challenged rather than defeated by problems.

Factors associated with neglect

Although each family's situation is unique with regard to stressors and characteristics that might precipitate neglect, there are some general factors that have been associated with neglect of a child. These factors include characteristics of the parental figure, and socioeconomic status.

Parental figures who neglect may have been neglected or abused themselves. There is a tendency for parental figures that neglect their children to have low self-esteem, poor impulse control, and to experience anxiety or depression. Other factors associated with neglect often include inadequate information about child development, including age-appropriate expectations of what children may be able to do. The parents may also feel overwhelmed by parenting responsibilities, and feel negatively about the child's demands on them. Such parents may never have fully adopted the role of parent or the caregiving the parental role requires. Internal pressures often push the caregivers to take care of their own needs (perhaps inappropriately), while ignoring the needs of the child. Substance abuse is often associated with neglect, particularly for those parents who are more self-absorbed and focused on their needs rather than their child's. This characteristic is also consistent with the findings of other studies indicating that some neglectful parents have an inability to be empathic, or to understand the feelings and needs of others.

Although **abuse** may occur across all levels of income and education, neglect is more often associated with severe levels of poverty and lower educational level. The external stressors may feel more extreme in single parent families as well, leading to neglectful behavior. Even in families where the parent is attempting to provide for the children, absence due to multiple work demands may lead to a neglectful situation. Families that are disorganized and socially isolated are more likely to neglect the children in their care.

Unlike victims of abuse, there are few consistent characteristics associated with victims of neglect. Retrospective studies of adults neglected as children indicate that females are slightly less resilient to neglect than men.

Prevalence

The number of children nationwide who are harmed or endangered by neglect is greater than any type of abuse. Neglect is consistently reported in more than half of the substantiated reports of mistreatment handled by the authorities.

Prevention and treatment

Interventions are usually aimed at two levels: community prevention efforts and individual parenting skills. A community-based program that actually combines the two facets of intervention is the "Parents as Teachers" program, which is available through many local school districts throughout the nation and is free of charge. Benefits of the program include its accessibility—parents simply need to call for the free service—and the in-home interventions provided by the program. Although the program is not part of the social service network of agencies, the fact that workers go into the home replicates that aspect of caseworker interventions. The simple act of having a paraprofessional in one's home can reduce the likelihood of neglect. Specific interventions that further reduce the likelihood of neglect include focusing on the parent-child relationship, reviewing appropriate expectations for the child's behavior (based on child development principles), and teaching basic parenting skills.

Other treatment options are generally more formal, and may be initiated by a call from a mandated reporter with concerns about neglect. Mandated reporters include physicians, teachers, and counselors. Any of these professionals may make the initial call if neglect is suspected. Concerned individuals may also call social services to report suspected neglect. In these cases of forced treatment, parents may be less willing participants in treatment efforts aimed at behavioral change for themselves and their families. In other instances, the parent or child may already be in treatment, and the focus on reducing neglectful behaviors may be incorporated into the existing treatment relationship. Factors to focus on in formal treatment aimed at reducing the likelihood of neglect may include specific parenting skills, home visits to allow monitoring of the relationship, as well as other individual needs such as substance abuse treatment, or empathy skill training.

Treatment efforts for the child should include family counseling aimed at communication skills and appropriate expression of affection and emotion within the family. Assertiveness skills training may be helpful for older adolescents in asking for their perceived needs.

See also Assertiveness skills training; Family therapy

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Neurolinguistic programming *see* **Hypnotherapy**

Neurontin *see* **Gabapentin**

Neuropsychological testing

Definition

Clinical neuropsychology is a field with historical origins in both psychology and neurology. The primary activity of neuropsychologists is assessment of **brain** functioning through structured and systematic behavioral observation. Neuropsychological tests are designed to examine a variety of cognitive abilities, including speed of information processing, attention, memory, language, and executive functions, which are necessary for goal-directed behavior. By testing a range of cognitive abilities and examining patterns of performance in different cognitive areas, neuropsychologists can make inferences about

underlying brain function. Neuropsychological testing is an important component of the assessment and treatment of traumatic brain injury, **dementia**, neurological conditions, and psychiatric disorders. Neuropsychological testing is also an important tool for examining the effects of toxic substances and medical conditions on brain functioning.

KEY TERMS

Abstraction—Ability to think about concepts or ideas separate from specific examples.

Battery—A number of separate items (such as tests) used together. In psychology, a group or series of tests given with a common purpose, such as personality assessment or measurement of intelligence.

Executive functions—A set of cognitive abilities that control and regulate other abilities and behaviors. Necessary for goal-directed behavior, they include the ability to initiate and stop actions, to monitor and change behavior as needed, and to plan future behavior when faced with novel tasks and situations.

Hemisphere—One side of the brain, right or left.

Psychometric—Pertaining to testing and measurement of mental or psychological abilities. Psychometric tests convert an individual's psychological traits and attributes into a numerical estimation or evaluation.

Syndrome—A group of symptoms that together characterize a disease or disorder.

Description

As early as the seventeenth century, scientists theorized about associations between regions of the brain and specific functions. The French philosopher, Descartes, believed the human soul could be localized to a specific brain structure, the pineal gland. In the eighteenth century, Franz Gall advocated the theory that specific mental qualities such as spirituality or aggression were governed by discrete parts of the brain. In contrast, Pierre Flourens contended that the brain was an integrated system that governed cognitive functioning in a holistic manner. Later discoveries indicated that brain function is both localized and integrated. Paul Broca and Karl Wernicke furthered understanding of localization and integration of function when they reported the loss of language abilities in patients with lesions to two regions in the left hemisphere of the brain.

The modern field of neuropsychology emerged in the twentieth century, combining theories based on anatomical observations of neurology with the techniques of psychology, including objective observation of behavior and the use of statistical analysis to differentiate functional abilities and define impairment. The famous Soviet neuropsychologist Alexander Luria played a major role in defining neuropsychology as it is practiced today. Luria formulated two principle goals of neuropsychology: to localize brain lesions and analyze psychological activities arising from brain function through behavioral observation. American neuropsychologist Ralph Reitan emphasized the importance of using standardized psychometric tests to guide systematic observations of brain-behavior relationships.

Before the introduction of neuroimaging techniques like the **computed tomography** (CAT scan) and **magnetic resonance imaging** (MRI), the primary focus of neuropsychology was **diagnosis**. Since clinicians lacked non-surgical methods for directly observing brain lesions or structural abnormalities in living patients, neuropsychological testing was the only way to determine which part of the brain was affected in a given patient. Neuropsychological tests can identify syndromes associated with problems in a particular area of the brain. For instance, a patient who performs well on tests of attention, memory, and language, but poorly on tests that require visual spatial skills such as copying a complex geometric figure or making designs with colored blocks, may have dysfunction in the right parietal lobe, the region of the brain involved in complex processing of visual information. When a patient complains of problems with verbal communication after a **stroke**, separate tests that examine production and comprehension of language help neuropsychologists identify the location of the stroke in the left hemisphere. Neuropsychological tests can also be used as screening tests to see if more extensive diagnostic evaluation is appropriate. Neuropsychological screening of elderly people complaining of memory problems can help identify those at risk for dementia versus those experiencing normal age-related memory loss.

As neuropsychological testing came to play a less vital role in localization of brain dysfunction, clinical neuropsychologists found new uses for their skills and knowledge. By clarifying which cognitive abilities are impaired or preserved in patients with brain injury or illness, neuropsychologists can predict how well individuals will respond to different forms of treatment or rehabilitation. Although patterns of test scores illustrate profiles of cognitive strength and weakness, neuropsychologists can also learn a great deal about patients by

observing how they approach a particular test. For example, two patients can complete a test in very different ways yet obtain similar scores. One patient may work slowly and methodically, making no errors, while another rushes through the test, making several errors but quickly correcting them. Some individuals persevere despite repeated failure on a series of test items, while others refuse to continue after a few failures. These differences might not be apparent in test scores, but can help clinicians choose among rehabilitation and treatment approaches.

Performance on neuropsychological tests is usually evaluated through comparison to the average performance of large samples of normal individuals. Most tests include tables of these normal scores, often divided into groups based on demographic variables like age and education that appear to affect cognitive functioning. This allows individuals to be compared to appropriate peers.

The typical neuropsychological examination evaluates sensation and perception, gross and fine motor skills, basic and complex attention, visual spatial skills, receptive and productive language abilities, recall and recognition memory, and executive functions such as cognitive flexibility and abstraction. Motivation and personality are often assessed as well, particularly when clients are seeking financial compensation for injuries, or cognitive complaints are not typical of the associated injury or illness.

Some neuropsychologists prefer to use fixed test batteries like the **Halstead-Reitan Battery** or the Luria-Nebraska Battery for all patients. These batteries include tests of a wide range of cognitive functions, and those who advocate their use believe that all functions must be assessed in each patient in order to avoid diagnostic bias or failure to detect subtle problems. The more common approach today, however, is to use a flexible battery based on hypotheses generated through a clinical interview, observation of the patient, and review of medical records. While this approach is more prone to bias, it has the advantage of preventing unnecessary testing. Since patients often find neuropsychological testing stressful and fatiguing, and these factors can negatively influence performance, advocates of the flexible battery approach argue that tailoring test batteries to particular patients can provide more accurate information.

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Neurosis

Definition

Neurosis is a term generally used to describe a nonpsychotic mental illness which triggers feelings of distress and anxiety and impairs functioning.

Description

Origins

The word *neurosis* means "nerve disorder," and was first coined in the late eighteenth century by William Cullen, a Scottish physician. Cullen's concept of neurosis encompassed those nervous disorders and symptoms that do not have a clear organic cause. Sigmund Freud later used the term *anxiety neurosis* to describe mental illness or distress with extreme anxiety as a defining feature.

There is a difference of opinion over the clinical use of the term neurosis today. It is not generally used as a diagnostic category by American psychologists and psychiatrists any longer, and was removed from the American Psychiatric Association's ***Diagnostic and Statistical Manual of Mental Disorders*** in 1980 with the publication of the third edition (it last appeared as a diagnostic category in *DSM-II*). Some professionals use the term to describe anxious symptoms and associated behavior, or to describe the range of mental illnesses outside of the psychotic disorders (such as **schizophrenia**, **delusional disorder**). Others, particularly psychoanalysts (psychiatrists and psychologists who follow a psy-

choanalytical model of treatment, as popularized by Freud and Carl Jung), use the term neurosis to describe the internal process itself (called an unconscious conflict) that triggers the anxiety characteristic.

Categories

The neurotic disorders are distinct from psychotic disorders in that the individual with neurotic symptoms has a firm grip on reality, and the psychotic patient does not. Before their reclassification, there were several major traditional categories of psychological neuroses, including: anxiety neurosis, depressive neurosis, obsessive-compulsive neurosis, somatization, **post-traumatic stress disorder**, and compensation neurosis—not a true neurosis, but a form of **malingering**, or feigning psychological symptoms for monetary or other personal gain.

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KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Catecholamine—A group of neurotransmitters synthesized from the amino acid tyrosine and released by the hypothalamic-pituitary-adrenal system in the brain in response to acute stress. The catecholamines include dopamine, serotonin, norepinephrine, and epinephrine.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

GABA—Gamma-aminobutyric acid, an inhibitory neurotransmitter in the brain.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Synaptic cleft—An area between nerve cells which can contain neurotransmitters.

Neurotransmitters

Definition

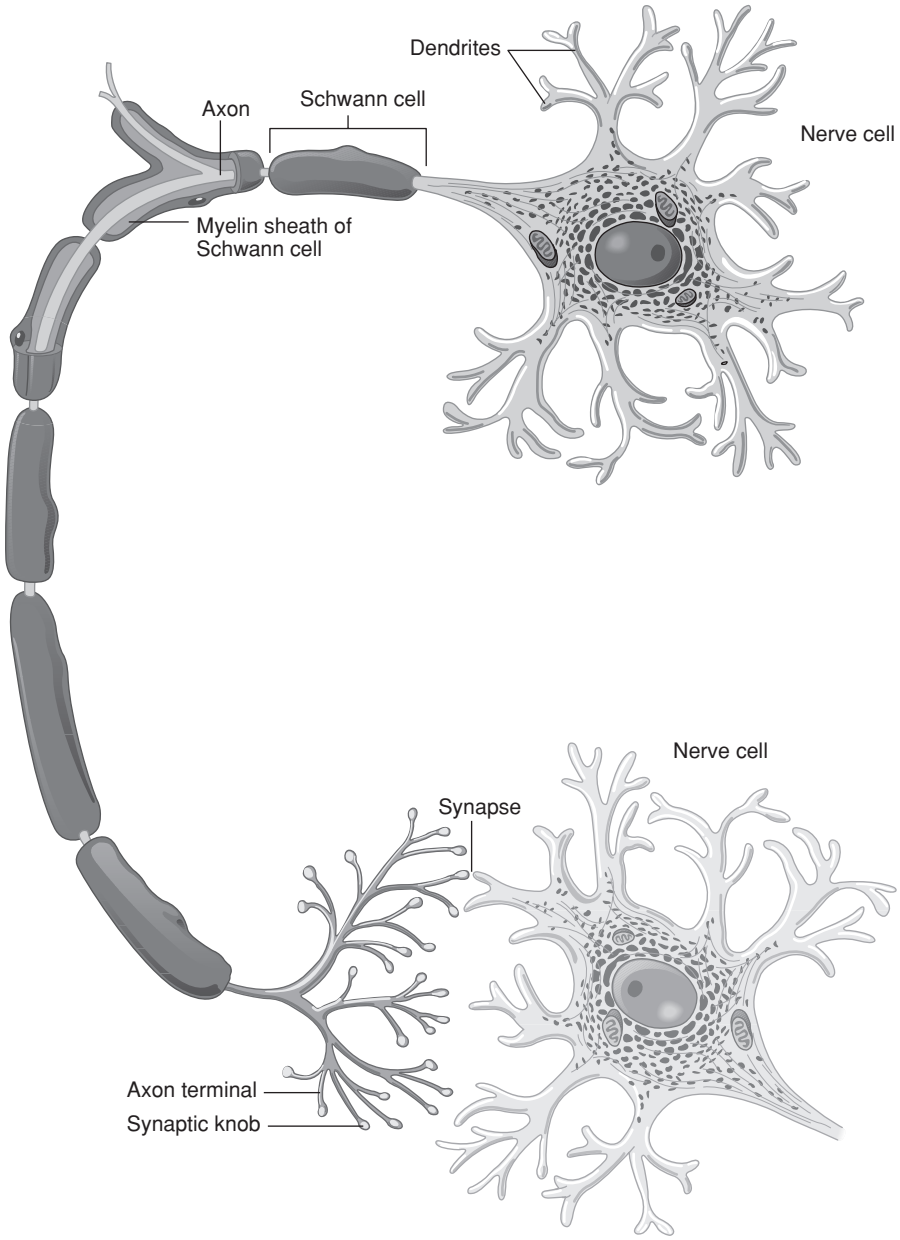
Neurotransmitters are chemicals located and released in the **brain** to allow an impulse from one nerve cell to pass to another nerve cell.

Description

There are approximately 50 neurotransmitters identified. There are billions of nerve cells located in the brain, which do not directly touch each other. Nerve cells communicate messages by secreting neurotransmitters. Neurotransmitters can excite or inhibit neurons (nerve cells). Some common neurotransmitters are acetylcholine, norepinephrine, dopamine, serotonin and gamma aminobutyric acid (GABA). Acetylcholine and norepinephrine are excitatory neurotransmitters while dopamine, serotonin, and GABA are inhibitory. Each neurotransmitter can directly or indirectly influence neurons in a specific portion of the brain, thereby affecting behavior.

Mechanism of impulse transmission

A nerve impulse travels through a nerve in a long, slender cellular structure called an axon, and it eventually reaches a structure called the presynaptic membrane, which contains neurotransmitters to be released in a free space called the synaptic cleft. Freely flowing neurotransmitter molecules are picked up by receptors (structures that appear on cellular surfaces that pick up molecules that fit into them like a "lock and key") locat-



Neurotransmitters are chemicals that transmit messages from one nerve cell (neuron) to another. The nerve impulse travels from the first nerve cell through the axon—a single smooth body arising from the nerve cell— to the axon terminal and the synaptic knobs. Each synaptic knob communicates with a dendrite or cell body of another neuron, and the synaptic knobs contain neurovesicles that store and release neurotransmitters. The synapse lies between the synaptic knob and the next cell. For the impulse to continue traveling across the synapse to reach the next cell, the synaptic knobs release the neurotransmitter into that space, and the next nerve cell is stimulated to pick up the impulse and continue it.

ed in a structure called the postsynaptic membrane of another nearby neuron. Once the neurotransmitter is picked up by receptors in the postsynaptic membrane, the molecule is internalized in the neuron and the impulse continues. This process of nerve cell communication is extremely rapid.

Once the neurotransmitter is released from the neurotransmitter vesicles of the presynaptic membrane, the normal movement of molecules should be directed to receptor sites located on the postsynaptic membrane. However, in certain disease states, the flow of the neurotransmitter is defective. For example, in depression, the

flow of the inhibitory neurotransmitter serotonin is defective, and molecules flow back to their originating site (the presynaptic membrane) instead of to receptors on the postsynaptic membrane that will transmit the impulse to a nearby neuron.

The mechanism of action and localization of neurotransmitters in the brain has provided valuable information concerning the cause of many mental disorders, including clinical depression and chemical dependency, and in researching medications that allow normal flow and movement of neurotransmitter molecules.

Neurotransmitters, mental disorders, and medications

Schizophrenia

Impairment of dopamine-containing neurons in the brain is implicated in **schizophrenia**, a mental disease marked by disturbances in thinking and emotional reactions. Medications that block dopamine receptors in the brain, such as **chlorpromazine** and **clozapine**, have been used to alleviate the symptoms and help patients return to a normal social setting.

Depression

In depression, which afflicts about 3.5% of the population, there appears to be abnormal excess or inhibition of signals that control mood, thoughts, pain, and other sensations. Depression is treated with antidepressants that affect norepinephrine and serotonin in the brain. The antidepressants help correct the abnormal neurotransmitter activity. A newer drug, **fluoxetine** (Prozac), is a selective serotonin reuptake inhibitor (SSRI) that appears to establish the level of serotonin required to function at a normal level. As the name implies, the drug inhibits the re-uptake of serotonin neurotransmitter from synaptic gaps, thus increasing neurotransmitter action. In the brain, then, the increased serotonin activity alleviates depressive symptoms.

Alzheimer's disease

Alzheimer's disease, which affects an estimated four million Americans, is characterized by memory loss and the eventual inability for self-care. The disease seems to be caused by a loss of cells that secrete acetylcholine in the basal forebrain (region of brain that is the control center for sensory and associative information processing and motor activities). Some medications to alleviate the symptoms have been developed, but presently there is no known treatment for the disease.

Generalized anxiety disorder

People with **generalized anxiety disorder** (GAD) experience excessive worry that causes problems at work and in the maintenance of daily responsibilities. Evidence suggests that GAD involves several neurotransmitter systems in the brain, including norepinephrine and serotonin.

Attention-deficit/hyperactivity disorder

People affected by **attention-deficit/hyperactivity disorder** (ADHD) experience difficulties in the areas of attention, overactivity, impulse control, and distractibility. Research shows that dopamine and norepinephrine imbalances are strongly implicated in causing ADHD.

Others

Substantial research evidence also suggests a correlation of neurotransmitter imbalance with disorders such as borderline **personality disorders**, **schizotypal personality disorder**, **avoidant personality disorder**, **social phobia**, **histrionic personality disorder**, and **somatization disorder**.

Drug addictions

Cocaine and crack cocaine are psychostimulants that affect neurons containing dopamine in the areas of the brain known as the limbic and frontal cortex. When cocaine is used, it generates a feeling of confidence and power. However, when large amounts are taken, people "crash" and suffer from physical and emotional exhaustion as well as depression.

Opiates, such as heroin and morphine, appear to mimic naturally occurring peptide substances in the brain that act as neurotransmitters with opiate activity called endorphins. Natural endorphins of the brain act to kill pain, cause sensations of pleasure, and cause sleepiness. Endorphins released with extensive aerobic exercise, for example, are responsible for the "rush" that long-distance runners experience. It is believed that morphine and heroin combine with the endorphin receptors in the brain, resulting in reduced natural endorphin production. As a result, the drugs are needed to replace the naturally produced endorphins and **addiction** occurs. Attempts to counteract the effects of the drugs involve using medications that mimic them, such as nalorphine, naloxone, and **naltrexone**.

Alcohol is one of the depressant drugs in widest use, and is believed to cause its effects by interacting with the GABA receptor. Initially anxiety is controlled, but greater amounts reduce muscle control and delay reaction time due to impaired thinking.

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Nicotine and related disorders

Definition

Nicotine disorders are caused by the main psychoactive ingredient in tobacco. Nicotine is a physically and psychologically addictive drug. It is the most influential dependence-producing drug in the United States and worldwide, and its use is associated with many serious health risks.

Description

Nicotine is the most addictive and psychoactive chemical in tobacco, a plant native to the New World. Early European explorers learned to smoke its leaves from indigenous peoples who had been using tobacco for hundreds of years. They took tobacco back to Europe, where it became immensely popular. Tobacco became a major source of income for the American colonies and later for the United States. Advances in cigarette-making technology caused a boom in cigarette smoking in the early 1900s. Before the early twentieth century, most people who smoked had used pipes, cigars, or chewing tobacco.

In the 1950s researchers began to link cigarette smoking to certain respiratory diseases and cancers. In 1964 the Surgeon General of the United States issued the first health report on smoking. Cigarette smoking peaked in the United States in the 1970s, then began to decline as health concerns about tobacco increased. In 1971 cigarette advertising was banned from television, although tobacco products continue to be heavily advertised in other media even today. By 1998, it was estimated that 25% of Americans (about 60 million people) were active smokers, 25% were former smokers, and the remaining half have never smoked. About 85% of active smokers are addicted to nicotine.

Pure nicotine is a colorless liquid that turns brown and smells like tobacco when exposed to air. Nicotine can be absorbed through the skin, the lining of the mouth and nose, and the moist tissues lining the lungs. Cigarettes are the most efficient nicotine delivery system. Once tobacco smoke is inhaled, nicotine reaches the **brain** in less than 15 seconds. Since people who smoke pipes and cigars do not inhale, they absorb nicotine more slowly. Nicotine in chewing tobacco and snuff is absorbed through the mucous membranes lining the mouth and nasal passages. In 2002 a new smokeless tobacco product was test-marketed in the United States. Called Ariva, it is compressed tobacco powder about the size of a vitamin pill that is placed between the cheek and gum until it dissolves completely. The nicotine it contains is also absorbed through the mucous membranes of the mouth.

Causes and symptoms

How nicotine works

Nicotine is the main addictive drug among the 4,000 compounds found in tobacco smoke. Such other substances in smoke as tar and carbon monoxide present documented health hazards, but they are not addictive and do not cause cravings or withdrawal symptoms to the extent that nicotine does.

Nicotine is both a stimulant and a sedative. It is a psychoactive drug, meaning that it works in the brain, alters brain chemistry, and changes mood. Once tobacco smoke is inhaled, nicotine passes rapidly through the linings of the lungs and into the blood. It quickly circulates to the brain where it indirectly increases the supply of dopamine, a chemical in the brain that affects mood. Dopamine is normally released in response to pleasurable sensations. Nicotine, like cocaine or heroin, artificially stimulates the release of dopamine. This release accounts for the pleasurable sensation that most smokers feel almost as soon as they light up a cigarette. Nicotine also decreases anger and increases the efficiency of a person's performance on long, dull tasks.

At the same time nicotine is affecting the brain, it also stimulates the adrenal glands. The adrenal glands are small, pea-sized pieces of tissue located above each kidney. They produce several hormones, one of which is epinephrine, also called adrenaline. Under normal circumstances, adrenaline is released in response to **stress** or a perceived threat. It is sometimes called the "fight or flight" hormone, because it prepares the body for action. When adrenaline is released, blood pressure, heart rate, blood flow, and oxygen use increase. Glucose, a simple form of sugar used by the body, floods the body to provide extra energy to muscles. The overall effect of the release of these hormones is strain on the cardiovascular (heart and blood vessels) system. Stressed this way many

times a day for many years, the body responds by increasing the buildup of plaque, a sticky substance, in the blood vessels. These deposits of plaque significantly increase a person's risk of **stroke** or heart attack.

Most people begin smoking between the ages of 12 and 20. Surprisingly few people start smoking as adults over 21. Adolescents who smoke tend to begin as casual smokers, out of rebelliousness or a need for social acceptance. Dependence on nicotine develops rapidly, however; one study suggests that 85–90% of adolescents who smoke four or more cigarettes become regular smokers. Nicotine is so addictive that being tobacco-free soon feels uncomfortable. In addition, smokers quickly develop tolerance to nicotine. Tolerance is a condition that occurs when the body needs a larger and larger dose of a substance to produce the same effect. For smokers, tolerance to nicotine means more frequent and more rapid smoking. Soon most smokers develop physical withdrawal symptoms when they try to stop smoking. Users of other forms of tobacco experience the same effects; however, the delivery of nicotine is slower and the effects may not be as pronounced.

Nicotine dependence

In addition to the physical dependence caused by the actions of nicotine on the brain, there is a strong psychological component to the dependency of most users of tobacco products, especially cigarette smokers. Most people who start smoking or using smokeless tobacco products do so because of social factors. These include:

- the desire to fit in with peers
- acceptance by family members who use tobacco
- rebelliousness
- the association of tobacco products with maturity and sophistication
- positive response to tobacco advertising

Such personal factors as mental illness (depression, anxiety, **schizophrenia**, or alcoholism); the need to reduce stress and anxiety; or a desire to avoid weight gain also influence people to start smoking. Once smoking has become a habit, whether physical **addiction** occurs or not, psychological factors play a significant role in continuing to smoke. People who want to stop smoking may be discouraged from doing so because:

- They live or work with people who smoke and who are not supportive of their quitting.
- They believe they are incapable of quitting.
- They perceive no health benefits to quitting.
- They have tried to quit before and failed.
- They associate cigarettes with specific pleasurable activities or social situations that they are not willing to give up.
- They fear gaining weight. Successful smoking cessation programs must treat both the physical and psychological aspects of nicotine addiction.

KEY TERMS

Adrenaline—Another name for epinephrine, the hormone released by the adrenal glands in response to stress. It is the principal blood-pressure raising hormone and a bronchial and intestinal smooth muscles relaxant.

Cold turkey—A slang term for stopping the use of nicotine (or any other addictive drug) suddenly and completely.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Epinephrine—A hormone secreted by the adrenal glands in response to stress.

Plaque—A sticky cholesterol-containing substance that builds up on the walls of blood vessels, reducing or blocking blood flow.

Supportive—An approach to smoking cessation that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or exploratory approaches to treatment.

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

Nicotine withdrawal

The American Psychiatric Association first recognized nicotine dependence and nicotine withdrawal as serious psychological problems in 1980. Today nicotine is considered an addictive drug, although a common and legalized one.

Studies show that three-quarters of smokers try to quit, but only about 5–10% are eventually successful. Even those who succeed often make between five and ten attempts to quit before finally succeeding. Symptoms of nicotine withdrawal occur in about half the smokers try-

Transdermal patches are worn on the skin between the neck and the waist, and provide a steady delivery of nicotine through the skin. Patches come in varying strengths, and after several weeks, users can move down to a patch that delivers a lower dose. *(Robert J. Huffman/ Field Mark Publications. Reproduced by permission.)*

ing to quit who do not use nicotine replacement therapy (nicotine patches, inhalers, or gum).

As former smokers can attest, the combination of physiological and psychological factors make withdrawal from nicotine very difficult. Symptoms of nicotine withdrawal include:

- irritability
- restlessness
- increased anger or frustration
- sleep disturbances
- inability to concentrate
- increased appetite or desire for sweets
- depression
- anxiety
- constant thoughts about smoking
- cravings for cigarettes
- decreased heart rate
- coughing

Withdrawal symptoms are usually more pronounced in smokers than in those who use smokeless tobacco products, and heavy smokers tend to have more symptoms than light smokers when they try to stop smoking. People with depression, schizophrenia, alcoholism, or mood disorders find it especially difficult to quit, as nicotine offers temporary relief for some of the symptoms of these disorders.

Symptoms of nicotine withdrawal begin rapidly and peak within one to three days. Because of this rapid onset of withdrawal symptoms, only about 30% of people who try to quit smoking remain tobacco-free for even two days. Withdrawal symptoms generally last three to four weeks, but a significant number of smokers have withdrawal symptoms lasting longer than one month. Some people have strong cravings for tobacco that last for months, even though the physical addiction to nicotine is gone. These cravings often occur in social settings in which the person formerly smoked, such as at a bar or party, or after sex. Researchers believe that much of this extended craving is psychological.

Demographics

About 60 million Americans smoke cigarettes, cigars, and pipes; and about six million more use smokeless tobacco. Worldwide, there are more than a billion smokers. Although the prevalence of smoking has gradually decreased in the United States and many other industrialized countries since the 1970s, the use of tobacco products is rapidly increasing in the developing nations of Africa and Asia. Use of tobacco products in developing countries is of particular concern, because these countries often lack adequate health care resources to treat smoking-related diseases, let alone support smoking cessation programs.

In the past, the number of American men who smoked outnumbered women, but by 2000, the rate of smoking was almost the same for these two groups—about 35% of the population. Men in the United States greatly outnumber women, however, in the use of smokeless tobacco (14% to 1%). In developing countries, male smokers outnumber women smokers by a margin of eight to one. People who smoke tend to have lower levels of income and formal education than those who don't. About half the patients diagnosed with psychiatric problems are smokers, while more than three-quarters of those who abuse other substances also smoke.

In 2001, the most recent year for which statistics are available, smoking among high school students decreased. Daily use of cigarettes among eighth graders decreased from 7.4% to 5.5% and among tenth graders from 14% to 12.2%—both significant declines. The rate of smokeless tobacco usage stayed constant at about 4% of eighth graders and 7% of tenth graders, almost all of whom were boys. Smoking among women with less than a high school education increased, however. Although African American men overall have the highest rate of smoking in the United States population, smoking among African American high school students has decreased significantly. Only about 19% of African American high

school students smoke, compared to about 38% of Caucasian high school students. The change in rates of smoking among different subgroups in the population may reflect to some extent changes in the target groups of the billion-dollar advertising campaigns of cigarette manufacturers.

Recent research suggests that there may be a genetic component to nicotine dependence, just as there is for alcohol dependence. Studies show that girls (but not boys) whose mothers smoked during pregnancy are four times more likely to smoke than those whose mothers were tobacco-free during pregnancy. Other research suggests that the absence of a certain enzyme in the body protects the body against nicotine dependence.

Diagnosis

Smokers usually self-diagnose their nicotine dependence and nicotine withdrawal. Such questionnaires as the Fagerstrom Test for Nicotine Dependence (FTND), a short six-item assessment of cigarette use, help to determine the level of tobacco dependence. Physicians and mental health professionals are less concerned with **diagnosis**, which is usually straightforward, than with determining the physical and psychological factors in each patient that must be addressed for successful smoking cessation.

Treatments

Most smokers want to quit; over 75% have tried to stop at least once. Each year, over 40% of smokers make at least one attempt to quit. Many people try a dozen times before they are successful in finding the right combination of medications, therapies, and support to achieve success. Even with repeated attempts, however, only half of all smokers are able to stop smoking completely and eliminate their dependence on nicotine.

Most people do not suddenly wake up one morning and decide to stop smoking. Instead, they go through several preparatory stages before taking action. First is the precontemplation stage, in which the smoker doesn¸t even think about quitting. Precontemplation is followed by the contemplation stage, in which the smoker thinks about quitting, but takes no action. Contemplation eventually turns to preparation, often when counselors or family members encourage or urge the smoker to quit. Now the smoker starts making plans to quit in the near future. Finally the smoker arrives at the point of taking action.

Having decided to stop smoking, a person has many choices of programs and approaches. When mental health professionals are involved in smoking cessation efforts, one of their first jobs is to identify the physical and psychological factors that keep the person smoking. This identification helps to direct the smoker to the most appropriate type of program. Assessment examines the frequency of the person's smoking, his or her social and emotional attachment to cigarettes, commitment to change, available support system, and barriers to change. These conditions vary from person to person, which is why some smoking cessation programs work for one person and not another.

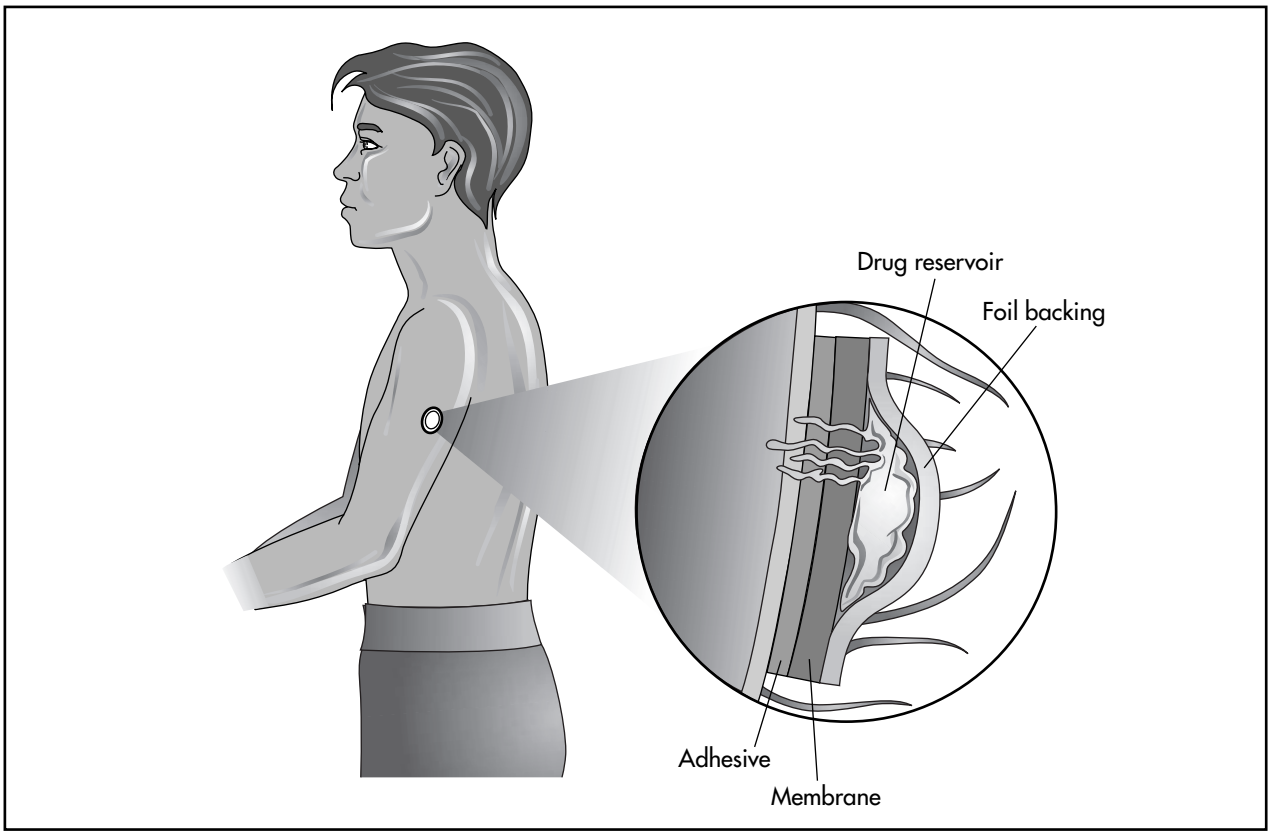
Medications

Before 1984, there were no medications to help smokers quit. In that year, a nicotine chewing gum (Nicorette) was approved by the United States Food and Drug Administration (FDA) as a prescription drug for smoking cessation. In 1996 it became available without prescription. Nicorette was the first of several medications that are used for nicotine replacement therapy, intended to gradually reduce nicotine dependence in order to prevent or reduce withdrawal symptoms. This approach, called tapering, is used in withdrawal of other addictive drugs.

About 15% of people using nicotine gum are able to stop smoking permanently. This rate is about three times higher than for people who simply go "cold turkey" and stop smoking without any assistance. Nicotine gum comes in two strengths. As it is chewed, nicotine is released and absorbed through the lining of the mouth. Over a six- to 12-week period, the amount and strength of gum chewed is decreased, until the smoker is weaned away from his or her dependence on nicotine. Chewing gum may not be pleasant or socially acceptable to some people, and cannot be used by people who have diseases of the jaw. In addition, some people report that the gum makes them feel queasy.

The nicotine transdermal patches have been available without prescription since 1996. They are marketed under several brand names, including Habitrol, Nicoderm, NicoDerm CQ, Prostep and Nicotrol. All but Nicotrol are 24-hour patches. Nicotrol is a 16-hour patch designed to be removed at night. The patches are worn on the skin between the neck and the waist, and provide a steady delivery of nicotine through the skin. Patches come in varying strengths. After several weeks, users can move down to a patch that delivers a lower dose. Some people using the 24-hour patches experience sleep disturbances, and a few develop mild skin irritations, but generally side effects are few.

Two other nicotine delivery devices are available by prescription only. One is a nicotine nasal spray. It has the advantage of delivering nicotine rapidly, just as a cigarette does. Treatment with nasal spray usually lasts four



The nicotine patch is a transepidermal patch designed to deliver nicotine, the addictive substance in cigarettes, directly through the skin into the bloodstream. The patch contains a drug reservoir sandwiched between a nonpermeable back layer and a permeable adhesive layer that attaches to the skin. The drug leaches slowly out of the reservoir, releasing small amounts of the drug at a constant rate for up to 24 hours. *(Illustration by Electronic Illustrators Group.)*

to six weeks. Side effects include cold-like symptoms (runny nose, sneezing, etc.). A nicotine inhaler is also available that delivers nicotine through the tissues of the throat. A major advantage of the inhaler is that it provides an alternative to having a cigarette in one's hands while it delivers nicotine.

One prescription drug that is not nicotine replacement therapy has also been approved for treatment of nicotine dependence. **Bupropion** (Zyban) was originally developed as antidepressant medication that appears to increase dopamine levels in the brain. Bupropion has been shown to be effective in smoking cessation. It has the advantage of being a pill taken twice a day. Its side effects include dry mouth and **insomnia**; in addition, it may not be suitable for people with certain medical conditions.

Behavioral treatments

Behavioral treatments are used to help smokers learn to recognize and avoid specific situations that trigger desire for a cigarette. They also help the smoker learn to substitute other activities for smoking. Behavioral treatments are almost always combined with smoker education, and usually involve forming a support network of other smokers who are trying to quit.

Behavioral treatments often take place in **support groups** either in person or online. They are most effective when combined with nicotine reduction therapy. Other supportive techniques include the use of rewards for achieving certain goals and contracts to clarify and reinforce the goals. Aversive techniques include asking the smoker to inhale the tobacco smoke deeply and repeatedly to the point of nausea, so that smoking is no longer associated with pleasurable sensations. Overall, quit rates are highest (about 30%) when **behavior modification** is combined with nicotine replacement therapy and tapering.

Alternative treatments

Many alternative therapies have been tried to help smokers withdraw from nicotine. Hypnosis has proved helpful in some cases, but has not been tested in controlled clinical trials. **Acupuncture**, relaxation tech-

niques, restricted environmental stimulation therapy (REST, a combination of relaxation and hypnosis techniques), special **diets**, and herbal supplements have all been used to help people stop smoking. Of these alternative techniques, clinical studies of REST showed substantial promise in helping people stop smoking permanently.

Prognosis

Smoking is a major health risk associated with nicotine dependence. About half of all smokers die of a smoking-related illness, often cancer. About 90% of lung cancers are linked to smoking. Smoking also causes such other lung problems as chronic bronchitis and emphysema, as well as worsening the symptoms of asthma. Other cancers associated with smoking include cancers of the mouth, esophagus, stomach, kidney, and bladder. Smoking accounts for 20% of cardiovascular deaths. It significantly increases the risk of heart disease, heart attack, stroke, and aneurysm. Women who smoke during pregnancy have more miscarriages, premature babies, and low-birth-weight babies. In addition, secondhand smoke endangers the health of nonsmokers in the smoker's family or workplace. Although most of these effects are not caused directly by nicotine, it is dependence on nicotine that keeps people smoking.

Even though it is difficult for smokers to break their chemical and psychological dependence on nicotine, they should remember that most of the negative health effects of smoking are reduced or reversed after quitting. Therefore, it is worth trying to quit smoking at any age, regardless of the length of time a person has had the habit.

Prevention

The best way to avoid nicotine dependence and withdrawal is to avoid the use of tobacco products.

See also Stress; Substance abuse and related disorders

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Nightmare disorder

Definition

Nightmare disorder, which is also called dream anxiety disorder, is characterized by the occurrence of repeated dreams during which the sleeper feels threatened and frightened. The sense of fear causes the person to awake.

Description

Nightmares are dreams that cause intense fear. These dreams are often complex and fairly long. During the dream, the sleeper usually encounters or experiences a threat to their life or safety. Nightmares are also reported that do not involve physical danger.

As the dream progresses, the threat to the person usually increases, as does their sense of fear. Waking usu-

KEY TERMS

Dream anxiety disorder—Another name for nightmare disorder.

Sleep terror disorder—A sleep disorder that is distinguished from nightmare disorder by the intensity of associated anxiety symptoms, the absence of complete wakefulness, and the person's difficulty recalling the episode.

ally occurs just as the threat or danger reaches its climax. It is often difficult for a person to return to sleep after waking from a nightmare. Nightmares usually occur during the second half of the night's sleep.

Causes and symptoms

During the course of a nightmare the sleeper may moan, talk, or move slightly, although these signs do not always appear. The person wakes from the nightmare with a profound sense of fear. Waking is complete, and usually accompanied by increased heart rate, sweating, and other symptoms of anxiety or fear. Once fully awake, the person usually has a good recall of the dream and what was so frightening about it. Because of the physical symptoms of anxiety and because clarity is achieved immediately upon waking, returning to sleep after a nightmare is often difficult. The vividness of the recall and the prominence of the dream images in the person's mind can also make it difficult to calm down and return to sleep.

Sometimes people may avoid going to sleep after a particularly intense nightmare because of the fear of having another bad dream. In addition, people may have problems falling asleep if they are experiencing anxiety caused by the fear of having nightmares. As a result, these people may have the signs and symptoms associated with mild sleep deprivation, such as decreased mental clarity, problems paying attention, excessive daytime sleepiness, irritability, or mild depression.

The causes of nightmares are not known for certain. Adults who have nightmares on a regular basis are a small minority of the American population. About half of these people are thought to suffer from psychiatric disorders that cause the nightmares. Nightmares may also be triggered by major psychological traumas, such as those experienced by patients with **post-traumatic stress disorder**. For most patients who do not have an underlying mental disorder, the nightmares are attributed to stress. Nightmares that occur on an irregular and occasional basis are usually attributed to life stressors and associated anxiety.

Some researchers think that artistic or creative people are at greater risk for nightmares, as are people who are generally sensitive. These people are considered to have well-developed imaginations and are very sensitive to environmental and social factors.

Nightmares can be a side effect of some medications or drugs of abuse, including drugs given for high blood pressure; levodopa and other drugs given to treat Parkinson's disease; **amphetamines**, cocaine, and other stimulants; and some antidepressants. Withdrawal from alcohol and other medications can also sometimes cause nightmares.

Demographics

The actual percentage of people that suffer from nightmare disorder is not known, as many people do not seek treatment for it. There are, however, estimates of the proportion of the population that experience occasional nightmares. Many children suffer from nightmares that concern their parents. Estimates on the number of children who have recurrent nightmares range from 10–50%. In children, however, nightmares are not usually associated with psychiatric illness.

The number of children experiencing nightmares decreases as they get older. More than 3% of young adults have frequent nightmares, but only about 1% of mature adults experience nightmares once or twice a week. Half of the adults in the United States who experience regular nightmares have diagnosable psychiatric illnesses. Women are estimated to have nightmares two to four times more frequently than men. There is some uncertainty as to whether this figure reflects an actual difference between the sexes in the frequency of nightmares, or whether women are simply more likely than men to report nightmares. Nightmares typically decrease in frequency as people grow older.

Diagnosis

A **diagnosis** of nightmare disorder is usually made because the person reports the problem to their family physician or a **psychiatrist**. There are no laboratory tests for nightmare disorder, although the doctor may give the patient a physical examination to rule out any medical conditions that may be causing anxiety or stress.

Nightmares are characterized by awakening with a sense of fear, a clear recollection of the dream, and physical symptoms of anxiety. Nightmares can occur during nighttime sleep or daytime naps. A patient experiencing

nightmares must meet the criteria listed in the ***Diagnostic and Statistical Manual of Mental Disorders*** to be diagnosed with nightmare disorder. The manual, which provides guidelines used by the American Psychiatric Association for diagnosing psychiatric disturbances, gives four distinct criteria:

- The patient must experience repeated awakenings from frightening dreams.
- When the patient awakes, he or she must wake fully and be aware of his or her surroundings.
- The nightmares must cause the patient distress in important areas of his or her life.
- The nightmares cannot be directly attributed to another disorder, or be the direct effects of medications, substance abuse, or a medical condition.

Nightmare disorder can be confused with **sleep terror disorder**. Both disorders are characterized by an arousal during sleep when the patient shows symptoms of anxiety or fear. Sleep terror, however, is characterized by a partial arousal from sleep during which the patient is generally nonresponsive. After a nightmare, the patient becomes fully awake and is aware of his or her surroundings. During an episode of sleep terror, a patient often gets out of bed and is active, and often screams or cries. During a nightmare, the patient may move slightly or moan but does not display such dramatic or active symptoms. Patients do not remember either the sleep terror episode or what caused the fear, but patients who have nightmares remember them with great clarity and often in considerable detail. Such symptoms of fear or anxiety as increased heart rate, dilated pupils, and sweating are not as dramatic in patients with nightmare disorder as they are in patients experiencing sleep terrors.

Treatments

Nightmares that are associated with a psychiatric disorder are managed by treating the underlying disorder. For patients without psychiatric disorders, psychological counseling to deal with any recurring themes in the nightmares may be helpful. Children may not require treatment for nightmares unless the dreams are causing significant distress, as nightmares generally resolve as children mature.

Because stress is thought to be the most common cause of nightmares, stress reduction techniques may prove to be effective complementary treatments. Typical relaxation techniques such as **yoga**, **meditation**, or exercise may be helpful. **Psychotherapy** can be an effective way to identify major stressors in the person's life, and to explore ways in which they may be reduced or eliminated.

Prognosis

Nightmare disorder can be a lifelong disorder. A general improvement in symptoms often takes place, however, as the patient gets older. Treatment for any underlying psychological disorders can be very successful.

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American Academy of Sleep Medicine. 6301 Bandel Road NW, Suite 101, Rochester, MN 55901. (507) 287-6006. <www.asda.org>.

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NLP *see* **Hypnotherapy**

Norpramin *see* **Desipramine**

Nortriptyline

Definition

Nortriptyline is a tricyclic antidepressant. It is sold in the United States under the brand names Aventyl and Pamelor, and is also available under its generic name.

Purpose

Nortriptyline is used to relieve symptoms of depression. The drug is more effective for endogenous depression than for other forms of depression. Endogenous depression is depression arising from metabolic changes within a person, such as chemical or hormonal imbalances. Nortriptyline is also used occasionally to treat

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Benign prostate hypertrophy—Enlargement of the prostate gland.

Bipolar syndrome—An abnormal mental condition characterized by periods of intense elation, energy and activity followed by periods of inactivity and depression.

Catecholamine—A group of neurotransmitters synthesized from the amino acid tyrosine and released by the hypothalamic-pituitary-adrenal system in the brain in response to acute stress. The catecholamines include dopamine, serotonin, norepinephrine, and epinephrine.

Endogenous depression—Depression arising from causes within a person, such as chemical or hormonal imbalances.

Manic—Referring to mania, a state characterized by excessive activity, excitement or emotion.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

premenstrual depression, **attention-deficit/hyperactivity disorder** in children, and bed-wetting (**enuresis**).

Description

Tricyclic antidepressants act to change the balance of naturally occurring chemicals in the **brain** that regulate the transmission of nerve impulses between cells. The precise way in which nortriptyline elevates mood is not fully understood. The drug inhibits the activity of **neurotransmitters** such as acetylcholine, histamine, and 5-hydroxytryptamine. Studies have indicated that nortriptyline interferes with the release, transport, and storage of catecholamines, another group of chemicals involved in nerve impulse transmission.

Recommended dosage

As with any antidepressant, the dose of nortriptyline must be carefully adjusted by the physician to produce the desired therapeutic effect. Nortriptyline is available in 10-, 25-, 50-, and 75-mg capsules as well as in a 10 mg/5mL solution. The usual dosage for nortriptyline is 25 mg given three or four times each day. The optimum total dose of the drug is 50 to 150 mg daily. Total dosage in excess of 150 mg is not recommended. The recommended dose for older adults (over age 60) and adolescents is 30 to 50 mg per day. Nortriptyline is not recommended for use by children.

The therapeutic effects of nortriptyline, like other tricyclic antidepressants, appear slowly. Maximum benefit is often not evident for two to three weeks after starting the drug. People taking nortriptyline should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Once symptoms of depression have been controlled, the lowest dosage that maintains the effect should be taken. People who take 100 mg or more of nortriptyline per day should have their blood tested periodically for nortriptyline concentrations. The results of these tests will show whether the dose is appropriate, too high, or too low.

Precautions

Like all tricyclic antidepressants, nortriptyline should be used cautiously and with close physician supervision in people, especially the elderly, who have benign prostatic hypertrophy, urinary retention, and glaucoma, especially angle-closure glaucoma (the most severe form). Before starting treatment, people with these conditions should discuss the relative risks and benefits of treatment with their doctors to help determine if nortriptyline is the right antidepressant for them.

A common problem with tricyclic antidepressants such as nortriptyline, is sedation (drowsiness, lack of physical and mental alertness). This side effect is especially noticeable early in therapy. In most patients, sedation decreases or disappears entirely with time, but until then, patients taking nortriptyline should not perform hazardous activities requiring mental alertness or coordination. The sedative effect is increased when nortriptyline is taken with other central nervous system depressants, such as alcoholic beverages, sleeping medications, other sedatives, or antihistamines. It may be dangerous to take nortriptyline in combination with these substances.

Nortriptyline may increase the possibility of having **seizures**. Patients should tell their physician if they have a history of seizures, including seizures brought on by the abuse of drugs or alcohol. These people should use nortriptyline only with caution and be closely monitored by their physician. Nortriptyline can also cause ringing in

the ears, tingling in the extremities, and numbness in the extremities, although none of these side effects are common when the drug is used as directed.

When used by people with **schizophrenia**, nortriptyline may worsen psychotic, increase hostility in some patients, or activate other symptoms that had not previously been expressed. When used by people with **bipolar disorder** (manic-depressive illness), symptoms of mania may be magnified. Patients with a history of **suicide** attempts, thoughts of suicide, or drug overdose should be monitored carefully when using nortriptyline. Nortriptyline can either increase or decrease blood sugar levels, depending on the patient and his or her medical condition. Nortriptyline should be used with great caution when a patient is receiving **electroconvulsive therapy**.

Nortriptyline may increase heart rate and cause irregular heartbeat. It may also raise or lower blood pressure. It may be dangerous for people with cardiovascular disease, especially those who have recently had a heart attack, to take this drug or other antidepressants in the same pharmacological class. In rare cases in which patients with cardiovascular disease must receive nortriptyline, they should be monitored closely for cardiac rhythm disturbances and signs of cardiac stress or damage.

Side effects

Nortriptyline shares side effects common to all tricyclic antidepressants. The most frequent of these are dry mouth, constipation, urinary retention, increased heart rate, sedation, irritability, dizziness, and decreased coordination. As with most side effects associated with tricyclic antidepressants, the intensity is highest at the beginning of therapy and tends to decrease with continued use.

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Patients may also chew sugarless gum or suck on sugarless candy in order to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement who take nortriptyline may be especially likely to have problems with urinary retention. Symptoms include having difficulty starting a urine flow and more difficulty than usual passing urine. In most cases, urinary retention is managed with dose reduction or by switching to another type of antidepressant.

Problems associated with the skin (loss of sensation, numbness and tingling, rashes, spots, itching and puffiness), seizures and ringing in the ears have also been reported. Nausea, vomiting, loss of appetite, diarrhea and abdominal cramping are associated with nortriptyline usage. Skin rash, sensitivity to sunlight and itching have been linked to nortriptyline use. People who think they may be experiencing any side effects from this or any other medication should talk to their physicians.

Interactions

Dangerously high blood pressure has resulted from the combination of tricyclic antidepressants, such as nortriptyline, and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Because of this, nortriptyline should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting nortriptyline or any other tricyclic antidepressant. The same holds true when discontinuing nortriptyline and starting an MAO inhibitor.

Cimetidine (Tagamet) may slow the elimination of nortriptyline, thus effectively increasing the dosage of nortriptyline. Quinidine also raises the circulating levels of the drug, requiring a decrease in the dosage of nortriptyline.

The sedative effects of nortriptyline are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or medications used for other mental disorders such as schizophrenia. The symptoms of increased heart rate, blurred vision, and difficulty urinating are additive with other drugs such as **benztropine**, **biperiden**, **trihexyphenidyl**, and antihistamines.

See also Neurotransmitters

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American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

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Nutrition and mental health

Nutrition and the brain

A person's food intake affects mood, behavior, and **brain** function. A hungry person may feel irritable and restless, whereas a person who has just eaten a meal may feel calm and satisfied. A sleepy person may feel more productive after a cup of coffee and a light snack. A person who has consistently eaten less food or energy than needed over a long period of time may be apathetic and moody.

The human brain has high energy and nutrient needs. Changes in energy or nutrient intake can alter both brain chemistry and the functioning of nerves in the brain. Intake of energy and several different nutrients affect levels of chemicals in the brain called **neurotransmitters**. Neurotransmitters transmit nerve impulses from one nerve cell to another, and they influence mood, sleep patterns, and thinking. Deficiencies or excesses of certain vitamins or minerals can damage nerves in the brain, causing changes in memory, limiting problem-solving ability, and impairing brain function.

Several nutritional factors can influence mental health, including: overall energy intake, intake of the energy-containing nutrients (proteins, carbohydrates, and fats), alcohol intake, and intake of vitamins and minerals. Often deficiencies of multiple nutrients rather than a single nutrient are responsible for changes in brain functioning.

In the United States and other developed countries, alcoholism is often responsible for nutritional deficiencies that affect mental functioning. Diseases can also cause nutritional deficiencies by affecting absorption of nutrients into the body or increasing nutritional requirements. Poverty, ignorance, and fad **diets** also contribute to nutritional deficiencies.

Energy intake and mental health

Energy, often referred to as the calorie content of a food, is derived from the carbohydrate, protein, fat, and alcohol found in foods and beverages. Although vitamins and minerals are essential to the body, they provide no energy. The human brain is metabolically very active and uses about 20 to 30% of a person's energy intake at rest. Individuals who do not eat adequate calories from food to meet their energy requirements will experience changes in mental functioning. Simply skipping breakfast is associated with lower fluency and problem-solving ability, especially in individuals who are already slightly malnourished. A hungry person may also experience lack of energy or motivation.

Chronic hunger and energy deprivation profoundly affects mood and responsiveness. The body responds to energy deprivation by shutting or slowing down nonessential functions, altering activity levels, hormonal levels, oxygen and nutrient transport, the body's ability to fight infection, and many other bodily functions that directly or indirectly affect brain function. People with a consistently low energy intake often feel apathetic, sad, or hopeless.

Developing fetuses and young infants are particularly susceptible to brain damage from malnutrition. The extent of the damage depends on the timing of the energy deprivation in relation to stage of development. Malnutrition early in life has been associated with below-normal intelligence, and functional and cognitive defects.

Carbohydrates and mental health

Carbohydrates include starches, naturally occurring and refined sugars, and dietary fiber. Foods rich in starches and dietary fiber include grain products like breads, rice, pasta and cereals, especially whole-grain products; fruits; and vegetables, especially starchy vegetables like potatoes. Foods rich in refined sugars include cakes, cookies, desserts, candy, and soft drinks.

Carbohydrates significantly affect mood and behavior. Eating a meal high in carbohydrates triggers release of a hormone called insulin in the body. Insulin helps let

blood sugar into cells where it can be used for energy, but insulin also has other effects in the body. As insulin levels rise, more tryptophan enters the brain. Tryptophan is an amino acid, or a building block of protein, that affects levels of neurotransmitters in the brain. As more tryptophan enters the brain, more of the neurotransmitter serotonin is produced. Higher serotonin levels in the brain enhance mood and have a sedating effect, promoting sleepiness. This effect is partly responsible for the drowsiness some people experience after a large meal.

Some researchers claim that a high sugar intake causes hyperactivity in children. Although carefully controlled studies do not support this conclusion, high sugar intake is associated with dental problems. Further, foods high in refined sugars are often low in other nutrients, making it prudent to limit their use.

Proteins and mental health

Proteins are made up of amino acids linked together in various sequences and amounts. The human body can manufacture some of the amino acids, but there are eight essential amino acids that must be supplied in the diet. A complete or high-quality protein contains all eight of the essential amino acids in the amounts needed by the body. Foods rich in high-quality protein include meats, milk and other dairy products, and eggs. Dried beans and peas, grains, and nuts and seeds also contain protein, although the protein in these plant foods may be low in one or more essential amino acid. Generally, combining any two types of plant protein foods together will yield a complete, high-quality protein. For example, a peanut butter and jelly sandwich combines grain protein from the bread with nut protein from the peanut butter to yield a complete protein. A bean-rice hot dish combines bean and grain protein for another complete protein combination.

Protein intake and intake of individual amino acids can affect brain functioning and mental health. Many of the neurotransmitters in the brain are made from amino acids. The neurotransmitter dopamine is made from the amino acid tyrosine. The neurotransmitter serotonin is made from the amino acid tryptophan. If the needed amino acid is not available, levels of that particular neurotransmitter in the brain will fall, and brain functioning and mood will be affected. For example, if there is a lack of tryptophan in the body, not enough serotonin will be produced, and low brain levels of serotonin are associated with low mood and even aggression in some individuals. Likewise, some diseases can cause a buildup of certain amino acids in the blood, leading to brain damage and mental defects. For example, a buildup of the amino acid phenylalanine in individuals with a disease called phenylketonuria can cause brain damage and **mental retardation**.

KEY TERMS

Amino acid—A building block of protein.

Anemia—Condition that results when there is a deficiency of oxygen in the blood. Can cause fatigue and impair mental functions.

Antioxidant—Substance that protects the body from damaging reactive oxygen molecules in the body. These reactive oxygen molecules can come from inside the body or from environmental pollution and are thought to play a role in the aging process and the development of degenerative disease.

Atherosclerosis—Clogging of the arteries, creating a risk factor for stroke.

Homocysteine—A chemical that builds up in the blood when methionine is not properly processed. High blood levels of homocysteine increase risk of heart disease and stroke.

Methionine—An amino acid that, when not metabolized properly, allows homocysteine to build up in the blood. Folic acid aids methionine metabolism.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Phenylketonuria—(PKU) An inherited disease in which the body cannot metabolize the amino acid phenylalanine properly. If untreated, phenylketonuria can cause mental retardation.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Trace mineral—An element essential to nutrition or bodily processes that is found in minute quantities.

Tryptophan—An essential amino acid released from proteins during the process of digestion. Tryptophan is an important ingredient in the body's production of serotonin.

Fats and mental health

Dietary intake of fats may also play a role in regulating mood and brain function. Dietary fats are found in both animal and plant foods. Meats, regular-fat dairy products, butter, margarine, and plant oils are high in fats.

Although numerous studies clearly document the benefits of a cholesterol-lowering diet for the reduction of heart disease risk, some studies suggest that reducing fat and cholesterol in the diet may deplete brain serotonin levels, causing mood changes, anger, and aggressive behavior.

Other studies have looked at the effects of a particular kind of fat, the omega-3 fatty acids found in fish oils, and brain functioning. Although a few studies suggest omega-3 fatty acids are helpful with bipolar affective disorder and **stress**, results are inconclusive.

High levels of fat and cholesterol in the diet contribute to atherosclerosis, or clogging of the arteries. Atherosclerosis can decrease blood flow to the brain, impairing brain functioning. If blood flow to the brain is blocked, a **stroke** occurs.

Alcohol and mental health

A high alcohol intake can interfere with normal sleep patterns, and thus can affect mood. Alcoholism is one of the most common causes of nutritional deficiencies in developed countries. Alcoholic beverages provide energy but virtually no vitamins or minerals. A person who consumes large amounts of alcohol will meet their energy needs but not their vitamin and mineral needs. In addition, extra amounts of certain vitamins are needed to break down alcohol in the body, further contributing to nutrient deficiencies.

Vitamins and mental health

Thiamin

Thiamin is a B vitamin found in enriched grain products, pork, legumes, nuts, seeds, and organ meats. Thiamin is intricately involved with metabolizing glucose, or blood sugar, in the body. Glucose is the brain's primary energy source. Thiamin is also needed to make several neurotransmitters.

Alcoholism is often associated with thiamin deficiency. Alcohol interferes with thiamin metabolism in the body, and diets high in alcohol are often deficient in vitamins and minerals. Individuals with a thiamin deficiency can develop **Wernicke-Korsakoff syndrome**, which is characterized by confusion, mental changes, abnormal eye movements, and unsteadiness that can progress to severe memory loss.

Vitamin B-12

Vitamin B-12 is found only in foods of animal origin like milk, meat, or eggs. Strict vegans who consume no animal-based foods need to supplement their diet with vitamin B-12 to meet the body's need for this nutrient.

Vitamin B-12 is needed to maintain the outer coating, called the myelin sheath, on nerve cells. Inadequate myelin results in nerve damage and impaired brain function. Vitamin B-12 deficiency can go undetected in individuals for years, but it eventually causes low blood iron, irreversible nerve damage, **dementia**, and brain atrophy.

Folic acid

Folic acid is another B vitamin found in foods such as liver, yeast, asparagus, fried beans and peas, wheat, broccoli, and some nuts. Many grain products are also fortified with folic acid. In the United States, alcoholism is a common cause of folic acid deficiency.

Folic acid is involved in protein metabolism in the body and in the metabolism of some amino acids, particularly the amino acid methionine. When folic acid levels in the body are low, methionine cannot be metabolized properly and levels of another chemical, homocysteine, build up in the blood. High blood homocysteine levels increase risk of heart disease and stroke.

Even modest folic acid deficiency in women causes an increased risk of neural tube defects, such as spina bifida, in developing fetuses. Folic acid deficiency also increases risk of stroke. Some studies suggest that folic acid deficiency leads to a range of mental disorders, including depression, but this concept remains controversial. Folic acid deficiency can lower levels of serotonin in the brain.

Niacin

The B vitamin niacin is found in enriched grains, meat, fish, wheat bran, asparagus, and peanuts. The body can also make niacin from the essential amino acid tryptophan, which is found in high-quality animal protein foods like meat and milk. Niacin deficiency used to be common in the southern United States but is now common only in developing countries such as India and China.

Niacin is involved in releasing energy in the body from carbohydrates, proteins, and fats. A deficiency of niacin produces many mental symptoms such as irritability, headaches, loss of memory, inability to sleep, and emotional instability. Severe niacin deficiency progresses to a condition called pellagra, which is characterized by the four D's: dermatitis (a rash resembling a sunburn), diarrhea, dementia, and ultimately, death. The mental

ESSENTIAL VITAMINS	
Vitamin	***What It Does For The Body***
Vitamin A (Beta Carotene)	Promotes growth and repair of body tissues; reduces susceptibility to infections; aids in bone and teeth formation; maintains smooth skin
Vitamin B-1 (Thiamin)	Promotes growth and muscle tone; aids in the proper functioning of the muscles, heart, and nervous system; assists in digestion of carbohydrates
Vitamin B-2 (Riboflavin)	Maintains good vision and healthy skin, hair, and nails; assists in formation of antibodies and red blood cells; aids in carbohydrate, fat, and protein metabolism
Vitamin B-3 (Niacinamide)	Reduces cholesterol levels in the blood; maintains healthy skin, tongue, and digestive system; improves blood circulation; increases energy
Vitamin B-5	Fortifies white blood cells; helps the body's resistance to stress; builds cells
Vitamin B-6 (Pyridoxine)	Aids in the synthesis and breakdown of amino acids and the metabolism of fats and carbohydrates; supports the central nervous system; maintains healthy skin
Vitamin B-12 (Cobalamin)	Promotes growth in children; prevents anemia by regenerating red blood cells; aids in the metabolism of carbohydrates, fats, and proteins; maintains healthy nervous system
Biotin	Aids in the metabolism of proteins and fats; promotes healthy skin
Choline	Helps the liver eliminate toxins
Folic Acid (Folate, Folacin)	Promotes the growth and reproduction of body cells; aids in the formation of red blood cells and bone marrow
Vitamin C (Ascorbic Acid)	One of the major antioxidants; essential for healthy teeth, gums, and bones; helps to heal wounds, fractures, and scar tissue; builds resistance to infections; assists in the prevention and treatment of the common cold; prevents scurvy
Vitamin D	Improves the absorption of calcium and phosphorous (essential in the formation of healthy bones and teeth) maintains nervous system
Vitamin E	A major antioxidant; supplies oxygen to blood; provides nourishment to cells; prevents blood clots; slows cellular aging
Vitamin K (Menadione)	Prevents internal bleeding; reduces heavy menstrual flow

Essential vitamins and their effects. *(Stanley Publishing.)*

symptoms in pellagra can progress to **psychosis**, **delirium**, coma, and death.

Vitamin B-6

Vitamin B-6, also known as pyridoxine, is found in many plant and animal foods, including chicken, fish, pork, whole wheat products, brown rice, and some fruits and vegetables. In healthy individuals, deficiency of vitamin B-6 is rare, but certain drugs, including some antidepressant drugs, can induce vitamin B-6 deficiency.

Vitamin B-6 is needed by the body to produce most of the brain's neurotransmitters. It is also involved in hormone production. Although rare, vitamin B-6 deficiency is characterized by mental changes such as **fatigue**, nervousness, irritability, depression, **insomnia**, dizziness, and nerve changes. These mental changes are related to the body's decreased ability to manufacture neurotransmitters with vitamin B-6 deficiency.

Just as vitamin B-6 deficiency causes mental changes, so does excess of vitamin B-6. Vitamin B-6 sup-

plements are used by many individuals for a variety of conditions, including carpal tunnel syndrome, premenstrual syndrome, and fibrocystic breast disease. Doses of 500 mg per day or more can cause nerve damage, dizziness, sensory loss, and numbness.

Vitamin E

Vitamin E is a fat-soluble vitamin that is plentiful in the diet, particularly in plant oils, green leafy vegetables, and fortified breakfast cereals. Vitamin E deficiency is very rare, except in disorders that impair absorption of fat-soluble vitamins into the body, such as cystic fibrosis, and liver diseases.

Vitamin E deficiency causes changes in red blood cells and nerve tissues. It progresses to dizziness, vision changes, muscle weakness, and sensory changes. If left untreated, the nerve damage from vitamin E deficiency can be irreversible. Because it is an antioxidant, vitamin E has also been studied for treatment of neurological conditions such as Parkinson's and **Alzheimer's disease**. Although results are inconclusive, vitamin E shows some promise in slowing the progression of Parkinson's disease.

Vitamin A

Vitamin A is a fat-soluble vitamin found in meats, fish and eggs. A form of vitamin A, beta-carotene, is found in orange and green leafy vegetables such as carrots, yellow squash, and spinach. Headache and increased pressure in the head is associated with both deficient and excess vitamin A intake. Among other effects, excess vitamin A intake can cause fatigue, irritability, and loss of appetite. Generally, doses must exceed 25,000 international units of vitamin A over several months to develop such symptoms.

Minerals and mental health

Iron

Iron is a trace mineral that is essential for formation of hemoglobin, the substance that carries oxygen to cells throughout the body. Iron is found in meat, poultry, and fish. Another form of iron that is not as well absorbed as the form in animal foods is found in whole or enriched grains, green leafy vegetables, dried beans and peas, and dried fruits. Consuming a food rich in vitamin C, such as orange juice, at the same time as an iron-containing plant food will enhance iron absorption from the food.

Iron deficiency eventually leads to anemia, with insufficient oxygen reaching the brain. The anemia can cause fatigue and impair mental functioning. Iron deficiency during the first two years of life can lead to permanent brain damage.

Magnesium

The mineral magnesium is found in green leafy vegetables, whole grains, nuts, seeds, and bananas. In areas with hard water, the water may provide a significant amount of magnesium. In addition to its involvement in bone structure, magnesium aids in the transmission of nerve impulses.

Magnesium deficiency can cause restlessness, nervousness, muscular twitching, and unsteadiness. Acute magnesium deficiency can progress to **apathy**, delirium, convulsions, coma, and death.

Manganese

Manganese is a trace mineral found in whole grains and nuts, and to a lesser extent, fruits and vegetables. Manganese is involved in carbohydrate metabolism and brain functioning. Although very rare, manganese deficiency can cause abnormalities in brain function. Miners of manganese in South America have developed manganese toxicity called manganese madness, with neurological symptoms similar to Parkinson's disease.

Copper

The richest sources of the trace mineral copper in the diet are organ meats, seafood, nuts, seeds, whole grain breads and cereals, and chocolate. In addition to other functions, copper is involved in iron metabolism in the body and in brain function. Deficiency of copper causes anemia, with inadequate oxygen delivery to the brain and other organs. Copper deficiency also impairs brain functioning and immune system response, including changes in certain chemical receptors in the brain and lowered levels of neurotransmitters.

Zinc

The trace mineral zinc is found in red meats, liver, eggs, dairy products, vegetables, and some seafoods. Among other functions, zinc is involved in maintaining cell membranes and protecting cells from damage. Zinc deficiency can cause neurological impairment, influencing appetite, taste, smell, and vision. It has also been associated with apathy, irritability, jitteriness, and fatigue.

Selenium

Good sources of the trace mineral selenium include seafood, liver, and eggs. Grains and seeds can also be good sources of selenium depending on the selenium content of the soil they are grown in. Selenium is needed for the synthesis of some hormones and helps protect cell membranes from damage.

Although selenium deficiency is very rare, selenium toxicity has occurred in regions of the world with high selenium soil content, such as China. Selenium toxicity causes nervous system changes, fatigue, and irritability.

See also Diets; Nutrition counseling

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Nutrition counseling

Definition

Nutrition counseling is an ongoing process in which a health professional, usually a registered dietitian, works with an individual to assess his or her usual dietary intake and identify areas where change is needed. The nutrition counselor provides information, educational materials, support, and follow-up to help the individual make and maintain the needed dietary changes.

KEY TERMS

Body mass index, or BMI—A measure of body fat, calculated as weight in kilograms over the square of height in meters.

Food frequency questionnaire—A listing of how often a person consumes foods from certain food groups in a given period of time.

Registered dietitian—A person who has met certain education and experience standards and is well-qualified to provide nutrition counseling.

Twenty-four-hour recall—A listing of the type and amount of all foods and beverages consumed by a person in a 24-hour period.

Purpose

The goal of nutrition counseling is to help a person make and maintain dietary changes. For a person with a mental disorder, dietary change may be needed to promote healthier eating, to adopt a therapeutic diet, or to avoid nutrient-drug interactions. Nutrition counseling is an integral part of treatment for persons with eating disorders or chemical dependencies. Persons taking certain drugs, such as monoamine oxidase inhibitors, used to treat depression and anxiety disorders, need to follow a tyramine-controlled diet to avoid dietary interference with their medication. Many drugs used to treat mental disorders can cause weight gain or loss, so persons taking these medications may also benefit from nutrition counseling.

The nutrition counselor and individual work together to assess current eating patterns and identify areas where change is needed. Registered dietitians have met certain education and experience standards and are well qualified to provide nutrition counseling, but nurses, physicians, and health educators also provide nutrition counseling.

Description

Assessing dietary habits

Nutrition counseling usually begins with an interview in which the counselor asks questions about a person's typical food intake. Nutrition counselors use different methods to assess typical food intake.

The 24-hour recall method is a listing of all the foods and beverages a person consumed within the previous 24-hour period. The nutrition counselor may ask a person to recall the first thing he or she ate or drank the previous morning. The counselor then records the estimated

amounts of all the foods and beverages the person consumed the rest of the day. The 24-hour food recall can be used to provide an estimate of energy and nutrient intake. However, people tend to over- or underestimate intake of certain foods, and food intake on one day may not accurately represent typical food intake.

A food frequency questionnaire can sometimes provide a more accurate picture of a person's typical eating patterns. The nutrition counselor may ask the client how often he or she consumes certain food groups. For example, the counselor may ask a person how many servings of dairy products, fruits, vegetables, grains and cereals, meats, or fats he or she consumes in a typical day, week, or month.

Daily food records are also useful in assessing food intake. An individual keeps a written record of the amounts of all foods and beverages consumed over a given period of time. The nutrition counselor can then use the food records to analyze actual energy and nutrient intake. Three-day food records kept over two weekdays and one weekend day are often used.

Assessing body weight

Nutrition counselors may assess an individual's body weight by comparing his or her weight to various weight-for-height tables. A rough rule of thumb for determining a woman's ideal body weight is to allow 100 lb (45 kg) for the first 5 ft (1.5 m) of height plus 5 lb (2.3 kg) for every additional inch. A man is allowed 106 lb (48 kg) for the first 5 ft (1.5 m) of height plus 6 lb (2.7 kg) for every additional inch. However, this guide does not take into account a person's frame size.

Body mass index, or BMI, is another indicator used to assess body weight. BMI is calculated as weight in kilograms divided by height in meters squared. A BMI of 20 to 25 is considered normal weight, a BMI of less than 20 is considered underweight, and a BMI of greater than 25 is considered overweight.

Identifying changes needed

The initial dietary assessment and interview provide the basis for identifying behaviors that need to be changed. Sometimes a person already has a good idea of what dietary changes are needed, but may require help making the changes. Other times the nutrition counselor can help educate a person on the health effects of different dietary choices. The nutrition counselor and client work together to identify areas where change is needed, prioritize changes, and problem-solve as to how to make the changes.

Making dietary change is a gradual process. An individual may start with one or two easier dietary changes the first few weeks and gradually make additional or more difficult changes over several weeks or months. For example, an easy change for a person might be switching from 2% to skim milk, or taking time for a quick yogurt or granola bar in the morning instead of skipping breakfast. More difficult changes might be learning to replace high-fat meat choices with leaner ones, or including more servings of vegetables daily.

In making dietary changes, each individual's situation and background must be carefully considered. Factors that affect food decisions include an individual's ethnic background, religion, group affiliation, socioeconomic status, and world view.

Identifying barriers to change

Once the needed changes have been identified, the client and nutrition counselor think through potential problems that may arise. For example, changing eating behaviors may mean involving others, purchasing different foods, planning ahead for social events, or bringing special foods to work. Some common barriers to changing eating habits include:

- inconvenience
- social gatherings
- food preferences
- lack of knowledge or time
- cost

Setting goals

The nutrition counselor and client set behavior-oriented goals together. Goals should focus on the behaviors needed to achieve the desired dietary change, not on an absolute value, such as achieving a certain body weight. For a person working to prevent weight gain associated with certain medications, for example, his or her goals might be to increase the amount of fruits, vegetables, and whole grains consumed each day. Such changes would help prevent weight gain while placing the emphasis on needed behaviors rather than on actual weight.

Finding support

Family members are encouraged to attend nutrition counseling sessions with the client, especially if they share responsibility for food selection and preparation. Although the individual must make food choices and take responsibility for dietary changes, having the support and understanding of family and friends makes success more likely.

Maintaining changes

The challenge for the nutrition client lies not in making the initial dietary changes, but in maintaining them over the long term. Self-monitoring, realistic expectations, and continued follow-up can help a person maintain dietary changes.

Self-monitoring involves regularly checking eating habits against desired goals and keeping track of eating behaviors. Keeping a food diary on a daily or periodic basis helps the individual be more aware of his or her eating behaviors and provides a ready tool to analyze eating habits. Sometimes a simplified checklist to assure adequate intake of different food groups may be used.

Individuals and nutrition counselors should not expect perfect dietary compliance—slips inevitably occur. The goal is to keep small slips, such as eating a few extra cookies, from becoming big slips, like total abandonment of dietary change. The counselor can help the client identify situations that may lead to relapse and plan ways to handle the situations ahead of time.

Nutrition counseling is an ongoing process that can take months or years. In follow-up nutrition counseling sessions, the individual and counselor analyze food records together and problem-solve behaviors that are especially difficult to change. Follow-up counseling also allows the opportunity to reevaluate goals and strategies for achieving those goals.

See also Diets; Nutrition and mental health

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O

Obesity

Definition

Obesity is the condition of having an excessive accumulation of fat in the body, resulting in a body weight more than 20% above the average for height, age, sex, and body type, and in elevated risk of disability, illness, and death.

Description

The human body is composed of bone, muscle, specialized organ tissues, and fat. Together, all of these tissues comprise the total body mass, which is measured in pounds. Fat, or adipose tissue, is a combination of essential fat (an energy source for the normal physiologic function of cells and organs) and storage fat (a reserve supply of energy for future needs). When the amount of energy consumed as food exceeds the amount of energy expended in the normal maintenance of life processes and in physical activity, storage fat accumulates in excessive amounts. Essential fat is tucked in and around internal organs, and is an important building block of all cells in the body. Storage fat accumulates in the chest and abdomen, and, in much greater volume, under the skin.

Causes and symptoms

The human body was designed for life forty thousand years ago, when the ability to store energy in times of plenty meant the difference between life and death during famine. This protective mechanism is a source of trouble when food, in unlimited quantities, is readily available,. This is evident in the increasing prevalence of obesity in modern times, particularly in Western cultures. While obesity is just an exaggeration of a normal body, the storage of energy for future is properly classified as a health problem. This is because excessive amounts of storage fat may interfere with the normal physiology of the body. Obesity is directly related to the increasing prevalence of Type II diabetes in American society and for the appearance of Type II diabetes in children, previously a rarity. Because obesity promotes degenerative disease of joints and heart and blood vessels, it increases the need for some surgical procedures. At the same time, surgical complication rates are higher in obese patients. Obesity contributes to **fatigue**, high blood pressure, menstrual disorders, infertility, digestive complaints, low levels of physical fitness, and to the development of some cancers. The social costs of obesity that include decreased productivity, discrimination, depression, and low self-esteem, are less easily described and measured. Worldwide, obesity has reached epidemic proportions in the last thirty years, affecting both sexes and all ethnic, age, and socioeconomic groups. More than 50% of adults in the United States currently fall into overweight or obese classifications, and 22% of preschool children are classified as overweight. The increasing prevalence of obesity and diabetes in children and young adults heralds spiraling health care costs in the near future.

Because obesity reflects an imbalance between the amount of energy taken into the body in the form of food and the amount of energy expended in metabolism and physical activity, and because eating is an activity that involves choice and volition, obesity is classified by the Health Care Financing Administration (HCFA) as a "behavior" rather than as a disease. In recent years, following a pattern established in other behavioral problems such as alcoholism, researchers have attempted to establish a biologic basis for the development of obesity. They have succeeded in identifying many markers of the biochemical mechanisms that appear to be involved in feedback loops that control energy balance. However, much of the information is extrapolated from experimental work in rodents. Leptin, a hormone produced in fat cells is an example of such a marker. Leptin excited a great deal of hope as a potential treatment of obesity, but, as with many other laboratory discoveries, the hormone has proved far more complex and less easily understood in humans. Research to date indicates that

KEY TERMS

Behavior—A stereotyped motor response to an internal or external stimulus.

Biochemical—Chemical reactions occurring in living systems.

Body mass—The quantity of matter in the body (measured by dividing weight by acceleration due to gravity).

Calorie—The quantity of heat necessary to raise the temperature of 1kg of water 1°C.

Energy—The capability of producing force, performing work, or generating heat.

Feedback loops—Chains of biochemical reactions in which the products of reactions limit or enhance the subsequent reactions, and in which the chain ends up back at the first reaction, either limiting or enhancing it.

Genetic pool—The genetic material of an entire population.

Ideal weight—A range of body weights recommended for generally healthy adults.

Physiology—The branch of medicine concerned with biological processes or functions in the human body or any of its parts.

Prevalence—Occurence in a population.

Type II diabetes—Resistance to the effects of insulin in the presence of normal or elevated insulin levels, resulting in failure of glucose to enter cells and in a cascade of other abnormal physiologic reactions.

obesity is the end product of numerous contributing factors, including genetics, hormonal influences, behavioral tendencies, medication effects, and the surrounding society. But the rapid and widespread increase in obesity in the last thirty years reflects changes in activity patterns and in eating habits, not a change in the human genetic pool or in physiology.

Diagnosis

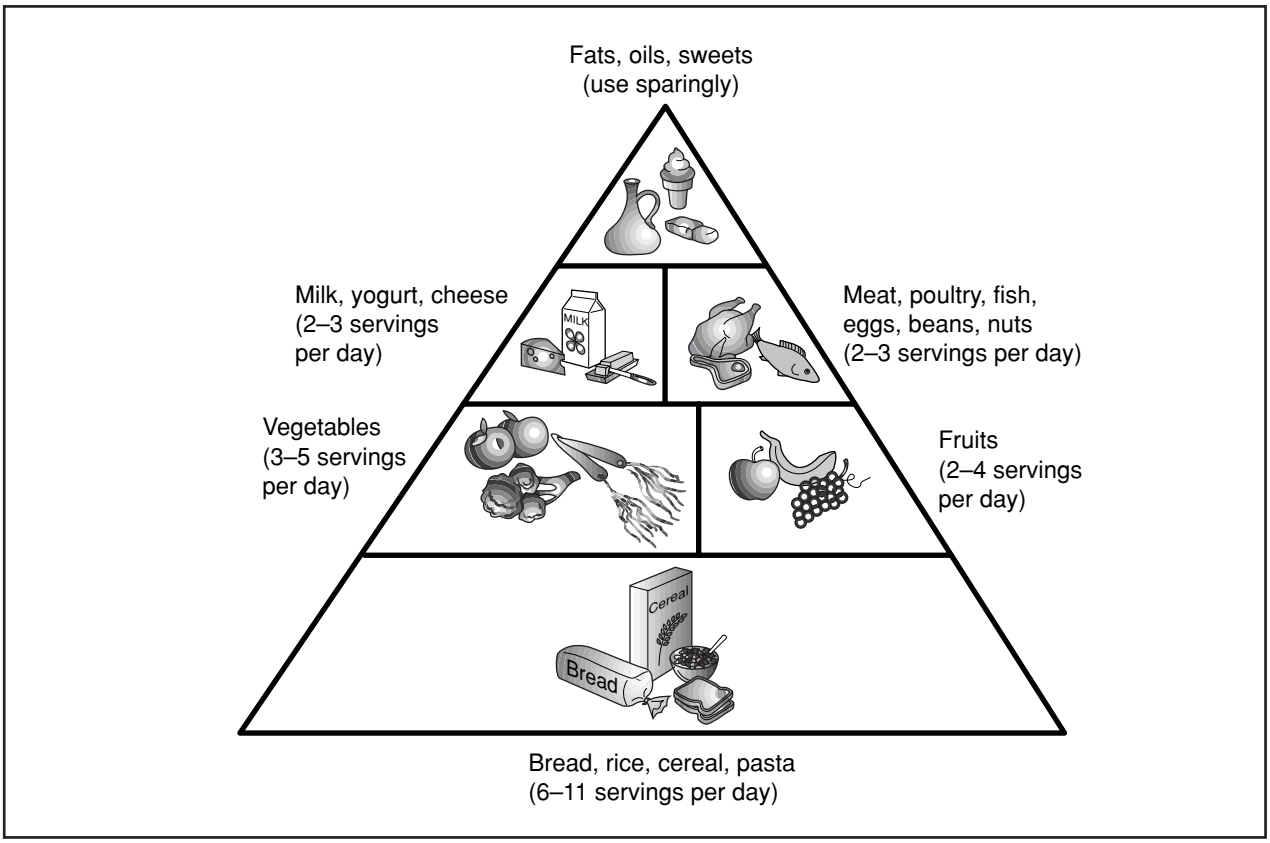
There are two methods of diagnosing obesity. The first method is inspection—whereby an excessive amount of storage fat is usually noticeable upon visual inspection. The second method is inference of body fat content, obtained from body measurements such as weight or skinfold thickness, and comparison with charts of similar measurements in broad populations. The determination of obesity is based on the amount of variance from "normal," a value that comes from statistics on death rates in people with similar measurements. Calculations such as the body mass index (BMI) use a height-weight relationship to calculate an individual's ideal weight and personal risk of developing obesity-related health problems. An individual with a BMI of 25.9–29, for example, is considered overweight; a person with a BMI over 30 is classified as obese.

The problem with using weight as a measure of obesity is the fact that weight does not accurately represent body composition. A heavily-muscled football player may weigh far more than a sedentary man of similar height, but have significantly less body fat. Chronic dieters, who have lost significant muscle mass during periods of caloric deprivation, may look slim and weigh little, but have elevated body fat percentages. The most accurate means of estimating body fat content involves weighing a person two ways: First, the person is weighed under water. The difference between dry and underwater weight is calculated to obtain the volume of water displaced by the mass of the body. While this method is impractical, it has the advantage of determining body composition most accurately, and is the truest reflection of the actual percentage of body mass that is fat. Women whose body fat exceeds 30% of total body mass and men whose body fat exceeds 25% are generally considered obese.

The pattern of fat distribution on the body may indicate whether an individual has a predisposition to develop certain diseases or conditions that may accompany obesity. "Apple-shaped" individuals who store most of their weight around the waist and abdomen are at greater risk for cancer, heart disease, **stroke**, and diabetes than "pear-shaped" people, whose extra pounds settle primarily on their hips and thighs.

Treatment

Since obesity develops when intake of the food required to produce energy exceeds the amount of energy used in metabolism and in physical activity, the treatment of obesity must alter one or both aspects of the energy stream. The options are to decrease energy intake or to increase energy output, or both. However, the problem does not yield rapidly to either method. Storage fat is meant to protect its bearer from starvation when food is unavailable, and before fat is tapped for energy. In the face of decreased intake of food, the body breaks down muscle to construct the sugar it needs to feed the **brain**. Much of the early weight loss on a very low calorie diet represents loss of muscle tissue rather than loss of fat. Similarly, fat



Suggested daily food servings. *(Illustration by Electronic Illustrators Group.)*

is not easy to access as fuel for exercise. A person of normal weight (according to one of the charts as described above) has enough body fat to fuel the muscles for days of continuous running, but will collapse long before burning any significant amount fat stored by the body.

When obesity develops in childhood, the total number of fat cells increases (hyperplastic obesity), whereas in adulthood, it is the total amount of fat in each cell that increases (hypertrophic obesity). Decreasing the amount of energy (food) consumed or increasing the amount of energy expended cannot change the number of fat cells already present. These actions can only reduce the amount of fat in each cell, and only if the process is slow and steady—as it was in reverse, when the excess fat accumulated. Prevention, as in so many problems, is far superior to any available treatment of obesity.

The strategy for weight loss in obese patients is first to change behavior; then, it is to decrease the expectation of rapid change. Behavioral treatment is goal-directed, process-oriented, and relies heavily on self-monitoring. Emphasis is on:

- Food intake: The potential energy provided by food is measured in calories, and the capacity of a certain type and amount of food to provide energy is called its caloric content. Keeping a food diary and developing a better understanding of the nutritional value and fat content of foods, changing grocery-shopping habits, paying attention to timing and appearance of meals, and slowing the speed of eating all help to modify food intake.
- Response to food: The body is capable of matching energy intake and output perfectly, but, in obese individuals, food intake is often unrelated from physiologic cues. Eating occurs for many reasons other than hunger. What psychological issues underlie the eating habits? Does **stress** cause **binge eating**? Is food seen as a reward? Recognition of psychological triggers is necessary for the development of alternate coping mechanisms that do not focus on food.
- Time usage: The body is suited for an ancient world in which physical activity was a necessity. In the modern world, physical activity must be a conscious choice. Making activity and exercise an integrated part of everyday life is a key to achieving and maintaining weight loss. Sedentary and overweight individuals have to reclaim slowly the endurance that is natural by managing their time to allow for gradual increases in both programmed and conscious lifestyle activity.

Behavior modification

For most individuals who are mildly obese, behavior modifications entail life-style changes they can make independently if they have access to accurate information and have reached the point of readiness to make a serious commitment to losing weight. A family physician's evaluation is helpful, particularly in regard to exercise capacity and nutritional requirements. Commercial weight-loss programs may be helpful for some mildly obese individuals, but they are of varying quality. A good program emphasizes realistic goals, gradual progress, sensible and balanced eating, and increased physical activity; it is often recommended by physicians. Programs that promise instant weight loss or feature severe restrictions in types and amounts of food are not effective, and, in some cases, can be dangerous.

For individuals who are moderately obese, medically supervised **behavior modification** and weight loss are more likely to be effective than an independent program. A realistic goal is loss of 10% of current weight over a six-month period. While doctors put most moderately obese patients on balanced, low-calorie **diets** (1,200–1,500 calories a day), occasionally they recommend a very low calorie liquid protein diet (400–700 calories), with supplementation of vitamins and minerals, for as long as three months. Professional help with behavior modification is of paramount importance in such cases; without changing eating habits and exercise patterns, weight lost will be regained quickly.

Surgery

For individuals who are morbidly obese, surgery to bypass portions of the stomach and small intestine may at times be the only effective means of producing sustained and significant weight loss. Such obesity surgery, however, can be risky, and it is performed only on patients for whom other strategies have failed and whose obesity seriously threatens health. Liposuction is a purely cosmetic procedure in which a suction device is used to remove fat from beneath the skin, and has no place in the treatment of obesity.

Medications

Most of the current research on obesity is aimed at identifying biochemical pathways that will be amenable to **intervention** with drug treatments. These medications would be specifically tailored to interfere with the energy cycles to facilitate weight loss. As of 2002, there are two major classes of drugs that are approved for the treatment of obesity by the U.S. Food and Drug Administration (FDA). History of the field is littered with drugs that have failed or that have caused serious side effects. Appetite suppressant drugs such as Dexatrim and Meridia (sibutramine) change the amounts of some **neurotransmitters** in the brain. These chemical changes result in decreased appetite, but only in the presence of the drug. Digestive inhibitors such as Orlistat (Xenical) are drugs that interfere with the breakdown and absorption of dietary fat in the intestines; they are, however, poorly tolerated by the person who is obese because the effects of fat malabsorption are unpleasant.

These drugs also interfere with the absorption of some necessary vitamins. Fat substitutes such as Olestra, while technically not drugs, attempt to recreate the pleasant taste that fat adds to food, but create the same negative side effects as digestive inhibitors. Unless an obese individual has also made necessary behavioral changes, excess weight returns quickly when **appetite suppressants** or malabsorptive agents are stopped.

The use of any drug is associated with unwanted side effects, so that the decision to take a drug must come after the potential side effects are weighed against the potential benefits. No drug, current or past, has had such dramatic effects on obesity that it warrants its casual use. While most of the immediate side effects that may occur are reversible, the long-term effects, in many cases, are unknown. Even after a new drug successfully negotiates the stringent FDA approval process, its widespread use over a longer time frame may lead to the side effects that were not initially observable in the test population. Two popular obesity drugs of the early 1990s have already been withdrawn from the market because of unanticipated and severe cardiac problems. Meridia, just released in 1997, is already under scrutiny by a consumer group for its relationship to several deaths. Nevertheless, studies show that when obesity drugs are combined with behavioral changes—and especially with a portion controlled diet—weight loss is significantly greater than in a control group treated with behavior modification alone, at least after six months. It remains to be proved whether drug-assisted weight loss is long lasting.

Alternative treatment

The Chinese herb, ephedra (*Ephedra sinica*), combined with caffeine, exercise, and a low-fat diet, can cause a temporary increase in weight loss, at best. However, ephedra and caffeine are both central nervous system (CNS) stimulants, and the large doses of ephedra required to achieve the weight loss can also cause anxiety, irritability, and **insomnia**. Further, ephedra has been implicated in more serious conditions, such as seizure and stroke. Ephedra should not be used by anyone with a history of diabetes, heart disease, or thyroid problems.

HEIGHT AND WEIGHT GOALS			
Men			
Height	***Small Frame***	***Medium Frame***	***Large Frame***
5'2"	128-134 lbs.	131-141 lbs.	138-150 lbs.
5'3"	130-136	133-143	140-153
5'4"	132-138	135-145	142-153
5'5"	134-140	137-148	144-160
5'6"	136-142	139-151	146-164
5'7"	138-145	142-154	149-168
5'8"	140-148	145-157	152-172
5'9"	142-151	148-160	155-176
5'10"	144-154	151-163	158-180
5'11"	146-157	154-166	161-184
6'0"	149-160	157-170	164-188
6'1"	152-164	160-174	168-192
6'2"	155-168	164-178	172-197
6'3"	158-172	167-182	176-202
6'4"	162-176	171-187	181-207
Women			
Height	***Small Frame***	***Medium Frame***	***Large Frame***
4'10"	102-111 lbs.	109-121 lbs.	118-131 lbs.
4'11"	103-113	111-123	120-134
5'0"	104-115	113-126	112-137
5'1"	106-118	115-129	125-140
5'2"	108-121	118-132	128-143
5'3"	111-124	121-135	131-147
5'4"	114-127	124-141	137-151
5'5"	117-130	127-141	137-155
5'6"	120-133	130-144	140-159
5'7"	123-136	133-147	143-163
5'8"	126-139	136-150	146-167
5'9"	129-142	139-153	149-170
5'10"	132-145	142-156	152-176
5'11"	135-148	145-159	155-176
6'0"	138-151	148-162	158-179

Height and weight goals as determined by the Metropolitan Life Insurance Company. *(Source: Based on the height and weight charts by Heart Screen, Inc. Available at <http://heartscreen.com/hw_info.html>.)*

Diuretic herbs, which increase urine production, can cause short-term weight loss, but cannot help patients achieve lasting weight control. The body responds to heightened urine output by increasing thirst to replace lost fluids, and patients who use diuretics for an extended period of time retain water even in the presence of the diuretic. In moderate doses, psyllium, a mucilaginous herb available in bulk-forming laxatives like Metamucil, absorbs fluid and makes patients feel as if they have eaten enough. Red peppers, mustard, and dandelion are said to generate weight loss by accelerating the metabolic rate. Dandelion also counteracts the desire for sweet foods. Walnuts contain serotonin, the brain chemical that signals satiety.

Acupressure and **acupuncture** can also suppress food cravings. Visualization and **meditation** can create

and reinforce a positive self-image that enhances determination to lose weight. By improving physical strength, mental concentration and emotional serenity, **yoga** can provide the same benefits.

The correct balance of the basic food groups is also important, and believed by some experts to enhance the metabolic rate.

Prognosis

As many as 85% of dieters who do not exercise on a regular basis regain their lost weight within two years. In five years, the figure rises to 90%. Repeatedly losing and regaining weight (yo-yo dieting) encourages the body to store fat and may increase a patient's risk of developing heart disease. The primary factor in achieving and maintaining weight loss is a lifelong commitment to regular exercise and sensible eating habits.

Prevention

Obesity experts suggest that a key to preventing excess weight gain is monitoring fat consumption rather than counting calories; in fact, the National Cholesterol Education Program maintains that only 30% of calories should be derived from fat. Only one-third of those calories should come from saturated fats (the kind of fat found in high concentrations in meat, poultry, and dairy products). However, total caloric intake cannot be ignored, since it usually the slow accumulation of excess caloric intake, regardless of its source, that results in obesity. Erring on the side of 25 excess calories a day, a single cookie will result in a five-pound weight gain by the end of a year. Without recognition of the problem, weight balloons up another 45 pounds by the end of 10 years, and the return to normal weight is an arduous process. Because most people eat more than they think they do, keeping a detailed and honest food diary is a useful way to recognize eating habits. Eating three balanced, moderate-portion meals a day—with the main meal at mid-day—is a more effective way to prevent obesity than fasting or crash diets, which convince the body that there is an ongoing famine. After 12 hours without food, the body has depleted its stores of readily available energy, and hunkers down to begin protecting itself for the long term. Metabolic rate starts to slow, and breakdown of muscle tissue for the raw materials needed for energy maintenance begins. Until more food appears, famine mode persists and deepens; when the fast is lifted, the body is in a state of slowed metabolism, has a bit less muscle, and requires less food than before the fast. Exercise increases the metabolic rate by creating muscle, which burns more calories than fat. When regular exercise is combined with consistent, healthful meals, calories continue to burn at an accelerated rate for several hours.

Finally, encouraging healthful habits in children is a key to preventing childhood obesity and the health problems that follow in adulthood.

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American Obesity Association. 1250 24th St. NW, Washington D.C. 20037. <http://www.obesity.org>.

Shape Up America. 6707 Democracy Blvd., Suite 306, Bethesda, MD 20817. <http://www.shapeup.org/general/index.html>.

Weight-Control Information Network. 1 Win Way, Bethesda, MD 20892-3665. <http://www.niddk.nih.gov/health/nutrit/win.html>.

Elizabeth Reid, M.D.

Obesity

Definition

An obsession is an unwelcome, uncontrollable, and persistent idea, thought, image, or emotion that a person

cannot help thinking even though it creates significant distress or anxiety.

Description

Obsessive ideas seem unnatural or alien to those who have them, but are nevertheless recognized as originating from the person's own thoughts—they are not seen as **delusions** sent or controlled by an outside party.

Typical obsessions include fear of contamination as from doorknobs or handshakes, worry about leaving things in their proper order, persistent doubts about one's responsible behavior, scary images involving violent acts, and images of sexual acts. People with obsessions may find themselves acting in compulsive ways in largely futile attempts to relieve the anxiety associated with their persistent, unpleasant thoughts. Others suffering from obsessions may try very hard to control or ignore them. It is important to note that legitimate worries about daily concerns—paying bills, studying for exams, keeping a job, interpersonal relationships—are not obsessions. Although they can occasionally be carried to obsessive lengths, these concerns can change with circumstances and, in most cases be controlled, with planning, effort, and action. Obsessions relate to problems that most people would consider far removed from normal, daily events and concerns.

See also Compulsion; Obsessive-compulsive disorder

Dean A. Haycock, Ph.D.

Obsessive-compulsive disorder

Definition

Obsessive-compulsive disorder (OCD) is currently classified as an anxiety disorder marked by the recurrence of intrusive or disturbing thoughts, impulses, images or ideas (obsessions) accompanied by repeated attempts to suppress these thoughts through the performance of certain irrational and ritualistic behaviors or mental acts (compulsions). The obsessions and compulsions take up large amounts of the patient's time (an hour or longer every day) and usually cause significant emotional distress for the patient and difficulties in his or her relationships with others.

Some researchers have questioned whether OCD really belongs with the other anxiety disorders. They think that it should be grouped with the spectrum of such obsessive-compulsive disorders as Tourette's syndrome, which are known to have biological causes.

OCD should not be confused with **obsessive-compulsive personality disorder** even though the two disorders have similar names. Obsessive-compulsive personality disorder is not characterized by the presence of obsessions and compulsions; rather, it is a lifelong pattern of insistence on control, orderliness, and perfection that begins no later than the early adult years. It is possible, however, for a person to have both disorders.

Description

Obsessive-compulsive disorder is a mental disorder with two components: obsessions, which consist of thoughts, impulses, or mental images; and compulsions, which are repetitive behaviors that the person feels driven to perform in response to the obsessions. In some cases, the **compulsion** may represent a strict rule that the patient must apply rigidly in every situation (tying one's shoes a certain number of times, for example) in order to feel "right." The exact content of obsessions varies from person to person, although certain themes are common. People with OCD experience their disturbing thoughts and images as intrusive and troublesome, but they recognize that their thoughts are products of their own minds. Obsessive thoughts are different from worries about such real-life problems as losing one's job or bad grades in school. In addition, obsessive thoughts are not usually related to any real-life problems.

The most common types of obsessions in persons with OCD in Western countries are:

- fear of contamination (impurity, pollution, badness)
- doubts (worrying about whether one has omitted to do something)
- an intense need to have or put things in a particular order
- aggressive or frightening impulses
- recurrent sexual thoughts or images

It is important to understand that patients diagnosed with OCD do not perform their compulsions for pleasure or satisfaction. A compulsive behavior becomes linked to an obsessional thought because the behavior lowers the level of anxiety produced by the obsession(s).

The most common compulsions in Western countries are:

- washing/cleaning
- counting

KEY TERMS

Basal ganglia—A group of masses of gray matter located in the cerebral hemispheres of the brain that control movement as well as some aspects of emotion and cognition.

Behavioral therapy—An approach to treatment that focuses on extinguishing undesirable behavior and replacing it with desired behavior.

Cognitive-behavioral therapy—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

Compulsion—A strong impulse to perform an act, particularly one that is irrational or contrary to one's will.

Epidemiology—The study of the causes, incidence, transmission, and control of diseases.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Obsession—A persistent image, idea, or desire that dominates a person's thoughts or feelings.

Onset—The point in time at which the symptoms of a disorder first became apparent.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Streptococcus (plural, streptococci)—A type of bacterium that is spherical in shape and occurs in chains or pairs. Some diseases that are caused by streptococci appear to be related to OCD.

Sydenham's chorea—A serious manifestation of actue rheumatic fever that commonly occurs in children ages seven through 14, peaking at age eight. This disease of the central nervous system is characterized by emotional instability, purposeless movements, and muscular weakness. At its peak in the 1950s it occured in nearly 50% of the acute rheumatic fever cases, but by 2002 had subsided to a degree of less than 10% of the acute cases.

Tic—A sudden involuntary behavior that is difficult or impossible for the person to suppress. Tics may be either motor (related to movement) or vocal, and may become more pronounced under stress.

Trichotillomania—A disorder marked by repeated pulling and tugging of one's hair, usually resulting in noticeable hair loss on the scalp or elsewhere on the body.

- hoarding
- checking
- putting objects in a certain order
- repeated "confessing" or asking others for assurance
- repeated actions
- making lists

Although descriptions of patients with OCD have been reported since the fifteenth century in religious and psychiatric literature, the condition was widely assumed to be rare until very recently. Epidemiological research since 1980 has now identified OCD as the fourth most common psychiatric illness, after phobias, substance use disorders, and major depressive disorders. OCD is presently classified as a form of anxiety disorder, but current studies indicate that it results from a combination of psychological, neurobiological, genetic, and environmental causes.

Causes and symptoms

Causes

PSYCHOSOCIAL. In the early part of the century, Sigmund Freud theorized that OCD symptoms were caused by punitive, rigid toilet-training practices that led to internalized conflicts. Other theorists thought that OCD was influenced by such wider cultural attitudes as insistence on cleanliness and neatness, as well as by the attitudes and parenting style of the patient's parents. Cross-cultural studies of OCD indicate that, while the incidence of OCD seems to be about the same in most countries around the world, the symptoms are often shaped by the patient's culture of origin. For example, a patient from a Western country may have a contamination **obsession** that is focused on germs, whereas a patient from India may fear contamination by touching a person from a lower social caste.

Studies of families with OCD members indicate that the particular expression of OCD symptoms may be

affected by the responses of other people. Families with a high tolerance for the symptoms are more likely to have members with more extreme or elaborate symptoms. Problems often occur when the OCD member's obsessions and rituals begin to control the entire family.

BIOLOGICAL. There is considerable evidence that OCD has a biological component. Some researchers have noted that OCD is more common in patients who have suffered head trauma or have been diagnosed with Tourette's syndrome. Recent studies using **positron emission tomography** (PET) scanning indicate that OCD patients have patterns of **brain** activity that differ from those of people without mental illness or with some other mental illness. Other studies using **magnetic resonance imaging** (MRI) found that patients diagnosed with OCD had significantly less white matter in their brains than did normal control subjects. This finding suggests that there is a widely distributed brain abnormality in OCD. Some researchers have reported abnormalities in the metabolism of serotonin, an important neurotransmitter, in patients diagnosed with OCD. Serotonin affects the efficiency of communication between the front part of the brain (the cortex) and structures that lie deeper in the brain known as the basal ganglia. Dysfunction in the serotonergic system occurs in certain other mental illnesses, including major depression. OCD appears to have a number of features in common with the so-called obsessive-compulsive spectrum disorders, which include Tourette's syndrome; Sydenham's chorea; eating disorders; **trichotillomania**; and delusional disorders.

There appear to be genetic factors involved in OCD. The families of persons who are diagnosed with the disorder have a greater risk of OCD and **tic disorders** than does the general population. Childhood-onset OCD appears to run in families more than adult-onset OCD, and is more likely to be associated with tic disorders. Twin studies indicate that monozygotic, or identical twins, are more likely to share the disorder than dizygotic, or fraternal twins. The concordance (match) rate between identical twins is not 100%, however, which suggests that the occurrence of OCD is affected by environmental as well as genetic factors. In addition, it is the general nature of OCD that seems to run in families rather than the specific symptoms; thus, one family member who is affected by the disorder may have a compulsion about washing and cleaning while another is a compulsive counter.

Large epidemiological studies have found a connection between streptococcal infections in childhood and the abrupt onset or worsening of OCD symptoms. The observation that there are two age-related peaks in the onset of the disorder increases the possibility that there is a common causal factor. Patients with childhood-onset OCD often have had one of two diseases caused by a group of bacteria called Group A beta-hemolytic streptococci ("strep" throat and Sydenham's chorea) prior to the onset of the OCD symptoms. The disorders are sometimes referred to as pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections, or PANDAS. It is thought that antibodies in the child's blood cross-react with structures in the basal ganglia, producing or worsening the symptoms of OCD or tic disorders.

Symptoms

The symptoms of OCD should not be confused with the ability to focus on detail or to check one's work that is sometimes labeled "compulsive" in everyday life. This type of attentiveness is an important factor in academic achievement and in doing well in fields that require close attention to detail, such as accounting or engineering. By contrast, the symptoms of OCD are serious enough to interfere with the person's day-to-day functioning. Historical examples of OCD include a medieval Englishman named William of Oseney, who spent twelve hours per day reading religious books in order to be at peace with God; and Freud's Rat Man, a patient who had repeated dreams of cursing Freud and covering him with dung. While the Rat Man was ashamed of these impulses and had no explanation for them, he could not control them.

More recent accounts of OCD symptoms include those of a young man who compulsively touched every electrical outlet as he passed, washed his hands several times an hour, and returned home repeatedly to check that the doors and windows were locked. Another account describes a firefighter who was worried that he had throat cancer. He spent three hours a day examining his throat in the mirror, feeling his lymph nodes, and asking his wife if his throat appeared normal.

Brief descriptions of the more common obsessions and compulsions follow.

CONTAMINATION. People with contamination obsessions are usually preoccupied with a fear of dirt or germs. They may avoid leaving home or allowing visitors to come inside in order to prevent contact with dirt or germs. Some people with contamination obsessions may wear gloves, coats, or even masks if they are forced to leave their house for some reason. Obsessions with contamination may also include abnormal fears of such environmental toxins as lead, asbestos, or radon.

Washing compulsions are commonly associated with contamination obsessions. For example, a person concerned about contamination from the outside may shower and launder all clothing immediately upon coming home. The compulsion may be triggered by direct contact with the feared object, but in many cases, even being

in its general vicinity may stir up intense anxiety and a strong need to engage in a washing compulsion. One man who was afraid of contamination could not even take a short walk down the street without experiencing a compulsion to disinfect the soles of his shoes, launder all his clothing, and wash his hands until they were raw after he returned to his apartment.

Washing compulsions may not always be caused by a fear of germs. That is, a need for perfection or for symmetry may also lead to unnecessary washing. In such cases, the individual may be concerned about being "perfectly" clean, or feel that he cannot leave the shower until his left foot has been washed exactly as many times as his right foot. Other people with washing compulsions may be unable to tolerate feeling sweaty or otherwise not clean.

OBSESSIONAL DOUBTING. Obsessional doubting refers to the fear of having failed to perform some task adequately, and that dire consequences will follow as a result. Although the person may try to suppress the worrisome thoughts or images, he or she usually experiences a rising anxiety which then leads to a compulsion to check the task. For example, someone may worry about forgetting to lock the door or turn off the gas burner on the stove and spend hours checking these things before leaving home. In one instance, a man was unable to throw away old grocery bags because he feared he might have left something valuable inside one of them. Immediately after looking into an empty bag, he would again have the thought, "What if I missed something in there?" In many cases, no amount of checking is sufficient to dispel the maddening sense of doubt.

NEED FOR SYMMETRY. Persons suffering from an obsession about symmetry often report feeling acutely uncomfortable unless they perform certain tasks in a symmetrical or balanced manner. Thus, crossing one's legs to the right must be followed by crossing legs to the left; scratching one side of the head must be followed by scratching the other; tapping the wall with a knuckle on the right hand must be followed by tapping with one on the left, etc. Sometimes the person may have a thought or idea associated with the compulsion, such as a fear that a loved one will be harmed if the action is not balanced, but often there is no clearly defined fear, only a strong sense of uneasiness.

AGGRESSIVE AND SEXUAL OBSESSIONS. Aggressive and sexual obsessions are often particularly horrifying to those who experience them. For some people, obsessive fears of committing a terrible act in the future compete with fears that they may already have done something awful in the past. Compulsions to constantly check and confess cause such individuals to admit to evildoing they had no part in, a phenomenon familiar to law enforcement following highly publicized crimes. These obsessions often involve violent or graphic imagery that is upsetting and disgusting to the person, such as rape, physical assault, or even murder. One case study concerned a young woman who constantly checked the news to reassure herself that she had not murdered anyone that day; she felt deeply upset by unsolved murder cases. A middle-aged man repeatedly confessed to having molested a woman at work, despite no evidence of such an action ever occurring in his workplace.

SYMPTOMS IN CHILDREN. Obsessions and compulsions in children are often focused on germs and fears of contamination. Other common obsessions include fears of harm coming to self or others; fears of causing harm to another person; obsessions about symmetry; and excessive moralization or religiosity. Childhood compulsions frequently include washing, repeating, checking, touching, counting, ordering and arranging. Younger children are less likely to have full-blown anxiety-producing obsessions, but they often report a sense of relief or strong satisfaction (a "just right" feeling) from completing certain ritualized behaviors. Since children are particularly skillful in disguising their OCD symptoms from adults, they may effectively hide their disorder from parents and teachers for years.

Unusual behaviors in children that may be signs of OCD include:

- Avoidance of scissors or other sharp objects. A child may be obsessed with fears of hurting herself or others.
- Chronic lateness or dawdling. The child may be performing checking rituals (repeatedly making sure all her school supplies are in her bookbag, for example).
- Daydreaming or preoccupation. The child may be counting or performing balancing rituals mentally.
- Spending long periods of time in the bathroom. The child may have a handwashing compulsion.
- Schoolwork handed in late or papers with holes erased in them. The child may be repeatedly checking and correcting her work.

For both children and adults, the symptoms of OCD wax and wane in severity; and the specific content of obsessions and compulsions may change over time. The disorder, however, very seldom goes away by itself without treatment. People with OCD in all age groups typically find that their symptoms worsen during major life changes or following highly stressful events.

Demographics

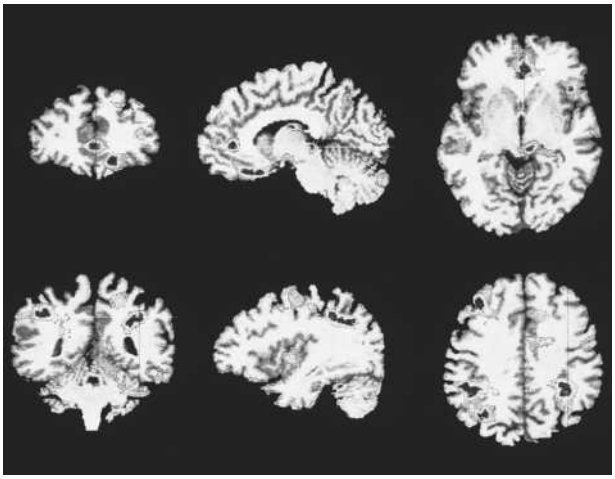
As noted above, OCD is a relatively common mental disorder, with about 2.3% of the population of the United States being diagnosed with the condition at some point in their lives. As of 2000, the annual social and economic costs of OCD in the United States are estimated at $9 billion. Although the disorder may begin at any age, the typical age of onset is late adolescence to young adulthood, with slightly more women than men being diagnosed with OCD. Interestingly, childhood OCD is more common in males, and the sex ratio does not favor females until adulthood. People with OCD appear to be less likely to marry than persons diagnosed with other types of mental disorders.

Diagnosis

OCD is a disorder that may not be diagnosed for years. People who suffer from its symptoms are often deeply ashamed, and go to great lengths to hide their ritualistic behaviors. The disorder may be diagnosed when family members get tired of the impact of the patient's behaviors on their lives, and force the patient to consult a doctor. In other cases, the disorder may be self-reported. The patient may have come to resent the amount of time wasted by the compulsions; or he or she may have taken a screening questionnaire such as the brief screener available on the NIMH website (listed in the Resources section below).

The **diagnosis** of OCD may be complicated because of the number of other conditions that resemble it. For example, major depression may be associated with self-perceptions of being guilty, bad, or worthless that are excessive and unreasonable. Similarly, eating disorders often include bizarre thoughts about size and weight, ritualized eating habits, or the hoarding of food. Delusional disorders may entail unusual beliefs or behaviors, as do such other mental disorders as trichotillomania, **hypochondriasis**, the **paraphilias**, and substance use disorders. Thus, accurate diagnosis of OCD depends on the careful analysis of many variables to determine whether the apparent obsessions and compulsions might be better accounted for by some other disorder, or to the direct effects of a substance or a medical condition.

In addition, OCD may coexist with other mental disorders, most commonly depression. It has been estimated that about 34% of patients diagnosed with OCD are depressed at the time of diagnosis, and that 65% will develop depression at some point in their lives.



Colored positron emission tomography scans (PET scans) of a human brain, showing active areas in obsessive-compulsive disorder. In this patient, some parts of the brain show increased activity as the symptoms strengthen (areas shown in the top row), while other brain areas show decreased activity as symptoms strengthen (bottom row). *(Wellcome Dept of Cognitive Neurology. Photo Researchers, Inc./ Science Photo Library. Reproduced by permission.) See color insert for color version of photo.*

Treatments

As of 2002, a combination of behavioral therapy and medications appears to be the most effective treatment for OCD. The goal of treatment is to reduce the frequency and severity of the obsessions and compulsions so that the patient can work more efficiently and have more time for social activities. Few OCD patients become completely symptom-free, but most benefit considerably from treatment.

Psychotherapy

Behavioral treatments using the technique of exposure and response prevention are particularly effective in treating OCD. In this form of therapy, the patient and therapist draw up a list, or hierarchy, of the patient's obsessive and compulsive symptoms. The symptoms are arranged in order from least to most upsetting. The patient is then systematically exposed to the anxiety-producing thoughts or behaviors, beginning with the least upsetting. The patient is asked to endure the feared event or image without engaging in the compulsion normally used to lower anxiety. For example, a person with a contamination obsession might be asked to touch a series of increasingly dirty objects without washing their hands. In this way, the patient learns to tolerate the feared object, reducing both worrisome obsessions and anxiety-reducing compulsions. About 75%–80% of patients respond

well to exposure and response prevention, with very significant reductions in symptoms.

Other types of **psychotherapy** have met with mixed results. **Psychodynamic psychotherapy** is helpful to some patients who are concerned about the relationships between their upbringing and the specific features of their OCD symptoms. Cognitive-behavioral psychotherapy may be valuable in helping the patient to become more comfortable with the prospect of exposure and prevention treatments, as well as helping to identify the role that the patient's particular symptoms may play in his or her own life and what effects family members may have on the maintenance and continuation of OCD symptoms. Cognitive-behavioral psychotherapy is not intended to replace exposure and response prevention, but may be a helpful addition to it.

Medications

The most useful medications for the treatment of OCD are the selective serotonin reuptake inhibitors (SSRIs), which affect the body's reabsorption of serotonin, a chemical in the brain that helps to transmit nerve impulses across the very small gaps between nerve cells. These drugs, specifically **clomipramine** (Anafranil), **fluoxetine** (Prozac), **fluvoxamine** (Luvox), **sertraline** (Zoloft), and **paroxetine** (Paxil) have been found to relieve OCD symptoms in over half of the patients studied. It is not always possible for the doctor to predict which of the SSRIs will work best for a specific patient. Lack of response to one SSRI does not mean that other drugs within the same family will not work. Treatment of OCD often proceeds slowly, with various medications being tried before the most effective one is found. While studies report that about half of those treated with SSRIs show definite improvement, relapse rates may be as high as 90% when medications are discontinued.

Other mainstream approaches

Some treatments that have been used for OCD include **electroconvulsive therapy** (ECT) and, as a technique of last resort, **psychosurgery** for truly intractable OCD. Some patients have benefited from ECT; however, the National Institute of Mental Health (NIMH) recommends reserving ECT for OCD patients who have not responded to psychotherapy or medication.

Prognosis

While most patients with OCD benefit from a combination of medications and psychotherapy, the disorder is usually a lifelong condition. In addition, the presence of **personality disorders** or additional mental disorders is associated with less favorable results from treatment. The total elimination of OCD symptoms is very rare, even with extended treatment.

The onset of OCD in childhood is the single strongest predictor of a poor prognosis. Treatment in children is also complicated by the fact that children may find the response and exposure techniques very stressful. It is also hard for children to understand the potential value of such treatments; however, creative therapists have learned to use anxiety reduction strategies, education, and behavioral rewards to help their young patients with the treatment tasks. Concern about the long-term use of medications in children with OCD has further encouraged the use of cognitive-behavioral techniques whenever possible.

See also: Exposure treatment; Tic disorders

Resources

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McLean, Peter D. and others. "Cognitive Versus Behavioral Therapy in the Group Treatment of Obsessive-Compulsive Disorder." *Journal of Consulting and Clinical Psychology* 69, no.2 (2001): 205-214.

ORGANIZATIONS

Anxiety Disorders Association of America (ADAA). 11900 Parklawn Drive, Suite 100, Rockville, MD 20852-2624. (301) 231-9350. <www.adaa.org>.

Freedom From Fear. 308 Seaview Avenue, Staten Island, NY 10305 (718) 351-1717. <www.freedomfromfear.com>.

Obsessive-Compulsive Foundation, Inc. 337 Notch Hill Road, North Branford, CT 06471. (203) 315-2196. <www.ocfoundation.org>.

OTHER

National Institute of Mental Health (NIMH). *Obsessive-Compulsive Disorder,* 3rd revised edition, 1999. NIH Publication No. 99-3755. <www.nimh.nih.gov/publicat/ocd.cfm>.

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Jane A. Fitzgerald, Ph.D.

KEY TERMS

Anankastic personality disorder—The European term for obsessive-compulsive personality disorder.

Compulsion—A strong impulse to perform an act, particularly one that is irrational or contrary to one's will.

Obsession—A persistent image, idea, or desire that dominates a person's thoughts or feelings.

Therapeutic alliance—The technical term for the cooperative relationship between therapist and patient that is considered essential for successful psychotherapy.

Obsessive-compulsive personality disorder

Definition

Obsessive-compulsive personality disorder (OCPD) is a type of personality disorder marked by rigidity, control, perfectionism, and an overconcern with work at the expense of close interpersonal relationships. Persons with this disorder often have trouble relaxing because they are preoccupied with details, rules, and productivity. They are often perceived by others as stubborn, stingy, self-righteous, and uncooperative.

The mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (2000), which is also called *DSM-IV-TR*, groups obsessive-compulsive personality disorder together with the avoidant and dependent **personality disorders** in Cluster C. The disorders in this cluster are considered to have anxiety and fearfulness as common characteristics. The ICD-10, which is the European counterpart of *DSM-IV-TR*, refers to OCPD as "anankastic personality disorder."

It is important to distinguish between OCPD and **obsessive-compulsive disorder** (OCD), which is an anxiety disorder characterized by the presence of intrusive or disturbing thoughts, impulses, images or ideas (obsessions), accompanied by repeated attempts to suppress these thoughts through the performance of irrational and ritualistic behaviors or mental acts (compulsions). It is unusual but possible, however, for a patient to suffer from both disorders, especially in extreme cases of hoarding behavior. In some reported cases of animal hoarding, the people involved appear to have symptoms of both OCD and OCPD.

Description

People suffering from OCPD have careful rules and procedures for conducting many aspects of their everyday lives. While their goal is to accomplish things in a careful, orderly manner, their desire for perfection and insistence on going "by the book" often overrides their ability to complete a task. For example, one patient with OCPD was so preoccupied with finding a mislaid shopping list that he took much more time searching for it than it would have taken him to rewrite the list from memory. This type of inflexibility typically extends to interpersonal relationships. People with OCPD are known for being highly controlling and bossy toward other people, especially subordinates. They will often insist that there is one and only one right way (their way) to fold laundry, cut grass, drive a car, or write a report. In addition, they are so insistent on following rules that they cannot allow for what most people would consider legitimate exceptions. Their attitudes toward their own superiors or supervisors depend on whether they respect these authorities. People with OCPD are often unusually courteous to superiors that they respect, but resistant to or contemptuous of those they do not respect.

While work environments may reward their conscientiousness and attention to detail, people with OCPD do not show much spontaneity or imagination. They may feel paralyzed when immediate action is necessary; they feel overwhelmed by trying to make decisions without concrete guidelines. They expect colleagues to stick to detailed rules and procedures, and often perform poorly in jobs that require flexibility and the ability to compromise. Even when people with OCPD are behind schedule, they are uncomfortable delegating work to others because the others may not do the job "properly." People

with OCPD often get so lost in the finer points of a task that they cannot see the larger picture; they are frequently described as "unable to see the forest for the trees." They are often highly anxious in situations without clearly defined rules because such situations arouse their fears of making a mistake and being punished for it. An additional feature of this personality disorder is stinginess or miserliness, frequently combined with an inability to throw out worn-out or useless items. This characteristic has sometimes been described as "pack rat" behavior.

People diagnosed with OCPD come across to others as difficult and demanding. Their rigid expectations of others are also applied to themselves, however; they tend to be intolerant of their own shortcomings. Such persons feel bound to present a consistent facade of propriety and control. They feel uncomfortable with expressions of tender feelings and tend to avoid relatives or colleagues who are more emotionally expressive. This strict and ungenerous approach to life limits their ability to relax; they are seldom if ever able to release their needs for control. Even recreational activities frequently become another form of work. A person with OCPD, for example, may turn a tennis game into an opportunity to perfect his or her backhand rather than simply enjoying the exercise, the weather, or the companionship of the other players. Many OCPD sufferers bring office work along on vacations in order to avoid "wasting time," and feel a sense of relief upon returning to the structure of their work environment. Not surprisingly, this combination of traits strains their interpersonal relationships and can lead to a lonely existence.

Causes and symptoms

Causes

No single specific cause of OCPD has been identified. Since the early days of Freudian **psychoanalysis**, however, faulty parenting has been viewed as a major factor in the development of personality disorders. Current studies have tended to support the importance of early life experiences, finding that healthy emotional development largely depends on two important variables: parental warmth and appropriate responsiveness to the child's needs. When these qualities are present, the child feels secure and appropriately valued. By contrast, many people with personality disorders did not have parents who were emotionally warm toward them. Patients with OCPD often recall their parents as being emotionally withholding and either overprotective or overcontrolling. One researcher has noted that people with OCPD appear to have been punished by their parents for every transgression of a rule, no matter how minor, and rewarded for almost nothing. As a result, the child is unable to safely develop or express a sense of joy, spontaneity, or independent thought, and begins to develop the symptoms of OCPD as a strategy for avoiding punishment. Children with this type of upbringing are also likely to choke down the anger they feel toward their parents; they may be outwardly obedient and polite to authority figures, but at the same time treat younger children or those they regard as their inferiors harshly.

Genetic contributions to OCPD have not been well documented. Cultural influences may, however, play a part in the development of OCPD. That is, cultures that are highly authoritarian and rule-bound may encourage child-rearing practices that contribute to the development of OCPD. On the other hand, simply because a culture is comparatively strict or has a strong work ethic does not mean it is necessarily unhealthful. In Japanese societies, for example, excessive devotion to work, restricted emotional expression, and moral scrupulosity are highly valued characteristics that are rewarded within that culture. Similarly, certain religions and professions require exactness and careful attention to rules in their members; the military is one example. OCPD is not diagnosed in persons who are simply behaving in accordance with such outside expectations as military regulations or the rule of a religious order. Appropriate evaluation of persons from other cultures requires close examination in order to differentiate people who are merely following culturally prescribed patterns from people whose behaviors are excessive even by the standards of their own culture.

Symptoms

The symptoms of OCPD include a pervasive overconcern with mental, emotional, and behavioral control of the self and others. Excessive conscientiousness means that people with this disorder are generally poor problem-solvers and have trouble making decisions; as a result, they are frequently highly inefficient. Their need for control is easily upset by schedule changes or minor unexpected events. While many people have some of the following characteristics, a person who meets the *DSM-IV-TR* criteria for OCPD must display at least four of them:

- Preoccupation with details, rules, lists, order, organization, or schedules to the point at which the major goal of the activity is lost.
- Excessive concern for perfection in small details that interferes with the completion of projects.
- Dedication to work and productivity that shuts out friendships and leisure-time activities, when the long hours of work cannot be explained by financial necessity.

- Excessive moral rigidity and inflexibility in matters of ethics and values that cannot be accounted for by the standards of the person's religion or culture.
- Hoarding things, or saving worn-out or useless objects even when they have no sentimental or likely monetary value.
- Insistence that tasks be completed according to one's personal preferences.
- Stinginess with the self and others.
- Excessive rigidity and obstinacy.

Demographics

Obsessive-compulsive personality disorder is estimated to occur in about 1% of the population, although rates of 3%–10% are reported among psychiatric outpatients. The disorder is usually diagnosed in late adolescence or young adulthood. In the United States, OCPD occurs almost twice as often in men as in women. Some researchers attribute this disproportion to gender stereotyping, in that men have greater permission from general Western culture to act in stubborn, withholding, and controlling ways.

Diagnosis

It is relatively unusual for OCPD to be diagnosed as the patient's primary reason for making an appointment with their doctor. In many cases the person with OCPD is unaware of the discomfort that his or her stubbornness and rigidity cause other people, precisely because these traits usually enable them to get their way with others. They are more likely to enter therapy because of such other issues as anxiety disorders, serious relationship difficulties, or stress-related medical problems. **Diagnosis** of OCPD depends on careful observation and appropriate assessment of the individual's behavior; the person must not only give evidence of the attitudes and behaviors associated with OCPD, but these must be severe enough to interfere with their occupational and interpersonal functioning.

The differential diagnosis will include distinguishing between obsessive-compulsive disorder (OCD) and OCPD. A person who has obsessions and compulsions that they experience as alien and irrational is more likely to be suffering from OCD, whereas the person who feels perfectly comfortable with self-imposed systems of extensive rules and procedures for mopping the kitchen floor probably has OCPD. In addition, the thoughts and behaviors that are found in OCD are seldom relevant to real-life problems; by contrast, people with OCPD are preoccupied primarily with managing (however inefficiently) the various tasks they encounter in their daily lives.

Some features of OCPD may occur in other personality disorders. For example, a person with a **narcissistic personality disorder** may be preoccupied with perfection and be critical and stingy toward others; narcissists are usually generous with themselves, however, while people with OCPD are self-critical and reluctant to spend money even on themselves. Likewise, a person with **schizoid personality disorder**, who lacks a fundamental capacity for intimacy, may resemble someone with OCPD in being formal and detached in dealing with others. The difference here is that a person with OCPD, while awkward in emotional situations, is able to experience caring and may long for close relationships. Certain medical conditions may also mimic OCPD, but are distinct in that the onset of the symptoms is directly related to the illness. Certain behaviors related to substance abuse may also be mistaken for symptoms of OCPD, especially if the substance problem is unrecognized.

As described earlier, diagnosis may also be complicated by the fact that behaviors similar to OCPD may be normal variants within a given culture, occupation, or religion; however, in order to fulfill criteria for the personality disorder, the behaviors must be sufficiently severe as to impair the patient's functioning.

Treatments

Psychotherapy

Psychotherapeutic approaches to the treatment of OCPD have found insight-oriented psychodynamic techniques and cognitive behavioral therapy to be helpful for many patients. This choice of effective approaches stands in contrast to the limitations of traditional forms of **psychotherapy** with most patients diagnosed with OCD. Learning to find satisfaction in life through close relationships and recreational outlets, instead of only through work-related activities, can greatly enrich the OCPD patient's quality of life. Specific training in relaxation techniques may help patients diagnosed with OCPD who have the so-called "Type A" characteristics of competitiveness and time urgency as well as preoccupation with work.

It is difficult, however, for a psychotherapist to develop a therapeutic alliance with a person with OCPD. The patient comes into therapy with a powerful need to control the situation and the therapist; a reluctance to trust others; and a tendency to doubt or question almost everything about the therapy situation. The therapist must be alert to the patient's defenses against genuine change and work to gain a level of commitment to the therapeutic process. Without this commitment, the thera-

pist may be fooled into thinking that therapy has been successful when, in fact, the patient is simply being superficially compliant.

Medications

For many years, medications for OCPD and other personality disorders were thought to be ineffective since they did not affect the underlying causes of the disorder. More recent studies, however, indicate that treatment with specific drugs may be a useful adjunct (help) to psychotherapy. In particular, the medications known as selective serotonin reuptake inhibitors (SSRIs) appear to help the OCPD patient with his or her rigidity and compulsiveness, even when the patient did not show signs of pre-existing depression. Medication can also help the patient to think more clearly and make decisions better and faster without being so distracted by minor details. While symptom control may not "cure" the underlying personality disorder, medication does enable some OCPD patients to function with less distress.

Prognosis

Individuals with OCPD often experience a moderate level of professional success, but relationships with a spouse or children may be strained due to their combination of emotional detachment and controlling behaviors. In addition, people with OCPD often do not attain the level of professional achievement that might be predicted for their talents and abilities because their rigidity and stubbornness make them poor "team players" or supervisors. Although there are few large-scale outcome studies of treatments for OCPD, existing reports suggest that these patients do benefit from psychotherapy to help them understand the emotional issues underlying their controlling behaviors and to teach them how to relax. Since OCPD sufferers, unlike people with OCD, usually view their compulsive behaviors as voluntary, they are better able to consider change, especially as they come to fully recognize the personal and interpersonal costs of their disorder.

Prevention

Most theories attribute the development of OCPD to early life experiences, including a lack of parental warmth; parental overcontrol and rigidity, and few rewards for spontaneous emotional expression. Little work has been done, however, in identifying preventive strategies.

See also Gender issues in mental health

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Anxiety Disorders Association of America (ADAA). 11900 Parklawn Drive, Suite 100, Rockville, MD 20852-2624. (301) 231-9350. <www.adaa.org>.

Freedom From Fear. 308 Seaview Avenue, Staten Island, NY 10305. (718) 351-1717. <www.freedomfromfear.com>.

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Olanzapine

Definition

Olanzapine is classified as an atypical antipsychotic drug. It is available in the United States under the brand names Zyprexa and Zyprexa Zydis.

Purpose

Olanzapine is used to treat **schizophrenia**, to control manic episodes of **bipolar disorder** (manic-depressive disorder), or to treat **dementia** related to **Alzheimer's disease**.

Description

Olanzapine is thought to modify the actions of several chemicals in the **brain**. Onlanzapine is chemically related to another atypical antipsychotic agent, **clozapine**, but differs both chemically and pharmacologically from the earlier phenothiazine antipsychotics.

Olanzapine is available as 2.5-mg, 5-mg, 7.5-mg, 10-mg, 15-mg, and 20-mg tablets that can be swallowed (Zyprexa) and 5-mg, 10-mg, 15-mg, and 20-mg tablets that disintegrate when placed under the tongue (Zyprexa Zydis). Olanzapine is broken down by the liver.

Recommended dosage

The dosage of olanzapine varies depending upon the reason for its use. When used to treat schizophrenia, 5–10 mg is the typical starting dosage. If dosage adjustments are needed, increases are made in 5-mg increments once a week. When treating schizophrenia, a total daily dosage of 10–15 mg is usually effective. When olanzapine is used to treat acute manic episodes, initial doses of olanzapine are often 10–15 mg; 20 mg per day may be needed for maximum effect. The safety of doses greater than 20 mg per day has not been determined.

Olanzapine is eliminated from the body more quickly in young people than in older (over age 60) individuals, in men than in women, and in smokers faster than in non-smokers. Dosage adjustments may be needed based upon individual patient characteristics.

Precautions

Caution should be used in patients with heart disease because the drug may cause blood pressure to fall too low resulting in dizziness, rapid heartbeats, or fainting. Olanzapine should be used carefully in people with known seizure disorders since olanzapine may alter properties of the brain making **seizures** occur more easily. People with liver disease should have their liver function monitored regularly while taking olanzapine. Women who are pregnant or breast-feeding should not take olanzapine. People with phenylketonuria, a disorder in which the body is unable to metabolize a protein called phenylalanine, should avoid olanzapine disintegrating tablets, because this form of the drug contains phenylalanine.

KEY TERMS

Antipsychotic—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Bipolar disorder (formerly manic-depressive disorder)—A mental disorder characterized by dramatic, and sometimes rapid mood swings, resulting in both manic and depressive episodes.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Neuroleptic malignant syndrome—An unusual but potentially serious complication that develops in some patients who have been treated with antipsychotic medications. NMS is characterized by changes in blood pressure, altered states of consciousness, rigid muscles, and fever. Untreated NMS can result in coma and death.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

Side effects

Side effects that occur in more than 5% of patients taking olanzapine include involuntary movements, weakness, dizziness, extreme drowsiness, nonviolent objectionable behavior, constipation, weight gain, dry mouth, low blood pressure, stomach upset, increased appetite, cold-such as symptoms, or fever.

Other side effects that are possible include rash, body aches and pains, elevated liver enzymes, vision abnormalities, chest pain, or rapid heartbeats.

Olanzapine has the potential to produce a serious side effect called **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements that may appear late in therapy and not disappear even after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage of olanzapine. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period.

An occasionally reported side effect of olanzapine is neuroleptic malignant syndrome. This is a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia (fast heartbeat), and arrhythmias (irregular heartbeat).

Interactions

Any drug that causes drowsiness may lead to decreased mental alertness and impaired motor skills when taken with olanzapine. Some examples include alcohol, antidepressants such as **imipramine** (Tofranil) or **paroxetine** (Paxil), antipsychotics such as **thioridazine** (Mellaril), and some antihistamines. Because olanzapine may lower blood pressure, it may reduce blood pressure to dangerously low levels if taken with drugs that are used to treat high blood pressure. **Carbamazepine** (Tegretol), a drug commonly used to treat seizures, may decrease the effectiveness of olanzapine.

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Opioids and related disorders

Definition

Opioids are a class of drugs that include both natural and synthetic substances. The natural opioids (referred to as opiates) include opium and morphine. Heroin, the most abused opioid, is synthesized from opium. Other synthetics (only made in laboratories) and commonly prescribed for pain, such as cough suppressants, or as anti-diarrhea agents, include codeine, oxycodone (OxyContin), meperidine (Demerol), fentanyl (Sublimaze), hydromorphone (Dilaudid), **methadone**, and propoxyphene (Darvon). Heroin is usually injected, either intravenously (into a vein) or subcutaneously (under the skin), but can be smoked or used intranasally (i.e., "snorted"). Other opioids are either injected or taken orally.

The manual that is used by mental health professionals to diagnose mental disorders is the ***Diagnostic and Statistical Manual of Mental Disorders***. The latest edition of this manual was published in 2000, and is also known as the *DSM-IV-TR*. *DSM-IV-TR* lists opioid dependence and opioid abuse as substance use disorders. In addition, the opioid-induced disorders of opioid intoxication and opioid withdrawal are listed in the substance-related disorders section as well.

Opioid dependence

Opioid dependence, or **addiction**, is essentially a syndrome in which a person continues to use opioids in spite of significant problems caused by or made worse by the use of opioids. Typically individuals with opioid dependence are physically dependent on the drug as evidenced by tolerance and/or withdrawal.

Opioid abuse

Opioid abuse is less severe than opioid dependence and typically does not involve physical dependence on the drug. Opioid abuse is essentially repeated significant negative consequences of using opioids recurrently.

Opioid intoxication

When an individual uses a sufficient amount of an opioid, they will get "high" from the drug. Some people, however, have negative experiences when they use an opioid. When too much of an opioid is taken, an individual can overdose.

Opioid withdrawal

Individuals who use opioids on a regular basis, even if only for a few days, may develop a tolerance to the drug and experience physiological and psychological symptoms when they stop using the drug. The "abstinence syndrome" related to opioids is very similar to a bad case of influenza (or the "flu").

Description

Opioid dependence

Dependence on opioids involves significant physiological and psychological changes, which make it extremely difficult for an individual to stop using the opioids. Recurrent use of opioids causes actual changes in how the **brain** functions. An individual who is addicted to opioids cannot simply just stop using, despite significant negative consequences related to their use. Marital difficulties, including divorce, unemployment, and drug-related legal problems are often associated with opioid dependence. People dependent on opioids often plan their day around obtaining and using opioids.

Opioid abuse

People who abuse opioids typically use them less frequently than those who are dependent on opioids. However, despite less frequent use, an individual with opioid abuse suffers negative consequences. For example, while intoxicated on opioids, an individual may get arrested for their behavior.

Opioid intoxication

An individual who uses opioids typically experiences drowsiness ("nodding off"), mood changes, a feeling of heaviness, dry mouth, itching, and slurred speech. Individuals who use heroin intravenously describe an intense euphoria (or "rush"), a floating feeling, and total indifference to pain. Symptoms of intoxication usually last several hours. Severe intoxication from an overdose of opioids is life-threatening because breathing may stop.

Opioid withdrawal

Tolerance to opioids occurs quickly. Regular users of opioids take doses that would kill someone who has never used before. After regular use, the human body adapts to the regular presence of the drug and the person only feels "normal" when they have opioids in their system. Therefore, when an opioid-dependent individual stops using opioids abruptly, he or she will experience withdrawal symptoms. Withdrawal symptoms from heroin usually begin six to eight hours after last use and peak after two days. Acute withdrawal typically lasts no more than seven to ten days, but some symptoms of withdrawal (such as craving, **insomnia**, anxiety, lack of interest) can last six months or longer. Although withdrawal is very uncomfortable, it is not life-threatening unless there is an underlying medical condition, such as heart disease. In addition to physical withdrawal, "psychological withdrawal" often occurs. The individual who is dependent on opioids has difficulty imagining living without the drug, since they were dependent on it to function. This is similar to how someone addicted to nicotine may feel after giving up cigarettes.

KEY TERMS

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

Causes and symptoms

Causes

There are no clear-cut causes of drug use other than the initial choice to use the drug. This decision to use may be highly influenced by peer group. Typically, the age of first use of heroin is about 16 years old, but this age has been dropping in recent years.

Certain social and behavioral characteristics, however, are more commonly seen among individuals who become dependent on opioids than those who do not. For instance, many heroin users come from families in which one or more family members use alcohol or drugs excessively or have mental disorders (such as **antisocial personality disorder**). Often heroin users have had health problems early in life, behavioral problems beginning in childhood, low self-confidence, and anti-authoritarian views.

Among opioid-dependent adolescents, a "heroin behavior syndrome" has sometimes been described. This syndrome consists of depression (often with anxiety symptoms), impulsiveness, fear of failure, low self-esteem, low frustration tolerance, limited coping skills, and relationships based primarily on mutual drug use.

Symptoms

OPIOID DEPENDENCE. The *DSM-IV-TR* specifies that three or more of the following symptoms must occur at any time during a 12-month period (and cause significant impairment or distress) in order to meet diagnostic criteria for opioid dependence:

- Tolerance: The individual either has to use increasingly higher amounts of the drug over time in order to achieve the same drug effect or finds that the same amount of the drug has much less of an effect over time than before.
- Withdrawal: The individual either experiences the characteristic abstinence syndrome (i.e., opioid-specific withdrawal) or the individual uses opioids or similar-acting drugs in order to avoid or relieve withdrawal symptoms.
- Loss of control: The individual either repeatedly uses more opioids than planned or uses the opioids over longer periods of time than planned.
- Inability to stop using: The individual has either unsuccessfully attempted to cut down or stop using the opioids or has a persistent desire to stop using.
- Time: The individual spends a lot of time obtaining opioids, getting money to buy opioids, using opioids, being under the influence of opioids, and recovering from the effects of opioids.
- Interference with activities: The individual either gives up or reduces the amount of time involved in recreational activities, social activities, and/or occupational activities.
- Harm to self: The individual continues to use opioids despite having either a physical or psychological problem (depression, for example) that is caused or made worse by the opioid use.

OPIOID ABUSE. The *DSM-IV-TR* specifies that one or more of the following symptoms must occur at any time during a 12-month period (and cause significant impairment or distress) in order to meet diagnostic criteria for opioid abuse:

- Interference with role fulfillment: The individual's use of opioids repeatedly interferes with the ability to fulfill obligations at work, home, or school.
- Danger to self: The individual repeatedly uses opioids in situations in which it may be physically hazardous (while driving a car, for example).
- Legal problems: The individual has recurrent opioid-related legal problems (such as arrests for possession of narcotics).
- Social problems: The individual continues to use opioids despite repeated interpersonal or relationship problems caused by or made worse by the use of opioids.

OPIOID INTOXICATION. The *DSM-IV-TR* specifies that the following symptoms must be present in order to meet diagnostic criteria for opioid intoxication:

- Use: The individual recently used an opioid.
- Changes: The individual experiences significant behavioral or psychological changes during, or shortly after, use of an opioid. These changes may include euphoria initially, followed by slowed movements or agitation, impaired judgment, **apathy** ("don't care attitude"), dysphoric mood (depression, for example), or impaired functioning socially or at work.
- Opioid-specific intoxication syndrome: The pupils in the eyes get smaller. In addition, drowsiness or coma, slurred speech, and/or impaired memory or attention during, or shortly after, opioid use occur.

OPIOID WITHDRAWAL. The *DSM-IV-TR* specifies that the following symptoms must be present in order to meet diagnostic criteria for opioid withdrawal:

- Abstinence: Either the individual has stopped using (or has reduced the amount of) opioids, or an opioid antagonist (i.e., a drug, such as naloxone, that blocks the action of opioids) has been administered.
- Opioid-specific withdrawal syndrome: Three or more symptoms develop after abstinence. These symptoms include dysphoric (negative) mood, nausea or vomiting, muscle aches, runny nose or watery eyes, dilated pupils, goosebumps, or sweating, diarrhea, yawning, fever, and insomnia.
- Impairment or distress: The withdrawal symptoms must cause significant distress to the individual or impairment in functioning (socially, at work, or any other important area).
- Not due to other disorder: The withdrawal symptoms cannot be due to a medical condition or other mental disorder.

Demographics

There are at least 600,000 individuals with opioid dependence living in the United States. It has been estimated that almost 1% of the population has met criteria for opioid dependence or abuse at some time in their lives.

In the late 1800s and early 1900s, individuals who were dependent on opioids were primarily white and from middle socioeconomic groups. However, since the 1920s, minorities and those from lower socioeconomic

groups have been overrepresented among those with opioid dependence. It appears that availability of opioids and subcultural factors are key in opioid use. Therefore, medical professionals (who have access to opioids) are at higher risk for developing opioid-related disorders.

Males are more commonly affected by opioid disorders than females—males are three to four times more likely to be dependent on opioids than females. Age also is a factor in opioid dependence. There is a tendency for rates of dependence to decrease beginning at 40 years of age. Problems associated with opioid use are usually first seen in the teens and 20s.

Diagnosis

Diagnosis of opioid-related disorders are based on patient interview and observations of symptoms, including signs of withdrawal such as dilated pupils, watery eyes, frequent yawning, and anxiety, among others.

Opioid dependence

Other mental disorders are common among individuals with opioid dependence. It has been estimated that 90% of those with opioid dependence have one or more other mental disorders. Depression (usually either major depression or substance-induced mood disorder) is the most common disorder. Opioid-dependent individuals frequently report suicidal ideation (thoughts) and insomnia. Other substance use disorders (such as alcoholism), anxiety disorders, antisocial personality disorder, **posttraumatic stress disorder**, and a history of **conduct disorder** are also fairly common.

Opioid intoxication

Intoxication on other substances, such as alcohol, sedatives, hypnotics, and anxiolytics, can resemble intoxication on opioids. Furthermore, dilated pupils can be seen in hallucinogen intoxication, amphetamine intoxication, and cocaine intoxication.

Opioid withdrawal

The restlessness and anxiety seen in opioid withdrawal is also seen in withdrawal from sedatives, hypnotics, and anxiolytics.

Treatments

Opioid dependence

Because opioid-related disorders are complex, multiple treatment approaches are often necessary. Generally, the more treatment (a combination of medication, individual therapy, and **self-help groups**, for example) and longer the treatment (i.e., at least three months), the better the outcomes. There are a wide variety of treatment options, both inpatient or residential and outpatient:

- Methadone maintenance treatment. Methadone is a long-acting opioid that is generally administered in an outpatient setting (a methadone maintenance clinic). The methadone prevents the individual from experiencing opioid withdrawal, reduces opioid craving, and enables the individual to have access to other services (such as individual counseling, medical services, and HIV-prevention education). A proper dose of methadone also prevents the individual from getting "high" from heroin. Methadone maintenance therapy can decrease criminal activity, decrease HIV-risk behaviors, and increase stability of employment. Low-dose methadone maintenance treatment is preferable for pregnant individuals who would otherwise use illicit opioids. A longer-acting alternative to methadone is LAAM (levo-alphacetylmethadol). Individuals receiving the proper doses of LAAM only need to take it three times per week, instead of every day as with methadone.
- Opioid antagonist treatment. An opioid antagonist is a medication that blocks the effects of opioids. Treatment with an antagonist, usually **naltrexone** (Trexan), typically takes place on an outpatient basis following an inpatient medical **detoxification** from opioids. The effects of taking any opioids are blocked by the naltrexone and prevent the individual from getting "high," thereby discouraging individuals from seeking opioids. By itself, this treatment is suitable for individuals highly motivated to discontinue opioid use. However, antagonists can be used in addition to other treatment modalities or with individuals who have been abstinent for some time but fear a relapse.
- Opioid agonist-antagonist treatment. An opioid agonist is a drug that has a similar action to morphine. Buprenorphine (Buprenex) is an example of an opioid agonist-antagonist, which means it acts as both an agonist (having some morphine-like action) and antagonist (it blocks the effects of additional opioids). Buprenorphine has been shown to effectively reduce opioid use. It is also being studied for opioid detoxification.
- Outpatient drug-free treatment. These are outpatient treatment approaches that do not include medications. There are a number of different types of programs ranging from simple drug education to intensive outpatient programs that offer most of the services of an inpatient setting. Some programs may specialize in treating specific groups of people who are opioid-dependent (those with co-occurring mental disorders, for example).

- Residential or inpatient treatment. These include inpatient rehabilitation programs (usually seven to 30 days in length) and long-term residential programs (such as therapeutic communities). Rehabilitation programs provide an inpatient atmosphere following detoxification and usually offer individual and group counseling as well as medical services. Therapeutic communities are designed to be more than six months long and are highly structured. The primary focus is on resocializing the individual to a drug-free and crime-free lifestyle.
- Individualized drug counseling. Individual counseling is often a part of a methadone maintenance program or inpatient rehabilitation program. The primary focus is on helping the individual learn strategies to reduce or stop their opioid use and learn coping mechanisms to maintain abstinence. Twelve-step participation is encouraged and referrals for medical, psychiatric, employment, or other services are made as necessary.
- Supportive-expressive **psychotherapy**. This type of individual psychotherapy may be a part of a methadone maintenance program or offered alone. The focus of this type of therapy is to help individuals feel comfortable talking about themselves, work on relationship issues, and solve problems without resorting to opioids or other drugs.
- **Self-help groups.** Narcotics Anonymous (NA) is a twelve-step group based on the same model as Alcoholics Anonymous. This self-help group can provide social support to an individual in the process of reducing or stopping opioid use. Participation in NA is often encouraged or is a required component of other types of treatment for opioid dependence. Nar-Anon is a group for family members and friends of opioid-dependent individuals.
- Alternative therapies. Hypnosis, guided imagery, **biofeedback**, massage, and **acupuncture** have all been studied as adjunctive treatments for opioid dependence, but none have been proven to be effective.

Opioid abuse

Most of the treatments for opioid dependence would be appropriate for opioid abuse except methadone maintenance and opioid antagonist treatment.

Opioid intoxication

An opioid antagonist, naloxone (Narcan), can be administered to reverse the effects of acute intoxication or overdose on most opioids.

Opioid withdrawal

Opioid withdrawal can be treated either on an inpatient basis (detoxification) or on an outpatient basis (methadone detoxification):

- Inpatient detoxification program. Typically, this would be from three to seven days. The withdrawal can be medically managed. **Clonidine** may be administered to help reduce some symptoms of withdrawal.
- Outpatient methadone detoxification. Methadone would be substituted for the illicit opioid and the dose would be gradually reduced. Detoxification from methadone is easier (i.e., the symptoms are less severe) than from heroin. However, the withdrawal or abstinence syndrome also lasts longer. Clonidine may also be administered during the methadone detoxification to help reduce withdrawal symptoms.

Prognosis

Opioid dependence

Recovering from opioid dependence is a long, difficult process. Typically, multiple treatment attempts are required. Relapsing, or returning to opioids, is not uncommon even after many years of abstinence. Brief periods of abstinence are common.

Inpatient detoxification from opioids alone, without additional treatment, does not appear to have any effect on opioid use. However, other treatments have been shown to reduce opioid use, decrease illegal activity, decrease rates of HIV-infection, reduce rates of death, and increase rates of employment. Benefits are greatest for those who remain in treatment longer and participate in many different types of treatment (individual and group counseling in addition to methadone maintenance, for example).

Opioid abuse

Very little is known about the course of opioid abuse.

Prevention

The best single thing an individual can do to prevent opioid-related disorders is never to use illicit opioids such as heroin. Opioids are powerfully addicting, especially if used intravenously. The risk of becoming dependent on appropriately prescribed opioids, however, is generally low except for individuals who already have a substance use disorder.

On a larger scale, comprehensive prevention programs that utilize family, schools, communities, and the media can be effective in reducing substance abuse. The

recurring theme in these programs is not to use drugs in the first place.

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American Psychiatric Association. 1400 K Street, Washington, DC 20005. (202) 682-6000. <http://www.psych.org>.

American Psychological Association. 750 First Street, NE, Washington, DC 20002-4242. (800) 374-2721. <http://www.apa.org>.

National Institute of Mental Health, 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

National Institute on Drug Abuse. 5600 Fishers Lane, Room 10-05, Rockville, MD 20857. Nationwide Helpline: (800) 662-HELP. <http://www.nida.nih.gov>.

National Library of Medicine. 8600 Rockville Pike, Bethesda, MD 20894. <http://www.nlm.nih.gov/medlineplus/drugabuse.html>.

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KEY TERMS

Attention-deficit/hyperactivity disorder—A learning and behavioral disorder characterized by difficulty in sustaining attention, impulsive behavior, and excessive activity.

Behavioral therapy—An approach to treatment that focuses on extinguishing undesirable behavior and replacing it with desired behavior.

Cognitive therapy—Psychological treatment aimed at changing a person's way of thinking in order to change his or her behavior and emotional state.

Conduct disorder—A behavioral and emotional disorder of childhood and adolescence in which children display physical aggression and infringe on or violate the rights of others. Youths diagnosed with conduct disorder may set fires, exhibit cruelty toward animals or other children, sexually assault others, or lie and steal for personal gain.

Oppositional defiant disorder—An emotional and behavioral problem of children and adolescents characterized by defiant, hostile, or disobedient behavior that has lasted for longer than six months.

Passive-aggressive behaviors—Behaviors that represent covert expressions of hostile or negative feelings that the person is unable or unwilling to express directly.

Oppositional defiant disorder

Definition

Oppositional defiant disorder (ODD) is a disorder found primarily in children and adolescents. It is characterized by negative, disobedient, or defiant behavior that is worse than the normal "testing" behavior most children display from time to time. Most children go through periods of being difficult, particularly during the period from 18 months to three years, and later during adolescence. These difficult periods are part of the normal developmental process of gaining a stronger sense of individuality and separating from parents. ODD, however, is defiant behavior that lasts longer and is more severe than normal individuation behavior, but is not so extreme that it involves violation of social rules or the rights of others.

The mental health professional's handbook, ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (*DSM-IV-TR*), classifies ODD as a disruptive behavior disorder.

Description

Children who have ODD are often disobedient. They are easily angered and may seem to be angry much of the time. Very young children with the disorder will throw temper tantrums that last for 30 minutes or longer, over seemingly trivial matters.

In addition, the child with ODD often starts arguments and will not give up. Winning the argument seems to be very important to a child with this disorder. Even if the youth knows that he or she will lose a privilege or otherwise be punished for continuing the tantrum or

argument, he or she is unable to stop. Attempting to reason with such a child often backfires because the child perceives rational discussion as a continuation of the argument.

Most children with ODD, however, do not perceive themselves as being argumentative or difficult. It is usual for such children to blame all their problems on others. Such children can also be perfectionists and have a strong sense of justice regarding violations of what they consider correct behavior. They are impatient and intolerant of others. They are more likely to argue verbally with other children than to get into physical fights.

Older children or adolescents with ODD may try to provoke others by being deliberately annoying or critical. For example, a teenager may criticize an adult's way or speaking or dressing. This oppositional behavior is usually directed at an authority figure such as a parent, coach, or teacher. Youths diagnosed with ODD, however, can also be bullies who use their language skills to taunt and **abuse** other children.

Causes and symptoms

Causes

ODD has been called a problem of families, not of individuals. It occurs in families in which some or all of the following factors are present:

- Limits set by parents are too harsh or too lax, or an inconsistent mix of both.
- Family life lacks clear structure; rules, limits, and discipline are uncertain or inconsistently applied.
- At least one parent models oppositional behavior in his or her own interactions with others. For example, mother or father may get into frequent disputes with neighbors, store clerks, other family members, etc., in front of the child.
- At least one parent is emotionally or physically unavailable to the child due to emotional problems of the parent (such as depression), separation or divorce, or work hours.

The defiant behavior may be an attempt by the child to feel safe or gain control. It may also represent an attempt to get attention from an unresponsive parent.

There may be a genetic factor involved in ODD; the disorder often seems to run in families. This pattern may, however, reflect behavior learned from previous generations rather than the effects of a gene or genes for the disorder.

Symptoms

According to *DSM-IV-TR*, a **diagnosis** of ODD may be given to children who meet the following criteria, provided that the behavior occurs more frequently than usual compared to children of the same age and developmental level.

A pattern of negativistic, hostile, and defiant behavior lasting at least six months, during which four (or more) of the following are present. The child:

- often loses his or her temper
- frequently argues with adults
- often disregards adults' requests or rules
- deliberately tries to provoke people
- frequently blames others for his or her mistakes or misbehavior
- is often easily irritated by others
- is often angry and resentful
- is often spiteful

In order to make the diagnosis of oppositional defiant disorder, the behavioral disturbances must cause significant impairment in the child's social, academic or occupational functioning, and the behaviors must not occur exclusively during the course of a psychotic or mood disorder. In addition, the child must not meet criteria for **conduct disorder**, which is a more serious behavioral disorder. If the youth is 18 years or older, he or she must not meet criteria for **antisocial personality disorder**.

Demographics

Oppositional defiant disorder is thought to occur in about 6% of all children in the United States. It is more common in families of lower socioeconomic status. In one study, 8% of children from low-income families were diagnosed with ODD. The disorder is often apparent by the time a child is about six years old. Boys tend to be diagnosed with this disorder more often than girls in the preteen years, but it is equally common in males and females by adolescence.

It is estimated that about one-third of children who have **attention-deficit/hyperactivity disorder** (ADHD) also have ODD. Children who have ODD are also often diagnosed with anxiety or depression.

Diagnosis

Oppositional defiant disorder is diagnosed when the child's difficult behavior lasts longer than six months. There is no standard test for diagnosing ODD. A full

medical checkup may be done to make sure that there is no medical problem causing the child's behavior. The medical examination is followed by a psychological evaluation of the child, which involves an interview with a mental health professional. The mental health professional may also interview the child's parents and teachers. Psychological tests are sometimes given to the child to rule out other disorders.

Evaluation for ODD includes ruling out a more disruptive behavioral disorder known as conduct disorder (CD). CD is similar to ODD but also includes physical aggression toward others, such as fighting or deliberately trying to hurt another person. Children with CD also frequently break laws or violate the rights of others, for example by stealing. They tend to be more covert than children with ODD, lying and keeping some of their unacceptable behavior secret.

The diagnosis of ODD may specify its degree of severity as mild, moderate, or severe.

Treatments

Treatment of ODD focuses on both the child and on the parents. The goals of treatment include helping the child to feel protected and safe and to teach him or her appropriate behavior. Parents may need to learn how to set appropriate limits with a child and how to deal with a child who acts out. They may also need to learn how to teach and reinforce desired behavior.

Parents may also need help with problems that may be distancing them from the child. Such problems can include alcoholism or drug dependency, depression, or financial difficulties. In some cases, legal or economic assistance may be necessary. For example, a single mother may need legal help to obtain child support from the child's father so that she won't need to work two jobs, and can stay at home in the evenings with the child.

Behavioral therapy is usually effective in treating ODD. Behavioral therapy focuses on changing specific behaviors, not on analyzing the history of the behaviors or the very early years of the child's life. The theory behind behavioral therapy is that a person can learn a different set of behaviors to replace those that are causing problems. As the person obtains better results from the new behavior, he or she will want to continue that behavior instead of reverting to the old one. To give an example, the child's parents may be asked to identify behaviors that usually start an argument. They are then shown ways to stop or change those behaviors in order to prevent arguments.

Contingency management techniques may be included in behavioral therapy. The child and the parents may be helped to draw up contracts that identify unwanted behaviors and spell out consequences. For example, the child may lose a privilege or part of his or her allowance every time he or she throws a temper tantrum. These contracts can include steps or stages—for example, lowering the punishment if the child begins an argument but manages to stop arguing within a set period of time. The same contract may also specify rewards for desired behavior. For example, if the child has gone for a full week without acting out, he or she may get to choose which movie the family sees that weekend. These contracts may be shared with the child's teachers.

The parents are encouraged to acknowledge good or nonproblematic behavior as much as possible. Attention or praise from the parent when the child is behaving well can reinforce his or her sense that the parent is aware of the child even when he or she is not acting out.

Cognitive therapy may be helpful for older children, adolescents, and parents. In cognitive therapy, the person is guided to greater awareness of problematic thoughts and feelings in certain situations. The therapist can then suggest a way of thinking about the problem that would lead to behaviors that are more likely to bring the person what they want or need. For example, a girl may be helped to see that much of her anger derives from feeling that no one cares about her, but that her angry behavior is the source of her problem because it pushes people away.

Although **psychotherapy** is the cornerstone of treatment for ODD, medicine may also be helpful in some cases. Children who have concurrent ADHD may need medical treatment to control their impulsivity and extend their attention span. Children who are anxious or depressed may also be helped by appropriate medications.

Prognosis

Treatment for ODD is usually a long-term commitment. It may take a year or more of treatment to see noticeable improvement. It is important for families to continue with treatment even if immediate results are not apparent.

If ODD is not treated or if treatment is abandoned, the child has a higher likelihood of developing conduct disorder. The risk of developing conduct disorder is lower in children who are only mildly defiant. It is higher in children who are more defiant and in children who also have ADHD. In adults, conduct disorder is called antisocial personality disorder, or ASD.

Children who have untreated ODD are also at risk for developing passive-aggressive behaviors as adults. Persons with passive-aggressive characteristics tend to

see themselves as victims and blame others for their problems.

Prevention

Prevention of ODD begins with good parenting. If at all possible, families and the caregivers they encounter should be on the lookout for any problem that may prevent parents from giving children the structure and attention they need.

Early identification of ODD and ADHD is necessary to obtain help for the child and family as soon as possible. The earlier ODD is identified and treated, the more likely it is that the child will be able to develop healthy patterns of relating to others.

Resources

BOOKS

Hales, R. E., S. C. Yudofsky, J. A. Talbott, eds. *Textbook of Psychiatry.* 3rd ed. Washington DC: American Psychiatric Press, 1999.

Sadock, B. J., and V. A. Sadock. *Kaplan & Sadock's Comprehensive Textbook of Psychology,* 7th ed. Philadelphia: Lippincott Williams and Wilkins, 1999.

PERIODICALS

Loeber, Rolf. "Oppositional defiant and conduct disorder: a review of the past 10 years, part I." *Journal of the American Academy of Child and Adolescent Psychiatry* Dec. 2000.

ORGANIZATIONS

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Orap *see* **Pimozide**

Origin of mental illnesses

History of theories about mental illness

Mental illness in the ancient world

Over the history of the healing arts, there has been an evolution of theories regarding the root causes of mental illness. Early writings from such ancient civilizations as those of Greece, Rome, India, and Egypt focused on demonic possession as the cause. This concept eventually disappeared only to resurface again in the Middle Ages in Europe, along with inadequate treatment of the mentally ill. Demons or "foul spirits" were believed to attach themselves to individuals and make them depressed ("poor-spirited") or "mad." The word *mad* became an early synonym for **psychosis**. Unfortunately, the "possessed" included people with seizure disorders as well as others suffering from what are now known to be medical disorders. Few genuinely helpful treatments were available to relieve the suffering of the mentally ill.

The Hippocratic tradition

Hippocrates, a Greek physician who lived around 400 B.C. and is regarded as the source of the Hippocratic Oath taken by modern physicians, first introduced the concept of disturbed physiology (organic processes or functions) as the basis for all illnesses, mental or otherwise. Hippocrates did not describe disturbances of the nervous system as we do today, in terms of a chemical imbalance or a low level of **neurotransmitters** (neurotransmitters are the chemical messengers sent between **brain** cells). Instead, he used the notion of an imbalance of "humors." Humors were defined as bodily fluids, and were believed to be influenced by the environment, the weather, foods, and so on, producing various imbalances in a person's state of health. Hippocrates' theory was an early version of the idea that physiological disturbances or body chemistry might play a role in the development of mental illness. Most importantly, perhaps, Hippocrates' concept placed mental illness on the same footing as other medical disorders by highlighting the belief that the mentally ill are genuinely suffering, and therefore to be treated like other sick persons rather than as moral degenerates. Sadly, modern society has not fully overcome the tendency to stigmatize persons with mental disorders. Hippocrates' more "enlightened" perspective, however, meant that someone with depression or **schizophrenia** could be viewed as being in a state of "dis-ease," just like a diabetic or someone with high blood pressure.

The nineteenth century

Toward the end of the nineteenth century, several European neurologists began actively investigating the causes of mental illness. Chief among them, and destined to change forever the understanding of mental illness, was Sigmund Freud. Although psychology and psychiatry have advanced considerably since Freud (as have other fields of medicine), his explorations were revolutionary. Freud introduced the concepts of the unconscious and the ego to modern thought, and reintroduced the ancient art of dream interpretation, but from a psychological standpoint. Freud also regarded human psychological states as an energy system in which blockages in the flow of thought (repression or suppression, for

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness.

Delirium tremens—Serious alcohol withdrawal symptoms that must be treated in a hospital and that may include shaking, delirium, and hallucinations.

Delusion—A false belief that is resistant to reason or contrary to actual fact.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Electrolytes—Substances or elements that dissociate into electrically charged particles (ions) when dissolved in the blood. The electrolytes in human blood include potassium, magnesium, and chloride.

Etiology—The cause or origin of a disease or disorder. The word is also used to refer to the study of the causes of disease.

Flashback—The re-emergence of a traumatic memory as a vivid recollection of sounds, images, and sensations associated with the trauma. The person having the flashback typically feels as if he or she is reliving the event.

Fugue state—A form of amnesia in which the person appears to be conscious and to make rational decisions, but upon recovery, the period is not remembered. Fugue states represent one type of reaction to traumatic experiences.

Hallucination—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Humor—In ancient medicine, one of four body fluids (blood, phlegm, yellow bile, and black bile) that were thought to determine a person's basic constitution and personality.

Insult—In medicine, an injury or trauma to the brain or other part of the body.

Metabolism—The group of biochemical processes within the body that release energy in support of life.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Physiology—The branch of medicine concerned with biological processes or functions in the human body or any of its parts.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Thyrotoxicosis—A disease characterized by an enlarged thyroid gland and speeded-up body metabolism caused by excessive thyroid secretion. It is also known as Graves' disease.

Tryptophan—An essential amino acid released from proteins during the process of digestion. Tryptophan is an important ingredient in the body's production of serotonin.

example) would result in disease or illness, expressed as mental or emotional loss of balance. He introduced the notion of a "talking cure"; through the use of **talk therapy** alone, many patients would improve. This method of treatment is still used today, although the technique of talk therapy itself has undergone further development. Freud's early advances in understanding the mind, however, awaited further anatomical and biochemical discoveries of the structures and functions of the human brain. As a result, early psychiatry (from two Greek words, *psyche*, meaning "soul" or "mind," and *iatros*, meaning "physician") split into two competing traditions, one that followed Freud in emphasizing thoughts, emotions and dreams as keys to the healing of mental disorders, and another that looked for clues to these disorders in the tissues of the brain.

In the first half of the twentieth century, psychiatry was advanced by the discovery of medications that helped to alleviate depression, mania, and psychosis. As often occurs in the history of medicine, physicians stumbled upon solutions before they understood the mechanisms that made the treatment work. Later studies began to reveal that certain patients responded to medications that increased certain neurotransmitters. Drugs that increased the levels of the neurotransmitters norepinephrine and serotonin seemed to help depressed patients. Similarly, medications that blocked the transmission of dopamine, another neurotransmitter, provided relief for patients suffering from **hallucinations** and **paranoia**. These insights have led to the present emphasis on the biochemistry of the human brain. If, however, the biochemical model becomes the only view of mental health, modern psychiatry risks becoming "mindless." Clearly, a unified theory is needed to understand all the factors that contribute to mental disorders, and to do justice to the complexity of each human being. Understanding all the factors that lead to a disease state has much to do with an adequate treatment response.

Nature and nurture

One attempt to unify the varied theories regarding the origin of mental illness is called simply the "nature versus nurture" theory. It is really the "nature *and* nurture" theory, however, as it establishes the importance of two forces in the development of mental illness. For example, "nature" refers to biological factors that produce a tendency or predisposition to develop certain diseases. For instance, parents who have high blood pressure have offspring who have a higher probability of developing the same condition. If, on the other hand, these offspring learn to eat properly, exercise, and live in a relatively peaceful home, for instance, they may be able to avoid the expression of high blood pressure that runs in their family. This example illustrates the impact that a person's environment may have on the development of physical disease. Researchers believe the same holds true for mental illnesses. For example, researchers know that patients with schizophrenia who return to a family environment in which there is a high level of expressed emotion, such as critical and angry remarks, have more frequent psychotic episodes that require **hospitalization**. Thus, it appears that the interaction between the biological and psychological dimensions of a person and his or her environment determines the likelihood of expressing a mental illness, or perhaps any illness whatsoever. There is, however, no accurate prediction or test that will determine whether or not a specific person will develop a certain mental illness, even if many members of his or her are positive for that disease.

Conversely, a child with minimal genetic predisposition to mental illness may develop mental illness if he or she is traumatized in any number of ways, such as being raised in a non-nurturing or a physically, mentally, or emotionally abusive household. As of 2002, scientists do not know why some people become mentally ill while others do not. Much research remains to be done; although theories abound, the precise etiology or origin of all mental illnesses remains uncertain.

Current theories about the origin of mental disorders

Biological theories

GENETICS. Genetics is at this time an important area of research for psychiatric disorders. For example, a specific gene has been associated with **bipolar disorder** (also known as manic-depressive disorder), but unfortunately, the ˚switch" that controls the expression of the disorder is still unknown. It is presently thought that many genes go into the expression or nonexpression of any human characteristic, such as a facial feature or a certain aspect of mental health. Research done on identical twins has provided strong support for a genetic component in the development of schizophrenia. For instance, the average person in the United States has a 1% chance of developing schizophrenia, while the identical twin of a person diagnosed with schizophrenia has a 50% chance, even if he or she has been reared by adoptive parents. Other researchers who are studying schizophrenia have found that during embryonic development, there are nerve cells that do not migrate to their proper position in the brain. On the other hand, none of the genetic or embryological findings can account for the rare but occasional recoveries from schizophrenia, indicating that biology alone does not determine the occurrence of mental disorders.

Dementias are also noted to run in families, but most of these disorders cannot be predicted with any certainty for the following generation. Only one disorder, Huntington's chorea, which is really a movement disorder with a psychiatric component, appears to be determined by a single gene. **Dementia** of the Alzheimer's type does seem to have familial pattern, but again, the expression of the disease in any specific individual is not predictable at this time. Scientists believe that similar statements can be made for many mental disorders that run in families, such as **obsessive-compulsive disorder** (OCD), depression, anxiety, and **panic disorder**. The roles of the environment and learning behavior in the ultimate expression of genetically predisposed individuals are, however, undisputed.

NEUROTRANSMITTER-RELATED CHEMICAL IMBALANCES. This theory regarding the origin of mental disorders has become the foundation of most psychiatric treatment today. It has legitimized psychiatry by returning it to the world of biological medicine. Diabetes may offer a helpful analogy. In diabetes, a chemical necessary to health (insulin) is missing and can be replaced, essentially restoring the patient's health. In mental illness, the neurotransmitters in the brain may be present in insufficient amounts. These chemicals or transmitters allow communication between nerve cells; as a result, they coordinate information processing throughout the brain. As a person reads, for example, chemical levels rise and fall in response to the letters; the meaning they have; the reader's eye movements, thoughts, reflections and associations; and to the feelings the reader may have while reading. Thus, a person's brain chemistry is changed by everything that influences him or her, whether internally or externally. While the discovery of certain neurotransmitters and their roles in mental disorders has led in turn to the discovery of effective medications to treat these disorders, it has also resulted in the unfortunate notion that medication is the only method of treatment that is helpful.

Major neurotransmitters identified thus far include acetylcholine, dopamine, epinephrine, norepinephrine, histamine, and serotonin. Serotonin and norepinephrine are most highly implicated in depression, panic disorder and anxiety, as well as OCD. Most of the medications found effective for these disorders are drugs that increase the availability of serotonin and norepinephrine (such as selective serotonin re-uptake inhibitors, or SSRIs). In particular, depression, panic disorder, anxiety disorders, and OCD have responded strongly to medications that increase serotonin levels. On the other hand, medications that block the effects of dopamine in certain parts of the brain are effective in controlling auditory and visual hallucinations as well as paranoia in patients with psychotic disorders.

STRESS-RELATED FACTORS. **Stress** is something everyone in modern society seems to understand. There are two basic kinds of stress: inner stress from previous traumas or wounds that affect one's present life; and outer stress, or the environmental issues that complicate life on a daily basis, such as work or family problems. The interplay of these two forms of stress affects brain chemistry just as it can affect physical health. Numerous studies have shown that when people are chronically stressed in life, they are vulnerable to depression, anxiety, and other disorders. Interestingly, 70% of the adults in one recent European war situation were found to have depression, which is a normal human response to relentless stress. Researchers presently think that the mechanism that triggers this depression is the depletion of certain neurotransmitters, particularly serotonin and norepinephrine, which may lead to other biochemical imbalances. For instance, most people diagnosed with schizophrenia have their first psychotic episode during such stressful situations as leaving home for college or military service.

Genetic factors may add to a person's susceptibility to mental illness by lowering the body's production of neurotransmitters during difficult life transitions. The same combination of circumstances might affect the development of high blood pressure, diabetes, or ulcers in some families.

MEDICAL CONDITIONS. It is important to note that bacterial and viral infections, metabolic illnesses, medications and street drugs can all affect a person's mental status. Insults (injuries) to the brain can cause a person to be disoriented, speak incoherently, have difficulty concentrating, hallucinate, or even act out violently. When clinicians see disorientation and an abrupt change in a person's level of alertness, they refer to the altered mental state as **delirium**. Delirium is considered a medical emergency because the underlying cause must be identified and treated as quickly as possible. The exact way in which infectious disease and chemical agents change human mental function is unclear, and thus may not be visible on **imaging studies**.

The elderly are particularly vulnerable to changes in mental status resulting from apparently minor changes in body chemistry. Fever, dehydration, electrolyte imbalances, and even aspirin or antibiotics can all have an abrupt effect on the mental status of the elderly. Older people are susceptible simply because older brain tissue is more sensitive to the slightest change in metabolism or the presence of toxins.

Certain diseases have severe effects on the brain. An example is HIV/AIDS, in which approximately 70% of patients suffering from full-blown AIDS develop demen-

tia, depression, or delirium. Similarly, at least 50% of patients with multiple sclerosis develop depression from the effects of the disease on brain tissues—not simply as a reaction to knowing that they have MS. Any infectious disease that causes inflammation inside the skull, such as meningitis or encephalitis, will usually result in some change in mental status; fortunately, these changes are usually completely reversible.

Recently, there has been an exciting development involving infectious disease and OCD as exemplified by "PANDAS," the acronym for Pediatric Autoimmune Neuropsychiatric Disorder Associated with Group A Streptococcus. Group A Streptococcus is an autoimmune disorder thought to cause OCD symptoms (neuropsychiatric symptoms) in children with streptococcal infection of the tonsils and pharynx (more commonly known as strep throat). The OCD symptoms resolve when the infection is treated with antibiotics. The neuropsychiatric symptoms are believed to result from an autoimmune reaction, meaning that antibodies made to fight the bacteria mistakenly attack part of the brain, resulting in symptoms of OCD. The discovery of this connection between a streptococcal infection and an autoimmune reaction may have great importance for treating certain mental illnesses in the future, since links between the onset of psychiatric disorders and physical infections have been observed from time to time.

Disorders of metabolism can certainly mimic depression, anxiety and sometimes, even psychosis. Overproduction of thyroid hormone (thyrotoxicosis) can cause agitation, anxiety, mania and even psychosis; while a lack of thyroid hormone produces symptoms of depression and is routinely checked in patients with depression of recent onset. Imbalances in glucose (sugar) management can result in mood swings and should always be evaluated. Less commonly, malfunctions of the adrenal glands can profoundly affect a person's energy level and mental activity. The role of estrogen in postmenopausal depression has been intensively studied in recent years, but the findings remain inconclusive.

NEUROPATHOLOGY. Neuropathology refers to damage to the brain tissue itself that results in mental illness. Dementias are placed in this category, since the brains of persons diagnosed with dementia exhibit microscopic changes in tissue structure when viewed under a microscope. These changes may ultimately appear on tests such as a CAT scan of the brain. Larger changes are seen with strokes, which result when the blood supply is cut off to a specific area of the brain and causes localized damage. In these instances, a person may have altered speech patterns but retain the ability to think clearly, or vice versa. The losses are somewhat predictable and specific, based on the area of the brain that was affected and the extent of oxygen starvation of the tissue in that region.

Brain tumors and accidental injuries are random in their effects, and the deficits are usually less predictable. Each case must be examined individually. As with strokes, however, the location of the injury or tumor will determine the resulting mental status changes or deficits.

Pancreatic and certain colon cancers are particularly interesting for psychiatrists. For reasons that are unknown as of 2002, these tumors are frequently accompanied by depression even though they are located in organs that are far removed from the brain. More research is needed on the relationship between mood disorders and certain illnesses; it is possible that the tumor releases compounds into the bloodstream that have depressive effects.

NUTRITIONAL FACTORS. There is no doubt that poor nutrition leads to mental imbalances. While few people in the United States are truly starving or completely depleted nutritionally, instances of mental disorders related to malnutrition still occur in this country. The B vitamins are essential for mental clarity and stability. Insufficient amounts of the B vitamins, which include thiamin, nicotinamide, pyridoxine, and B, can result in confusion, irritability, **insomnia**, depression, and in extreme cases, psychosis. The body does not store these vitamins, so one should monitor one's daily intake to ensure a sufficient supply. Tryptophan is an amino acid and supplement that is a building block for serotonin, the neurotransmitter that has been found to be essential in treating depression, anxiety, panic, and OCD, among others. Tryptophan is so important nutritionally that studies have shown that its absence in the diet will result in depression even when the person is taking a prescription antidepressant to increase the availability of serotonin.

Psychological/interpersonal theories

PSYCHODYNAMIC THEORIES. Freud certainly opened the doors for humans to understand themselves in terms of psychology, or the notion that how one thinks and feels affects one's view of the world. Freud also found that simple conversation could help some very sick people out of depressions and other mental disorders. His work essentially demonstrated that extreme inner conflicts can become a source of mental illness. These extreme internal conflicts can occur, for instance, when one loves another deeply but also feels that that person is hurting them or limiting their development in some way. If the person who is causing pain or hindering growth is a parent or other powerful figure, these intense feelings can be hidden away or repressed. Also, a lack of honesty about reality can lead to any number of illnesses. For

instance, feelings of anger and powerlessness, if unrecognized, may place the person at risk for developing aggressive behaviors or depression if insights and appropriate coping skills are not gained. These psychological dis-harmonies, if ignored, can lead to dis-ease if they are sufficiently intense or associated with central relationships in the person's life.

Freud's view of psychological conflicts as rooted in sexual repression was questioned by Jung, a **psychiatrist** and protégé of Freud, who felt that people's lives were affected by deep spiritual forces. Jung's work centered on psychological imbalances stemming from spiritual distress. There were other theorists after Freud, such as Adler, who regarded power as the central motivating force of human personality, or Melanie Klein, who emphasized the significance of envy.

Since the Second World War, behavioral and cognitive theories have emphasized the role of learning in the development of mental disorders. Children growing up in an abusive home, for example, may be "rewarded" by not getting beaten if they learn to be quiet and internalize everything. This internalized state may be a precursor of full-blown depression in later years. Unconscious assumptions based on early experiences may spill over into other situations later in life. As another example, children may learn to be "good" for their parents or society by taking on careers they don't like or belief systems that don't fit them, all for approval by the perceived higher authority.

Cognitive approaches to therapy maintain that people construct their view of the world from beliefs and feelings based on deeper assumptions about their own competencies. Depression, for instance, would be seen as a spiral downward into negative "self-talk" and feelings of inadequacy. Re-examining these negative assumptions then breaks the cycle based on erroneous thinking (cognition) which is causing the depression, anxiety, or aberrant behavior. Studies have shown that three months of cognitive therapy is as effective as medication in the treatment of depression. This finding shows clearly that talk therapy does change the chemistry of the brain.

TRAUMA-RELATED FACTORS. Psychological traumas refer to events that are outside the experience of everyday life, although the exact definition of a traumatic experience may vary from person to person, country to country, and century to century. Traumas in early life, such as sexual or physical **abuse**, can lead to mood disorders and contribute to the development of **personality disorders**. Horrendous early traumas involving torture of a child, other people, or animals, may result in **dissociative identity disorder**, formerly called multiple personality disorder. Dissociation is a self-protective mechanism for separating conscious awareness from repeated traumas. It has sometimes been described as self-hypnosis, but most clinicians believe that it is not under the patient's control, at least initially.

In later life, such severe traumas as war, rape, natural disasters, or any similar event, can lead to psychiatric difficulties. **Post-traumatic stress disorder** (PTSD) is a well-known disorder that affects war veterans. Extreme trauma causes the brain to record impressions in a way that is different from ordinary formation of memories. These disjointed impressions may re-emerge as flashbacks months or years after the traumatic experience. Chronic and repetitive trauma, exemplified by intermittent abuse or hostage situations, can lead to a chronic form of PTSD as well.

A subcategory of psychiatric disorders that occur in response to traumatic shock are termed fugue states. Fugue states are poorly understood, but can be described as conditions of total memory loss after witnessing an overwhelmingly horrible accident or atrocity. These states of memory loss can last from minutes to years.

SOCIOCULTURAL FACTORS. Some mental disorders are influenced by social values and social interactions shaped by those values. **Anorexia nervosa**, **bulimia nervosa**, and **body dysmorphic disorder** are the most commonly used examples of mental illnesses in this category. With the increased visibility of unnaturally slender women in modern society (as seen everywhere in advertising, television shows, movies, and celebrity fan magazines) doctors have seen a tremendous rise in the occurrence eating disorders. "You can never be too thin or too rich," a saying attributed to the Duchess of Windsor, is a phrase that has many women, and some men, monitoring their every ounce of food intake. The core of the illness is a lack of self-esteem combined with feelings that one's world is out of control. Some clinicians add fear of sexual maturation to this list of psychological causes of eating disorders. The common denominator is that these patients apparently believe they can control their world by controlling their food intake. Although neurotransmitter deficits have been found in patients with bulimia, whose vomiting may actually change their body chemistry, the desire to be thin is the conscious motivating force.

Modern society also values activity over rest, doing over being, thinking over feeling, resulting in many people becoming slaves to work and productivity, and having little respect for their inner life. Many cases of mild stress-related disorders run the risk of developing into full-blown generalized anxiety, panic, and depressive disorders. Mental health requires a reasonable balance between work and activity on the one hand and periods of rest and relaxation on the other.

ALCOHOL AND SUBSTANCE ABUSE. Alcohol is a central nervous system depressant. It plays a prominent role in the development of at least depression and is often involved in other mental disorders. In addition, people who abuse alcohol are at increased risk of mental disorders related to nutritional deficiencies. A lack of thiamin, a B-vitamin, can result in permanent brain damage in the form of severe dementia even at an early age. People in withdrawal from alcohol are also at risk for delirium tremens, a serious condition that can result in cardiovascular shock and death.

Street drugs are well known for their effects on young people's mood and behavior. Permanent brain damage may result from the use of some "designer" drugs. One example is "Ecstasy," which can cause permanent memory loss and severe depression that responds only slowly to treatment. Street drugs must always be considered as a possible factor in the sudden onset of a mental illness in a young person. Moreover, drugs may precipitate a first psychotic episode in a person with a genetic predisposition to schizophrenia. In this case, the drug is the stressor that reveals the person's dormant susceptibility to the disorder.

Current theory and future directions

The biopsychosocial model of mental illness

All of the above factors are most succinctly summarized in terms of the biopsychosocial model of mental illness. Biological contributions, thoughts and perceptions, social pressures, and environmental stressors, the presence or absence of nurturing and consistency of love, core values, and self-worth are just a few of the things that contribute to making up the psychological uniqueness of every human being on the planet. In addition to the above, researchers are actively examining the role of spirituality in mental health and recovery. No one factor can be said to be the sole cause of mental illness; rather, disorders result from a complex set of forces that act upon each person as an individual. Finding the various elements that contributed to the onset of an illness requires considerable patience from the patient, his or her family, and health workers. Identifying all factors, if possible, provides the best road map for the healing process.

New directions

In the future, scientists will certainly modify and expand our thought-models about the mind and brain. For example, a new treatment called transcranial magnetic stimulation (TMS) is being evaluated as an alternative to electric shock therapy. TMS uses powerful magnets instead of electricity, and is delivered to specific areas of the brain. Hence, in the future scientists must integrate some of the electromagnetic aspects of nature into the mind-brain puzzle. In addition, the National Institute of Mental Health (NIMH) is researching alternative healing modalities. Prominent among them is **acupuncture**, which has been used to treat depression, anxiety and panic disorder. Other alternative treatments being studied include the effects of prayer, **meditation**, creative writing, and **yoga**.

Deeper exploration of the human condition is both inevitable and desirable. Perhaps researchers will find better answers by asking the question, "What makes people healthy?" instead of simply looking at what makes us sick. In the end, researchers may find proof of some of the ancient truths taught by spiritual teachers from all traditions; and that the physical changes seen with human eyes or under a microscope are really just the symptoms of and not the causes of imbalances.

See also Genetic factors and mental disorders; Psychoanalysis

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Oxazepam

Definition

Oxazepam is a member of a family of tranquilizers known as benzodiazepines. It is sold in the United States under the brand name Serax and in Canada under the brand name Ox-Pam. Generic forms of oxazepam are also available.

Purpose

Oxazepam is prescribed to treat feelings of tension and anxiety. It is also used to calm patients who are suffering from the symptoms of alcohol withdrawal.

Description

Oxazepam is one of several drugs in the class called benzodiazepines. Oxazepam slows down certain **brain** functions by blocking specific chemicals that transmit messages among the nerve cells in the brain.

Recommended dosage

The typical starting dose for adults ranges from 5–15 mg per day. The dosage is sometimes increased by the doctor, but 80 mg is usually the maximum amount prescribed per day. The amount used each day is typically divided into at least two doses. Oxazepam is taken by mouth, and is available in tablets and capsules. It can be taken with food if the patient is having side effects in the digestive tract.

Oxazepam is not FDA-approved for use in children under six years. However, often in clinical practice, the medication is used with close physician supervision. The typical starting dose for children aged two to 16 years is 5 mg. The doctor may increase this dose if necessary. Typically, the dose does not exceed 40 mg per day, and is given in divided doses. Children under two years of age may receive a dose based on body weight. The doctor must determine whether the child needs the drug as well as the dosage.

Precautions

The doctor should monitor the patient at regular intervals to ensure that the medicine is not causing troublesome side effects. Monitoring the patient is particularly important if the drug is being taken over a long period of time. Patients should not stop taking oxazepam suddenly, especially if they are taking large doses. The dose should be tapered (gradually decreased), and then stopped. Suddenly discontinuing oxazepam may cause a rebound effect. In a few cases patients have reported serious withdrawal symptoms when they stopped taking oxazepam, including nausea, vomiting, muscle cramps, and unusual irritability.

Oxazepam should be given with great care to elderly patients; to people who are significantly disabled; and to people with a history of liver or kidney disease, drug abuse, or breathing problems. Pregnant women should not take oxazepam because of the risk of birth defects in the baby. Likewise, nursing mothers should not use oxazepam while they breast-feed. Oxazepam and other benzodiazepines should never be combined with alcohol or other drugs that depress (lower the activity of) the central nervous system. Oxazepam and other benzodiazepines should be prescribed and used very carefully if they are given for long-term treatment because they are habit-forming. Patients who have been diagnosed with glaucoma or serious psychological disorders should not receive oxazepam. Patients who have a history of alcohol abuse, drug abuse, brain disease, mental depression, mental illness, sleep apnea, or myasthenia gravis should tell their doctor about their condition. Similarly, a woman who becomes pregnant while she is taking the drug should tell her doctor at once.

KEY TERMS

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Hallucination—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Myasthenia gravis—A disease characterized by weakness of the muscles caused by an autoimmune reaction.

Paranoid—A mental attitude characterized by unjustified or excessive distrust of other people, usually combined with anger.

Rebound effect—A physical reaction to stopping a medication characterized by the reappearance of the symptom(s) that the medication was given to suppress. For example, people who stop taking oxazepam may experience rebound excitability and sleeping problems.

Sleep apnea—Temporary stoppage of breathing during sleep that occurs often enough to significantly disrupt the patient's sleeping pattern.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

Side effects

Rare but serious side effects associated with the use of oxazepam include: anxiety, mental depression, reduced memory, and confusion. Even more rare are disorientation, **delusions**, **seizures**, unusually low blood pressure, sleeping difficulties, muscle weakness, and changes in behavior.

Less serious but more common side effects include: difficulty talking, dizziness, clumsiness, and drowsiness. Less

common but not particularly serious side effects include dry mouth, general weakness, headache, mild abdominal pain, constipation, diarrhea, nausea, and vomiting.

When the patient stops taking oxazepam, nervousness, irritability, and sleeping problems are common withdrawal side effects. Less common withdrawal side effects can include confusion, hearing problems, stomach cramps, increased sweating, mental depression, nausea, and vomiting. Rare withdrawal side effects can include seizures, **hallucinations**, and paranoid ideas.

Interactions

Patients should always inform every health professional that they deal with— doctors, pharmacists, nurses, dentists, and others— about every medication they take. Oxazepam, alcohol, and other medications that cause drowsiness can intensify one another's effects. Some medications that are used to treat viral infections, fungal infections, high blood pressure, and some heart rhythm problems can increase the effects of oxazepam.

Heavy smoking decreases the effectiveness of oxazepam.

See also: Alcohol and related disorders

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Pain disorder

Definition

Pain disorder is one of several somatoform disorders described in the revised, fourth edition of the mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders*** *(known as* DSM-IV-TR). The term "somatoform" means that symptoms are physical but are not entirely understood as a consequence of a general medical condition or as a direct effects of a substance, such as a drug. Pain in one or more anatomical sites is the predominant complaint and is severe enough to require medical or therapeutic **intervention**. Pain disorder is classified as a mental disorder because psychological factors play an important role in the onset, severity, worsening, or maintenance of pain.

Earlier names for this disorder include psychogenic pain disorder and somatoform pain disorder. There is some overlap in the meaning of these terms, but views regarding the nature of pain have been changing and they are, therefore, not equivalent diagnostic categories. Sometimes pain disorder is referred to as somatization, but this is an imprecise term and is easily confused with **somatization disorder**.

Description

In 1994, the International Association for the Study of Pain (IASP) defined pain as an unpleasant sensory or emotional experience arising from real or probable tissue damage. In other words, the perception of pain is, in part, a psychological response to noxious stimuli. This definition addresses the complex nature of pain and moves away from the earlier dualistic idea that pain is either psychogenic (of mental origin) or somatogenic (of physical origin). The contemporary view characterizes pain as multidimensional; the central nervous system, emotions, cognitions (thoughts), and beliefs are simultaneously involved.

When a patient's primary complaint is the experience of pain and when impairment at home, work, or school causes significant distress, a **diagnosis** of pain disorder may be warranted. The diagnosis is further differentiated by subtype; subtype is assigned depending on whether or not pain primarily is accounted for by psychological factors or in combination with a general medical condition, and whether the pain is acute (less than six months) or chronic (six months or more). The classification of pain states is important since the effectiveness of treatment depends on the aptness of the diagnosis of pain disorder and its type.

Causes and symptoms

Causes

Common sites of pain include the back (especially lower back), the head, abdomen, and chest. Causes of pain vary depending on the site; however, in pain disorder, the severity or duration of pain or the degree of associated disability is unexplained by observed medical or psychological problems.

The prevailing biopsychosocial model of mental disorders suggests that multiple causes of varying kinds may explain pain disorder, especially when the pain is chronic. There are four domains of interest:

- The underlying organic problem or medical condition, if there is one. For example, fibromyalgia (a pain syndrome involving fibromuscular tissue), skeletal damage, pathology of an internal organ, migraine headache, and peptic ulcer all have characteristic patterns of pain and a particular set of causes.
- The experience of pain. The severity, duration, and pattern of pain are important determinants of distress. Uncontrolled or inadequately managed pain is a significant stressor.
- Functional impairment and disability. Pain is exacerbated by loss of meaningful activities or social rela-

KEY TERMS

Biopsychosocial model—A hypothetical explanation for why something occurs that includes biological, psychological, and social causes or correlates.

Inter-rater reliability—The degree to which judgments about a person are consistent among raters or diagnosticians.

Multiaxial—Refers to a type of classification system that involves numeric measurement along more than one dimension and is not based on assignment to mutually exclusive categories.

Multimodal—Involving several types of therapeutic interventions such as heat or ice packs, electrical stimulation, ultrasound; sometimes refers to a mix of physical and psychological therapies.

Neuropathic—Relating to neural damage.

Painstates—Refers to the four-way classification of pain disorder as being (1) acute with psychological factors, (2) acute with psychological factors and a general medical condition, (3) chronic with psychological factors, and (4) chronic with psychological factors and a general medical condition.

Somatization—When mental or emotional distress is expressed physically in a way that disrupts body function.

tionships. Disruption or loss may lead to isolation and resentment or anger, which further increases pain.

- Emotional distress. Depression and anxiety are the most common correlates of pain, especially when the person suffering feels that the pain is unmanageable, or that the future only holds more severe pain and more losses.

In sum, there are multiple causes of pain disorder. A therapist or team of health professionals will weigh the relative causal contributions, assign priorities for therapeutic intervention, and address the several domains in a multimodal fashion. For example, the design of a treatment plan in a pain clinic may involve a physician, psychotherapist, occupational therapist, physical therapist, anesthesiologist, **psychologist**, and nutritionist.

Symptoms

Symptoms vary depending on the site of pain and are treated medically. However, there are common symptoms associated with pain disorder regardless of the site:

- negative or distorted cognition, such as feeling helpless or hopeless with respect to pain and its management
- inactivity, passivity, and/or disability
- increased pain requiring clinical intervention
- **insomnia** and **fatigue**
- disrupted social relationships at home, work, or school
- depression and/or anxiety

Demographics

There is very little information regarding rates of pain disorder. A major difficulty is that the diagnostic categories for psychogenic pain disorder in *DSM-III*, somatoform pain disorder in *DSM-III-R*, and pain disorder in *DSM-IV* and *DSM-IV-TR* are not equivalent. Furthermore, many criticize the somatoform disorder group (which includes pain disorder) as being an aggregate of disorders that are not truly distinct from one another. This lack of distinctiveness suggests to some researchers that a more appropriate system of classification should be dimensional rather than categorical. In other words, if shared dimensions or characteristics of the several somatoform disorders exist, differences among disorders should be a matter of degree along the possible dimensions. The critics of the *DSM* categorical approach would prefer a dimensional or multiaxial system because when classification systems are improved, the reliability and validity of measures assessing disorder improve, and better estimates of rates are possible.

Nevertheless, some researchers find the *DSM-IV* category for pain disorder useful. For example, in one study of psychiatric pain clinic outpatients, 79% met the criteria for pain disorder of the subtype where psychological factors and a general medical condition co-exist; 9% of the outpatients met the criteria for pain disorder with psychological factors and no medical condition. In another study of patients at a psychiatric clinic, 38% of the patients at admission and 18% of the outpatients reported significant pain. In comparison, 51% in a study of general medical and surgical inpatients met the criteria for pain disorder.

Currently, there are no good estimates for rates of pain disorder in the general population.

Diagnosis

A **psychiatrist** or mental health professional arrives at the diagnosis of pain disorder after considering several questions. An important preliminary question is whether the pain is entirely accounted for by a general medical condition. If so, the diagnosis of pain disorder is ruled out; and if not, the psychiatrist considers whether the pain

is feigned. If the psychiatrist believes the patient is pretending to be in pain, the patient is diagnosed as **malingering** for external rewards, such as seeking mood-altering drugs, or as having a **factitious disorder** that reflects the patient's need to adopt a sick role. Neither malingering nor factitious disorder is in the somatoform group.

The psychiatrist may employ a variety of methods to assess the severity of pain and the contribution of psychological factors to the experience of pain. These include structured interviews (where the questions asked are standardized), open or unstructured interviews, numerical rating scales, visual analog scales (where the patient makes a mark along a line to indicate severity of pain, or if the patient is a child, or is illiterate, selects a face to represent the degree of pain), and instruments such as the McGill Pain Questionnaire or the West Haven-Yale Multidimensional Pain Inventory.

There are several conditions that rule out a diagnosis of pain disorder:

- Dyspareunia. (The patient's primary complaint relates to the experience of painful sexual intercourse.)
- Somatization disorder. (The patient has a long history of pain that began prior to age 30 and involves the gastrointestinal, reproductive, and nervous systems.)
- Conversion disorder. (In addition to pain, there are other symptoms associated with motor or sensory dysfunction.)
- Mood, anxiety, or psychotic disorder. (Any one of these more fully accounts for the pain. This last exclusion rests upon a very subjective opinion. Subjectivity reduces inter-rater reliability and is one of the points raised by critics of the *DSM* category for pain disorder.)

A final consideration is whether the pain is acute or chronic.

Treatments

Depending on whether the pain is acute or chronic, management may involve one or more of the following: pharmacological treatment (medication); **psychotherapy** (individual or group); family, behavioral, physical, hypnosis, and/or occupational therapy. If the pain is acute, the primary goal is to relieve the pain. Customary agents are acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs); if opioid analgesics are prescribed, they often are combined with NSAIDs so that the dosage of opioids may be reduced. Psychotherapy is less important for the treatment of acute pain as compared to chronic pain disorder. In comparison, treatment of chronic pain disorder usually requires some sort of psychotherapy in combination with medication.

Antidepressants

Tricyclic antidepressants (TCAs) reduce pain, improve sleep, and strengthen the effects of opioids (such as codeine and oxycodone), as well as moderate depression. Relief of pain may occur in a few days while lessening of depression may take several weeks. Usually, TCAs for pain are prescribed at doses 33% to 50% lower than when prescribed for depression. TCAs are particularly effective for neuropathic pain, headache, facial pain, fibromyalgia, and arthritis.

Treatment of sleep dysfunction

Pain and depression diminish the restorative quality of sleep. When the cycle of pain, depression, insomnia, and fatigue is established, it tends to be self-perpetuating. Treatment may include antidepressants, relaxation training, and education regarding good sleep hygiene.

Cognitive-behavioral therapy

Many people who suffer chronic pain experience isolation, distress, frustration, and a loss of confidence regarding their ability to cope; subsequently, they may adopt a passive, helpless style of problem solving. The goal of **cognitive-behavioral therapy** (CBT) is to restore a sense of self-efficacy by educating patients about the pain-and-tension cycle, by teaching them how to actively manage pain and distress, and by informing them about the therapeutic effects of their medications. CBT is time-limited, structured, and goal-oriented.

Some tension-reducing techniques include progressive muscle relaxation, visual imagery, hypnosis, and **biofeedback**. Pain diaries are useful for describing daily patterns of pain and for helping the patient identify activities, emotions, and thoughts that alleviate or worsen pain. Diaries also are useful in evaluating the effectiveness of medication. Patients may be taught pacing techniques or scheduling strategies to restore and maintain meaningful activities.

The cognitive aspect of CBT is based on cognitive-social learning theory. The focus is on helping the patient to restructure his or her ideas about the nature of pain and the possibility of effective self-management. In particular, the patient is taught to identify and then modify negative or distorted thought patterns of helplessness and hopelessness.

Operant conditioning

The principles of operant conditioning are taught to the patient and family members so that activity and non-pain behaviors are reinforced or encouraged. The goal is

to eliminate pain behaviors, such as passivity, inactivity, and over-reliance on medication.

Other Treatments

Other treatments effective in the management of pain include **acupuncture**, transcutaneous electrical nerve stimulation (TENS), trigger point injections, massage, nerve blocks, surgical ablation (removal of a part or pathway), **meditation**, exercise, **yoga**, music and art therapy.

Prognosis

The prognosis for total remission of symptoms is good for acute pain disorder and not as promising for chronic pain disorder. The typical pattern for chronic pain entails occasional flare-ups alternating with periods of low to moderate pain. The prognosis for remission of symptoms is better when patients are able to continue working; conversely, unemployment and the attendant isolation, resentment, and inactivity are correlates of a continuing pain disorder. Additionally, if **reinforcement** of pain behavior is in place (for example, financial compensation for continuing disability, an overly solicitous spouse, abuse of addictive drugs), remission is less likely.

The results of outcome studies comparing pain disorder treatments point to cognitive-behavioral therapy in conjunction with antidepressants as the most continually effective regimen. However, people in chronic pain may respond better to other treatments and it is in keeping with the goal of active self-management for the patient and health professional(s) to find an individualized mix of effective coping strategies.

Prevention

Pain disorder may be prevented by early intervention i.e., at the onset of pain or in the early stages of recurring pain. When pain becomes chronic, it is especially important to find help or learn about and implement strategies to manage the distress before inactivity and hopelessness develop. Most patients in pain first contact their primary care physician who may make a referral to a mental health professional or pain clinic. Many physicians will reassure the patient that a referral for psychological help is not stigmatizing, does not in any way minimize the experience of pain or the medical condition, and does not imply that the physician believes the pain is imaginary. On the contrary, the accepted IASP definition of pain fully recognizes that all pain is, in part, an emotional response to actual damage or to the threat of damage.

See also: Abuse; Assessment and diagnosis; Creative therapies; Personality disorders

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American Academy of Pain Medicine. 4700 W. Lake, Glenview, IL 60025. (847) 375-4731. <http://www.painmed.org>.

The American Chronic Pain Association. PO Box 850, Rocklin, CA 95677. (916) 632-3208. <http://www.theacpa.org>.

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Pamelor *see* **Nortriptyline**

Panic attack

Definition

Panic attacks, the hallmark of **panic disorder**, are discrete episodes of intense anxiety. Panic attacks can also be experienced by people with specific phobia, **social phobia**, or by people who have used or consumed certain substances, such as cocaine.

Description

Panic attacks are intense anxiety experiences that are usually accompanied by symptoms in the affected person's body and thinking. The panic attack can occur unexpectedly during early stages of panic disorder illness. As panic disorder progresses, panic attacks may become associated with certain situations that trigger attacks. Panic attacks triggered by a specific experience are called situational panic attacks, since a certain situation initiates the intense anxiety.

Persons affected with panic attacks usually exhibit a broad range of clinical signs and symptoms that include:

- heart palpitations (accelerated heart rate)
- shaking or trembling
- sweating
- shortness of breath or sensation of feeling smothered or choked
- feeling of tingling
- chest discomfort or pain
- nausea or abdominal distress
- feeling dizzy, light headed, unsteady or faint
- perceptions of being detached from oneself (**depersonalization**), or a feeling out of touch with reality (derealization)
- chills or hot flashes
- fear of dying
- fear of going crazy or losing control

A person meets the criteria for a panic attack if the symptoms start abruptly, reach a quick peak (usually within 10 minutes), and if the affected individual has at least four symptoms as listed above. In persons who have less than four symptoms during an attack, the disorder is called a limited symptom attack.

It is typical that affected persons who seek treatment usually have one to two attacks a week and in worse periods may have one daily attacks or several within a week.

As stated, panic attacks can be experienced as a result of stimulant chemical usage, such as cocaine usage. There is evidence to suggest that persons with panic attacks are sensitive to certain chemicals such as caffeine, carbon dioxide, antihistamines, and, in women, progesterone replacement. Exposure to these substances may precipitate an attack.

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Panic disorder

Definition

Panic disorder is a condition in which the person with the disorder suffers recurrent panic attacks. Panic attacks are sudden attacks that are not caused by a substance (like caffeine), medication, or by a medical condition (like high blood pressure), and during the attack, the sufferer may experience sensations such as accelerated or irregular heartbeats, shortness of breath, dizziness, or a fear of losing control or "going crazy." The sudden attack builds quickly (usually within 10 minutes) and is almost paralyzing in its severity. When a **diagnosis** of panic disorder is given, the disorder can be considered one of two different types—panic disorder with or without **agoraphobia**.

The handbook for mental health professionals (called the ***Diagnostic and Statistical Manual of Mental Disorders****, or the* DSM-IV-TR) classifies both types of panic disorder as anxiety disorders.

Panic disorder without agoraphobia

Panic disorder without agoraphobia is defined by the *DSM-IV-TR* as a disorder in which patients are plagued by panic attacks that occur repeatedly and without warning. After these attacks, the affected individual worries for one month or more about having more embarrassing attacks, and may change his or her behavior with regard to these attacks. For example, a patient may fear that he or she has a cardiac condition, and may quit a job or quit exercising because of the fear. Patients may also worry that they are going to lose control or appear insane to other people. Panic disorder without agoraphobia has a less severe set of symptoms than panic disorder with agoraphobia. Patients without agoraphobia do not become housebound—they suffer panic attacks but do not have significant interference in their level of function and are still able to accomplish their daily activities.

Panic disorder with agoraphobia

People who suffer from this kind of panic disorder may experience their agoraphobia in one of two ways. They may experience sudden, unexpected panic attacks

KEY TERMS

Agoraphobia—People with this condition worry that they will not be able to get help or flee a place if they have a panic attack; they may refuse to go to places that might trigger a panic attack.

Amygdala—An almond-shaped brain structure in the brain's limbic system that is activated in acute stress situations to trigger the emotion of fear.

Cognitive-behavioral therapy (CBT)—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

GABA—Gamma-aminobutyric acid, an inhibitory neurotransmitter in the brain.

Hypersensitive internal suffocation alarm—A sensitive alarm goes off and the affected person's brain sends the body false signals that not enough oxygen is being received, causing an increase in their breathing rate.

Locus ceruleus—A part of the brain where the neurotransmitter causes excitation.

Panic attack—Specific periods of time when a person has a feeling that s/he is dying or having a heart attack with chest pain, a feeling as though s/he could pass out, and fear that s/he is going insane.

Panic disorder with agoraphobia—Repeated panic attacks in which the patient is worried about the attacks enough that the worry restricts their activity.

Panic disorder without agoraphobia—Repeated panic attacks without symptoms of agoraphobia.

that cause them to fear being in a place where help might not be available; or, they may experience sudden panic attacks in specific, known situations, and fear those situations or places that may trigger attacks. In either case, the fear of further panic attacks restricts the affected person's activities. For example, people whose attacks are triggered by being in crowds may avoid shopping malls for fear that they will be in a crowd and have a **panic attack**. Or, a person may experience sudden, debilitating panic attacks without a particular trigger, and, as a result, he or she is afraid to go to a supermarket (or similar place) for fear that a panic attack could occur while there and no one could help.

Description

Panic disorder can be very difficult to distinguish from other mental illnesses such as major depression, other anxiety disorders, or medical conditions such as heart attacks. Panic attacks differ from general anxiety in that they are episodes that last for discrete periods of time and the symptoms that people suffer are more intense. Panic attacks have three types: unexpected, situationally bound, and situationally predisposed. The unexpected attacks occur without warning and without a trigger. The situationally bound attacks happen repeatedly when the person is performing some activity, about to do that activity, or even when the person thinks about doing that activity. For example, a person whose panic attacks are triggered by being in crowds can have an attack just by thinking about going to a shopping mall. Situationally predisposed attacks are similar to the situationally bound attacks, except that they do not always occur when the trigger stimulus is encountered. For example, someone who experiences panic attacks while in crowds may sometimes be in crowds and not experience attacks, or may experience attacks in other, non-crowded situations, as well.

Patients who suffer from panic disorder without treatment usually have a diminished quality of life and end up spending excessive money on health care because of frequent visits to emergency rooms and to other medical doctors. However, very effective treatments for panic disorder exist.

Agoraphobia is a fear of being in a place or situation from which escape might be difficult or embarrassing, or in which help may not be available in the case of a panic attack. It is not clear why some people develop agoraphobia and other people do not. Many people may develop their agoraphobia symptoms right after their first attack, but others do not develop agoraphobia until sometimes years after their attacks began.

Causes and symptoms

Causes

BIOCHEMICAL/PHYSIOLOGICAL CAUSES. It is extremely difficult to study the **brain** and the underlying causes of psychiatric illness; and understanding the chemistry of the brain is the key to unlocking the mystery of panic disorder. The amygdala is the part of the brain that causes fear and the response to **stress**. It has been implicated as a vital part of anxiety disorders. Sodium lactate, a chemical that the body produces when muscles are fatigued, and carbon dioxide are known to induce panic attacks. These substances are thought to inhibit the release of **neurotransmitters** in the brain, which leads to the panic attacks. One hypothesis is that sodium lactate

stimulates the amygdala and causes panic attacks. Another hypothesis is that patients with panic disorder have a hypersensitive internal suffocation alarm. This means that the patient's brain sends the body false signals that not enough oxygen is being received, causing the affected person to increase his or her breathing rate. Panic disorder patients have attacks when their overly sensitive alarm goes off unpredictably. Yohimbine, a drug used to treat male sexual dysfunction, stimulates a part of the brain called the locus ceruleus and induces panic symptoms thus pointing to this area of the brain's involvement in panic disorder. Brain neurotransmitters serotonin and GABA are suspected to be involved in causing the disorder, as well.

GENETICS. Genetics also plays a pivotal role in the development of panic disorder. Twin studies have demonstrated that there is a higher concordance in identical versus fraternal twins thus supporting the idea that panic disorders are inherited. Family studies have also demonstrated that panic attacks run in families. Relatives of patients with panic disorder are four to 10 times more likely to develop panic disorder. People who develop early onset of panic attacks in their mid-20s are more likely to have relatives who have panic disorder. When relatives of patients with panic disorder are exposed to high levels of carbon dioxide, they have panic attacks. Another hypothesis is that patients with panic disorder who develop agoraphobia have a more severe form of the disease. Current efforts to identify a gene for panic disorder have not been successful.

PERSONAL VARIABLES. There are several themes in the psychology of panic disorder. Research has shown that patients who develop panic disorder have difficulty with anger. They also have difficulty when their job responsibilities are increased (as in the case of a promotion), and are sensitive to loss and separation. People with this disorder often have difficulty getting along with their parents, whom they see as controlling, critical, and demanding, causing the patients to feel inadequate. Early maternal separation is thought to be an underlying cause of panic disorder.

Panic disorder patients also have a pattern of dependency in their interpersonal relationships. As children, people with panic disorder relied on parents to protect them from fear. As a result, they develop an angry dependence on their parents and fear detaching from them. They constantly feel as though they are trapped.

There is also an association between sexual **abuse** and patients who have panic attacks. Sixty percent of female patients with panic disorder were sexually abused as children. This explains their difficulty with developing trusting relationships.

Symptoms

PANIC ATTACK SYMPTOMS. The *DSM-IV-TR* lists thirteen symptoms to meet the criteria for a diagnosis of panic attack. The affected person must have four or more of these symptoms within ten minutes of the beginning of an attack in order to meet the panic attack criteria:

- bounding or pounding heartbeat or fast heart rate
- sweating
- shaking
- shortness of breath
- feeling of choking
- pains in the chest; many people they feel as though they are having a heart attack
- nausea or stomach ache
- feeling dizzy or lightheaded as if he or she is going to pass out
- feeling of being outside of one's body or being detached from reality
- fear that he or she is out of control or crazy
- fear that he or she is going to die
- feeling of tingling or numbness
- chills or hot flashes

Symptoms of panic disorder without agoraphobia

The *DSM-IV-TR* criteria for panic disorder without agoraphobia include:

- recurrent panic attacks (see above) that occur without warning for one month
- persistent worry that panic attacks will recur
- possible change in behavior because of that fear
- no agoraphobia
- not due to a medical condition or substance abuse
- not due other mental illness like specific phobia, **social phobia**, **obsessive-compulsive disorder**, **separation anxiety disorder**, or **post-traumatic stress disorder**

Symptoms of panic disorder with agoraphobia

The *DSM-IV-TR* criteria for panic disorder with agoraphobia are the same as above, but agoraphobia is present. The symptoms of agoraphobia include fear of being in situations that can trigger panic attacks, and avoiding places where attacks have occurred because of the affected person's fear that he or she will not be able to leave, or will not be able to get help. People with this condition may need to have another person accompany them when going to a place that may trigger anxiety attacks.

Sometimes this fear can be so severe that the person becomes housebound. This fact is important to consider because 15% of the general population can have one spontaneous panic attack without the recurrence of symptoms.

Demographics

Factors such as race, gender and socioeconomic status are important factors in the development of panic disorder. An individual has a chance of between one and two percent of developing panic disorder with or without agoraphobia. The symptoms usually begin when the person is in his or her early to mid-twenties. Women are twice as likely as men to develop panic attacks regardless of age. The National Institute of Mental Health Epidemiologic Catchment Area Study (ECA) shows no real significant differences between the races or ethnic groups, although it appears that African American and Hispanic men between the ages of 40 and 50 have lower rates of panic disorder than white men. Panic disorder patients are at increased risk for major depression and the development of agoraphobia. According to ECA studies, an individual with panic disorder has a 33% chance of developing agoraphobia. People without panic disorder only have a 5.5% chance of developing agoraphobia. Again, women were more likely to develop agoraphobia than men. Over the course of their lifetime, African Americans were more likely to develop agoraphobia than whites or Hispanics. Agoraphobia is more prevalent among people with less education and lower economic class.

Diagnosis

Differential diagnosis

Differential diagnosis is the process of distinguishing one diagnosis from other, similar diagnoses. Panic disorder can be difficult to distinguish from other anxiety disorders such as specific phobia and social phobia. However, in general, specific phobia is cued by a specific trigger or stimulus and social phobia by specific social situations, while the panic attacks of panic disorder are completely uncued and unexpected. In certain cases, it may be difficult to distinguish between certain, situational phobias and panic disorder with agoraphobia, and the mental health professional must use the *DSM* and professional judgment in these cases. Panic attacks that occur during sleep and wake the person up are more characteristic of panic disorder, than are the other disorders that include panic attacks. It can be distinguished from posttraumatic stress disorder (PTSD), obsessive-compulsive disorder (OCD), and **generalized anxiety disorder** (GAD) again by what cues the attacks. In PTSD, thinking about the traumatic event can trigger attacks. In obsessive-compulsive disorder, worries about getting dirty can fuel an attack of anxiety. In generalized anxiety disorder, general worries or concerns can lead to the symptoms of panic. However, in panic disorder, a main component is that the affected individual fears recurrent panic attacks.

Panic attacks can often be difficult to distinguish from other physical problems such as hyperthyroidism, hyperparathyroidism, seizure disorder, and cardiac disease. If patients are middle aged or older and have other complaints, including dizziness and headaches, their attacks are more likely to be another medical problem and not panic attacks. Panic attacks can also be difficult to distinguish from drug abuse since any drug that stimulates the brain can cause the symptoms. For example, cocaine, caffeine, and **amphetamines** can all cause panic attacks. Therefore, a person must be free of all drugs before a diagnosis of panic disorder can be made. Many patients may attempt to self-medicate with alcohol to try to calm down. Withdrawal from alcohol can lead to worse panic symptoms. The patient may believe that he or she is reducing symptoms while actually exacerbatng their panic attacks.

Dual diagnosis

Individuals with panic disorders have a high rate of coexisting depression. Patients who have panic disorder have about a 40–80% chance of developing major depression. In most situations, the panic disorder happens first and the depression comes later. Patients are also at risk for substance abuse difficulties as a result of attempts to stop attacks. These attempts may involve the use of alcohol, illicit or unprescribed sedatives, or benzodiazepines (medications that slow down the central nervous system, having a calming effect). Patients with panic disorder are not at high risk for **suicide** attempts. A recent Harvard-Brown study showed that people with panic disorder with or without agoraphobia are not at risk for suicide unless they have other conditions such as depression or substance abuse.

Psychological measures and diagnostic testing

Currently there is no diagnostic test for panic disorder. Any patient who has panic attacks should receive a thorough medical examination to rule out any medical condition. Patients should have baseline blood counts and glucose should be measured. Patients with cardiac symptoms need a cardiac workup and should see their primary medical doctor. Patients who have complaints of dizziness should receive a thorough neurological evaluation. There are several psychological inventories that can help the clinician diagnose panic disorder including the **Beck**

Depression Inventory (BDI), Beck Anxiety Inventory (BAI), Specific Fear Inventory, Clinical Anxiety Scale (CAS), and the Clinical Global Inventory (CGI).

Treatments

Psychological and social interventions

A psychotherapeutic technique that is critical to the treatment of panic disorder is **cognitive-behavioral therapy** (CBT). Patients are panic-free within six months in about 80–90% of cases. Some people even experience long-term effects after the treatments have been stopped. About half of the patients say that they have rare attacks even two years after treatment has ended.

New studies reveal that the approach to treating panic disorder should have three aspects: the cognitive, the physiological, and the behavioral. The cognitive techniques try to focus on changing the patient's negative thoughts—for example, "I will die if I don't get help." Patient education about symptoms is also critical to the treatment of panic attacks. In one physiological approach, patients are taught breathing techniques in an effort to try to help them lower their heart rate and decrease their anxiety. Repeated exposure to physical symptoms associated with the panic disorder is also a part of treatment. The patients cause themselves to hyperventilate in effort to reproduce the panic symptoms. In behavioral approaches, the individual who experiences panic attacks also needs to be exposed to situations that he or she may have previously feared. A patient can also be taken to places associated with agoraphobia with the therapist.

Some patients may benefit from **psychodynamic psychotherapy** and **group therapy**. Psychodynamic **psychotherapy** explores thoughts and ideas of the person's subconscious. It takes a longer time to achieve efficacy than cognitive-behavioral therapy, but it can be just as effective for patients with panic disorder. Group therapy is also just as helpful to some patients as CBT. **Support groups** can also be helpful to some patients. It can be very therapeutic and healing to the individual to discuss their problems with someone who has actually experienced the same symptoms. Patients can learn from each other's coping styles.

Medical treatments

Panic disorder patients have a 50–80% chance of responding to treatment, which attempts to block the symptoms of panic attacks. Treating the agoraphobia symptoms is more challenging. Developing some anti-panic regimens that address all symptoms is important. The Food and Drug Administration (FDA) to treat panic disorder approves only five classes of drugs. They are:

- benzodiazepines
- Selective serotonin reuptake inhibitors (SSRIs), which cause a buildup of serotonin. This buildup is thought to cause the antidepressant effect.
- Tricylic antidepressants (TCAs).
- Monoamine oxidase inhibitors (MAOIs) and reversible MAOIs, which inhibit the breakdown of neurotransmitters in the brain, including dopamine and serotonin.
- Atypical antidepressants, including **bupropion** (Wellbutrin), **mirtazapine** (Remeron), **trazodone** (Desyrel), and others.

Patients should first be started on a low-dose SSRI and then the dose should be increased slowly. Patients with panic disorder are extremely sensitive to the side effects that many patients experience in the first weeks of antidepressant therapy. Patients should also have a benzodiazepine, such as **clonazepam** (Klonopin) or **alprazolam** (Xanax), in the first weeks of treatment until the antidepressant becomes therapeutic. Most people need the same dose of antidepressant as patients with major depression. About 60% of patients will have improvement in their symptoms while taking an antidepressant and a benzodiazepine. Patients with mitral valve prolapse may benefit from a beta blocker. Patients who have tried an SSRI, and after six weeks, show no improvement can be switched to another SSRI, benzodiazepine, TCA, MAOI, or **venlafaxine** (Effexor). An SSRI should be stopped if the patient has intolerable side effects such as loss of sexual libido, weight gain, or mild form of manic depression. When SSRIs are stopped, it is important that the dosage is gradually tapered because patients can suffer symptoms when it is abruptly withdrawn. These symptoms may include confusion, anxiety and poor sleep.

Alternative therapies

Some alternative therapies for panic disorder are hypnosis, **meditation**, **yoga**, proper nutrition, exercise, and abdominal breathing techniques that foster relaxation and visualization. Visualization is imagining oneself in the stressful situation while relaxed so that coping strategies can be discovered. The herb **kava kava** has been studied in trials to treat anxiety attacks and has been found to be effective in some clinical trials; but has not been studied intensely enough to determine its benefits and side effects, and has been associated liver toxicity. The National Center for Complementary and Alternative Medicine was going to conduct two research studies of kava kava but as of 2002 it has suspended the trials until

the FDA has determined whether or not the herbal supplement is safe.

Prognosis

Patients with panic disorder have a poor prognosis particularly if untreated. Patients often relapse when they attempt to discontinue treatment. However, if patients are compliant and willing to stay in treatment, then the long-term prognosis is good. According to one study, eight years after treatment has been done, 30–40% of patient are doing better. Only 10–20% of patients do poorly. The patient with panic attacks has a relapsing and remitting course that can be worsened by significant stressors such as the death of the spouse or divorce. Cognitive-behavioral therapy has an 80–90% chance that the patient will benefit six months after treatment. Medications have a 50–80% efficacy. If patients are committed to staying in treatment, their prognosis is very favorable.

Prevention

Although panic disorder is not totally preventable, individuals with a strong family history of them who are susceptible to panic atacks are encouraged to be aware of the symptoms and get treatment early. **Compliance** with treatment is important to the recovery from panic disorder.

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National Center for Complementary and Alternative Medicine. P.O. Box 7923, Gaithersburg, MD 20898. (888) 644-6226. <http://nccam.nih.gov>.

National Institute of Mental Health. 6001 Executive Boulvevard, Rm.8184, MSC9663,Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov/anxiety/panicmenu.cfm>.

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Susan Hobbs, M.D.

Paranoia

Definition

Paranoia is a symptom in which an individual feels as if the world is "out to get" him or her. When people are paranoid, they feel as if others are always talking about them behind their backs. Paranoia causes intense feelings of distrust, and can sometimes lead to overt or covert hostility.

Description

An individual suffering from paranoia feels suspicious, and has a sense that other people want to do him or her harm. As a result, the paranoid individual changes his or her actions in response to a world that is perceived as personally threatening. Objective observers may be quite clear on the fact that no one's words or actions are actu-

ally threatening the paranoid individual. The hallmark of paranoia is a feeling of intense distrust and suspiciousness that is not in response to input from anybody or anything in the paranoid individual's environment.

Other symptoms of paranoia may include

- Self-referential thinking: The sense that other people in the world (even complete strangers on the street) are always talking about the paranoid individual.
- Thought broadcasting: The sense that other people can read the paranoid individual's mind.
- Magical thinking: The sense that the paranoid individual can use his or her thoughts to influence other people's thoughts and actions.
- Thought withdrawal: The sense that people are stealing the paranoid individual's thoughts.
- Thought insertion: The sense that people are putting thoughts into the paranoid individual's mind.
- Ideas of reference: The sense that the television and/or radio are specifically addressing the paranoid individual.

Demographics

Paranoia is a very human feeling. Nearly everyone has experienced it at some or another time, to varying degrees. Paranoia exists on a continuum, ranging from a feeling of distrust due to an occasional misinterpretation of cues that can be appropriately dealt with and reinterpreted, to an overarching pattern of actual paranoia that affects every interpersonal interaction.

Some research studies have suggested that 6% of all women and 13% of all men have some chronic level of mistrust towards the motivations of others towards them. Only about 0.5% to 0.25% of men and women can actually be diagnosed with **paranoid personality disorder**, however. It remains interesting to researchers that men are more prone to paranoid traits and mental disorders with paranoid features than are women.

Causes of paranoia

Researchers do not understand fully what chemical or physical changes in the **brain** cause paranoia. Paranoia is a prominent symptom that occurs in a variety of different mental disorders, as well as a symptom of certain physical diseases. Furthermore, use of certain drugs or chemicals may cause symptoms of paranoia in an otherwise normal individual.

Paranoia is often manifested as part of the symptom complex of **schizophrenia**. In fact, one of the subtypes of schizophrenia is termed "paranoid schizophrenia," which actually refers to a type of schizophrenia in which the individual is particularly preoccupied with **delusions** in which the world seems to be pitted against him or her. As with other forms of schizophrenia, sufferers often lack contact with reality, and display **hallucinations**, flat or emotionless **affect**, and disorganized thinking and behavior.

Paranoid personality disorder is diagnosed when an individual does not have other symptoms of schizophrenia, but a personality that is driven by chronic manifestations of paranoia. These individuals are mistrustful, suspicious, and convinced that the world is out to get them.

In order for an individual to be diagnosed with paranoid personality disorder, he or she must display at least four of the following traits:

- chronically suspicious that people are lying or cheating him or her in some way
- frequently preoccupied with whether people are loyal or trustworthy
- cannot confide in others for fear of being betrayed
- misinterprets benign comments or events as being personally threatening
- harbors long-term grudges against others who are perceived as having been threatening or insulting in some way
- sees others' actions and/or words attacking him or her in some way, and therefore goes on the counterattack
- repeatedly assumes that partner or spouse is unfaithful

Paranoia can also occur as a symptom of other neurological diseases. Individuals suffering from the aftereffects of strokes, brain injuries, various types of **dementia** (including **Alzheimer's disease**), Huntington's disease, and Parkinson's disease may manifest paranoia as part of their symptom complex. The paranoia may decrease in intensity when the underlying disease is effectively treated, although since many of these diseases are progressive, the paranoia may worsen over time along with the progression of the disease's other symptoms.

A number of different medications and drugs can cause paranoia. These include corticosteroid medications, H-2 blockers (cimetidine, ranitidine, famotidine), some muscle relaxants (Baclofen), antiviral/anti-Parkinson drugs (**amantadine**), some **amphetamines** (including methylphenidate, or Ritalin), anti-HIV medications, antidepressants (Nardil). Abused drugs that can prompt paranoia include alcohol, cocaine, marijuana, ecstasy (MDMA), amphetamines (including Ritalin), LSD, and PCP (angel dust). Withdrawal from addictive drugs may also cause symptoms of paranoia.

Treatments

It can be quite challenging to get an individual who is suffering from paranoia to accept treatment. Their paranoid condition makes them distrustful of people's motivations towards them, so that even a medical doctor appears to be a suspicious party. Medications that may be offered are usually looked at with great distrust, and efforts at **psychotherapy** are considered "mind control" by a profoundly paranoid individual.

The first step to be taken when someone is suffering from paranoia is that of determining whether an easily reversible situation (such as an adverse reaction to a medication) might be causing the paranoia. If so, discontinuing the drug (either immediately or by gradually weaning the dose) might end the symptoms of paranoia.

Patients who have other diseases, such as Alzheimer's disease or other forms of dementia, Huntington's disease, or Parkinson's disease may notice that their paranoid symptoms improve when their general medical condition is treated. The circumstance that can occur as their underlying disease progresses, is that the paranoia may return or worsen over time.

People who are suffering from diagnosable mental conditions such as schizophrenia or paranoid personality disorder may benefit from the use of typical antipsychotic medications, such as **chlorpromazine** or **haloperidol**, or from the newer, atypical antipsychotic medications, such as **clozapine**, **olanzapine**, or **risperidone**.

Cognitive-behavioral therapy (CBT) or other forms of psychotherapy may be helpful for certain people who have paranoia. CBT attempts to make a person more aware of his or her actions and motivations, and tries to help the individual learn to more accurately interpret cues around him or her, in an effort to help the individual change dysfunctional behaviors. Difficulty can enter into a therapeutic relationship with a paranoid individual, due to the level of mistrust and suspicion that is likely to interfere with their ability to participate in this form of treatment.

Support groups can be helpful for some paranoid individuals—particularly helpful in assisting family members and friends who must learn to live with, and care for paranoid individuals.

Prognosis

It is difficult to predict the prognosis of an individual who has paranoia. If there is an underlying mental illness, such as schizophrenia or paranoid personality disorder, then the paranoia is likely to be a lifelong condition. It may improve with some treatments (remission), only to become exacerbated under other more stressful conditions, or with changes in medication.

Individuals who have symptoms of paranoia as part of another medical condition may also have a waxing-and-waning-course.

When paranoia is caused by the use of a particular drug or medication, it is possible that discontinuing that substance may completely reverse the symptoms of paranoia.

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National Institute for Mental Health. 6001 Executive Blvd., Room 8184, MSC 9663, Bethesda, MD 20892. (301)443-4513. <http://www.nimh.nih.gov>.

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Paranoid personality disorder

Definition

People with paranoid personality disorder (PPD) have long-term, widespread and unwarranted suspicions that other people are hostile, threatening or demeaning. These beliefs are steadfastly maintained in the absence of any real supporting evidence. The disorder, whose name comes from the Greek word for "madness," is one of ten **personality disorders** described in the 2000 edition of the ***Diagnostic and Statistical Manual of Mental Disorders,*** (the fourth edition, text revision or *DSM-IV-TR*), the standard guidebook used by mental health professionals to diagnose mental disorders.

Despite the pervasive suspicions they have of others, patients with PPD are not delusional (except in rare, brief instances brought on by **stress**). Most of the time, they are in touch with reality, except for their misinterpretation of others' motives and intentions. PPD patients are not psychotic but their conviction that others are trying to "get them" or humiliate them in some way often leads to hostility and social isolation.

Description

People with PPD do not trust other people. In fact, the central characteristic of people with PPD is a high degree of mistrustfulness and suspicion when interacting with others. Even friendly gestures are often interpreted as being manipulative or malevolent. Whether the patterns of distrust and suspicion begin in childhood or in early adulthood, they quickly come to dominate the lives of those suffering from PPD. Such people are unable or afraid to form close relationships with others.

They suspect strangers, and even people they know, of planning to harm or exploit them when there is no good evidence to support this belief. As a result of their constant concern about the lack of trustworthiness of others, patients with this disorder often have few intimate friends or close human contacts. They do not fit in and they do not make good "team players." Interactions with others are characterized by wariness and not infrequently by hostility. If they marry or become otherwise attached to someone, the relationship is often characterized by pathological jealousy and attempts to control their partner. They often assume their sexual partner is "cheating" on them.

People suffering from PPD are very difficult to deal with. They never seem to let down their defenses. They are always looking for and finding evidence that others are against them. Their fear, and the threats they perceive in the innocent statements and actions of others, often contributes to frequent complaining or unfriendly withdrawal or aloofness. They can be confrontational, aggressive and disputatious. It is not unusual for them to sue people they feel have wronged them. In addition, patients with this disorder are known for their tendency to become violent.

Despite all the unpleasant aspects of a paranoid lifestyle, however, it is still not sufficient to drive many people with PPD to seek therapy. They do not usually walk into a therapist's office on their own. They distrust mental health care providers just as they distrust nearly everyone else. If a life crisis, a family member or the judicial system succeeds in getting a patient with PPD to seek help, therapy is often a challenge. Individual counseling seems to work best but it requires a great deal of patience and skill on the part of the therapist. It is not unusual for patients to leave therapy when they perceive some malicious intent on the therapist's part. If the patient can be persuaded to cooperate— something that is not easy to achieve— low-dose medications are recommended for treating such specific problems as anxiety, but only for limited periods of time.

If a mental health care provider is able to gain the trust of a patient with PPD, it may be possible to help the patient deal with the threats that they perceive. The disorder, however, usually lasts a lifetime.

KEY TERMS

Delusion—A false belief that is resistant to reason or contrary to actual fact.

Delusional disorder of the persecutory type—A psychotic disorder characterized by a patient's belief that others are conspiring against him or her.

Hallucination—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Neuroleptic—Another name for the older antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

Paranoia—A mental disorder characterized by baseless suspicions or distrust of others, often delusional in intensity.

Paranoid personality—A personality disorder characterized by unwarranted suspicion, jealousy, hypersensitivity, social isolation and a tendency to detect malicious intent in the words and actions of others.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Rapport—A relation of empathy and trust between a therapist and patient.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Supportive—An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or exploratory approaches to treatment.

Causes and symptoms

Causes

No one knows what causes paranoid personality disorder, although there are hints that familial factors may

influence the development of the disorder in some cases. There seem to be more cases of PPD in families that have one or more members who suffer from such psychotic disorders as **schizophrenia** or **delusional disorder**.

Other possible interpersonal causes have been proposed. For example, some therapists believe that the behavior that characterizes PPD might be learned. They suggest that such behavior might be traced back to childhood experiences. According to this view, children who are exposed to adult anger and rage with no way to predict the outbursts and no way to escape or control them develop paranoid ways of thinking in an effort to cope with the stress. PPD would emerge when this type of thinking becomes part of the individual's personality as adulthood approaches.

Studies of identical (or monozygotic) and fraternal (or dizygotic) twins suggest that genetic factors may also play an important role in causing the disorder. Twin studies indicate that genes contribute to the development of childhood personality disorders, including PPD. Furthermore, estimates of the degree of genetic contribution to the development of childhood personality disorders are similar to estimates of the genetic contribution to adult versions of the disorders.

Symptoms

A core symptom of PPD is a generalized distrust of other people. Comments and actions that healthy people would not notice come across as full of insults and threats to someone with the disorder. Yet, generally, patients with PPD remain in touch with reality; they don't have any of the **hallucinations** or **delusions** seen in patients with psychoses. Nevertheless, their suspicions that others are intent on harming or exploiting them are so pervasive and intense that people with PPD often become very isolated. They avoid normal social interactions. And because they feel so insecure in what is a very threatening world for them, patients with PPD are capable of becoming violent. Innocuous comments, harmless jokes and other day-to-day communications are often perceived as insults.

Paranoid suspicions carry over into all realms of life. Those burdened with PPD are frequently convinced that their sexual partners are unfaithful. They may misinterpret compliments offered by employers or coworkers as hidden criticisms or attempts to get them to work harder. Complimenting a person with PPD on their clothing or car, for example, could easily be taken as an attack on their materialism or selfishness.

Because they persistently question the motivations and trustworthiness of others, patients with PPD are not inclined to share intimacies. They fear such information might be used against them. As a result, they become hostile and unfriendly, argumentative or aloof. Their unpleasantness often draws negative responses from those around them. These rebuffs become "proof" in the patient's mind that others are, indeed, hostile to them. They have little insight into the effects of their attitude and behavior on their generally unsuccessful interactions with others. Asked if they might be responsible for negative interactions that fill their lives, people with PPD are likely to place all the blame on others.

A brief summary of the typical symptoms of PPD includes:

- suspiciousness and distrust of others
- questioning hidden motives in others
- feelings of certainty, without justification or proof, that others are intent on harming or exploiting them
- social isolation
- aggressiveness and hostility
- little or no sense of humor

Demographics

As of 2002, it has not been possible to determine the number of people with PPD with any accuracy. This lack of data might be expected for a disorder that is characterized by extreme suspiciousness. Such patients in many cases avoid voluntary contact with such people as mental health workers who have a certain amount of power over them. There are, nonetheless, some estimates of the prevalence of PPD. According to the *DSM-IV-TR*, between 0.5% and 2.5% of the general population of the United States may have PPD, while 2%–10% of outpatients receiving psychiatric care may be affected. A significant percentage of institutionalized psychiatric patients, between 10% and 30%, might have symptoms that qualify for a **diagnosis** of PPD. Finally, the disorder appears to be more common in men than in women.

There are indications in the scientific literature that relatives of patients with chronic schizophrenia may have a greater chance of developing PPD than people in the general population. Also, the incidence of the disorder may be higher among relatives of patients suffering from another psychotic disorder known as delusional disorder of the persecutory type.

Diagnosis

There are no laboratory tests or **imaging studies** as of 2002 that can be used to confirm a diagnosis of PPD. The diagnosis is usually made on the basis of the doctor's interview with the patient, although the doctor may also give

the patient a diagnostic questionnaire. In addition, input from people who know the patient may be requested.

Diagnostic criteria

Mental health care providers look for at least five distinguishing symptoms in patients who they think might suffer from PPD. The first is a pattern of suspiciousness about, and distrust of, other people when there is no good reason for either. This pattern should be present from at least the time of the patient's early adulthood.

In addition to this symptom that is required in order to make the PPD diagnosis, the patient should have at least four of the following seven symptoms as listed in the *DSM-IV-TR*:

- The unfounded suspicion that people want to deceive, exploit or harm the patient.
- The pervasive belief that others are not worthy of trust or that they are not inclined to or capable of offering loyalty.
- A fear that others will use information against the patient with the intention of harming him or her. This fear is demonstrated by a reluctance to share even harmless personal information with others.
- The interpretation of others' innocent remarks as insulting or demeaning; or the interpretation of neutral events as presenting or conveying a threat.
- A strong tendency not to forgive real or imagined slights and insults. People with PPD nurture grudges for a long time.
- An angry and aggressive response in reply to imagined attacks by others. The counterattack for a perceived insult is often rapid.
- Suspicions, in the absence of any real evidence, that a spouse or sexual partner is not sexually faithful, resulting in such repeated questions as "Where have you been?" "Whom did you see?" etc., and other types of jealous behavior.

Differential diagnosis

Psychiatrists and clinical psychologists should be careful not to confuse PPD with other mental disorders or behaviors that have some symptoms in common with the paranoid personality. For example, it is important to make sure that the patient is not a long-term user of amphetamine or cocaine. Chronic abuse of these stimulants can produce paranoid behavior. Also, some prescription medications might produce **paranoia** as a side effect; so it is important to find out what drugs, if any, the patient is taking.

There are other conditions that, if present, would mean a patient with paranoid traits does not have PPD. For example, if the patient has symptoms of schizophrenia, hallucinations or a formal thought disorder, a diagnosis of PPD can't be made. The same is true of delusions, which are not a feature of PPD.

Also, the suspiciousness and other characteristic features of PPD must have been present in the patient for a long time, at least since early adulthood. If the symptoms appeared more recently than that, a person can't be given a diagnosis of this disorder.

There are at least a dozen disorders or other mental health conditions listed in the *DSM-IV-TR* that could be confused with PPD after a superficial interview because they share similar or identical symptoms with PPD. It is important, therefore, to eliminate the following entities before settling on a diagnosis of PPD: paranoid schizophrenia; **schizotypal personality disorder**; **schizoid personality disorder**; persecutory delusional disorder; mood disorder with psychotic features; symptoms and/or personality changes produced by disease, medical conditions, medication or drugs of abuse; paranoia linked to the development of physical handicaps; and borderline, histrionic, avoidant, antisocial or narcissistic personality disorders.

In some individuals, symptoms of PPD may precede the development of schizophrenia. Should a patient who as been correctly diagnosed with PPD later develop schizophrenia, the *DSM-IV-TR* suggests that the diagnosis on the patient's medical record be changed from "Paranoid Personality Disorder" to "Paranoid Personality Disorder (Premorbid)."

Treatments

Because they are suspicious and untrusting, patients with PPD are not likely to seek therapy on their own. A particularly disturbing development or life crisis may prompt them to get help. More often, however, the legal system or the patient's relatives order or encourage him or her to seek professional treatment. But even after a patient finally agrees or is forced to seek treatment, the nature of the disorder poses very serious challenges to therapists.

Psychotherapy

The primary approach to treatment for such personality disorders as PPD is **psychotherapy**. The problem is that patients with PPD do not readily offer therapists the trust that is needed for successful treatment. As a result, it has been difficult to gather data that would indicate what kind of psychotherapy would work best. Therapists face the challenge of developing rapport with someone

who is, by the nature of his personality disorder, distrustful and suspicious; someone who often sees malicious intent in the innocuous actions and statements of others. The patient may actively resist or refuse to cooperate with others who are trying to help.

Mental health workers treating patients with PPD must guard against any show of hostility on their part in response to hostility from the patient, which is a common occurrence in people with this disorder. Instead, clinicians are advised to develop trust by persistently demonstrating a nonjudgmental attitude and a professional desire to assist the patient.

It is usually up to the therapist alone to overcome a patient's resistance. **Group therapy** that includes family members or other psychiatric patients, not surprisingly, isn't useful in the treatment of PPD due to the mistrust people with PPD feel towards others. This characteristic also explains why there are no significant **self-help groups** dedicated to recovery from this disorder. It has been suggested, however, that some people with PPD might join cults or extremist groups whose members might share their suspicions.

To gain the trust of PPD patients, therapists must be careful to hide as little as possible from their patients. This transparency should include note taking; details of administrative tasks concerning the patient; correspondence; and medications. Any indication of what the patient would consider "deception" or covert operation can, and often does, lead the patient to drop out of treatment. Patients with paranoid tendencies often don't have a well-developed sense of humor; those who must interact with people with PPD probably should not make jokes in their presence. Attempts at humor may seem like ridicule to people who feel so easily threatened.

With some patients, the most attainable goal may be to help them to learn to analyze their problems in dealing with other people. This approach amounts to supportive therapy and is preferable to psychotherapeutic approaches that attempt to analyze the patient's motivations and possible sources of paranoid traits. Asking about a patient's past can undermine the treatment of PPD patients. Concentrating on the specific issues that are troubling the patient with PPD is usually the wisest course.

With time and a skilled therapist, the patient with PPD who remains in therapy may develop a measure of trust. But as the patient reveals more of his paranoid thoughts, the clinician will continue to face the difficult task of balancing the need for objectivity about the paranoid ideas and the maintenance of a good rapport with the patient. The therapist thus walks a tightrope with this type of patient. If the therapist is not straightforward enough, the patient may feel deceived. If the therapist challenges paranoid thoughts too directly, the patient will be threatened and probably drop out of treatment.

Medications

While individual supportive psychotherapy is the treatment of choice for PPD, medications are sometimes used on a limited basis to treat related symptoms. If, for example, the patient is very anxious, anti-anxiety drugs may be prescribed. In addition, during periods of extreme agitation and high stress that produce delusional states, the patient may be given low doses of antipsychotic medications.

Some clinicians have suggested that low doses of neuroleptics should be used in this group of patients; however, medications are not normally part of long-term treatment for PPD. One reason is that no medication has been proven to relieve effectively the long-term symptoms of the disorder, although the selective serotonin reuptake inhibitors such as **fluoxetine** (Prozac) have been reported to make patients less angry, irritable and suspicious. Antidepressants may even make symptoms worse. A second reason is that people with PPD are suspicious of medications. They fear that others might try to control them through the use of drugs. It can therefore be very difficult to persuade them to take medications unless the potential for relief from another threat, such as extreme anxiety, makes the medications seem relatively appealing. The best use of medication may be for specific complaints, when the patient trusts the therapist enough to ask for relief from particular symptoms.

Prognosis

Paranoid personality disorder is often a chronic, lifelong condition; the long-term prognosis is usually not encouraging. Feelings of paranoia, however, can be controlled to a degree with successful therapy. Unfortunately, many patients suffer the major symptoms of the disorder throughout their lives.

Prevention

With little or no understanding of the cause of PPD, it is not possible to prevent the disorder.

See also Paranoia

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Paraphilias

Definition

Paraphilias are sexual feelings or behaviors that may involve sexual partners that are not human, not consenting, or that involve suffering by one or both partners.

Description

According to the ***Diagnostic and Statistical Manual of Mental Disorders*** *(known as the* DSM) fourth edition text revised (*DSM-IV-TR*), the manual used by mental health professionals to diagnose mental disorders, it is not uncommon for an individual to have more than one paraphilia. The *DSM-IV-TR* lists the following paraphilias: **exhibitionism**, **fetishism**, **frotteurism**, **pedophilia**, **sexual masochism**, **sexual sadism**, **transvestic fetishism**, and **voyeurism**. The *DSM-IV-TR* also includes a category for paraphilia not otherwise specified, which is the category for the less common paraphilias, including necrophilia, zoophilia, and others.

Exhibitionism

Exhibitionism is the exposure of genitals to a non-consenting stranger. In some cases, the individual may also engage in autoeroticism while exposing himself. Generally, no additional contact with the observer is sought; the individual is stimulated sexually by gaining the attention of and startling the observer.

Fetishism

People with this disorder achieve sexual gratification with the use of objects, most commonly women's undergarments, shoes, stockings, or other clothing items.

Frotteurism

Individuals with this disorder are gratified by touching or rubbing a non-consenting person. This behavior often occurs in busy, crowded places, such as on busy streets or on crowded buses or subways.

Pedophilia

Pedophilia involves sexual activity with a child, generally under age 13. The *DSM-IV-TR* describes a criterion that the individual with pedophilia be over 16 years of age and be at least five years older than the child. Individuals with this disorder may be attracted to either males or females or both, although incidents of pedophilic activity are almost twice as likely to be repeated by those individuals attracted to males. Individuals with this disorder develop procedures and strategies for gaining access to and trust of children.

Sexual masochism

Masochism is a term applied to a specific sexual disorder but which also has a broader usage. The sexual disorder involves pleasure and excitement produced by pain, either inflicted by others or by oneself. It usually

begins in childhood or adolescence and is chronic. An individual with this disorder achieves gratification by experiencing pain. Masochism is the only paraphilia in which any noticeable number of women participate—about 5% of masochists are female. The term comes from the name of a nineteenth-century Austrian writer, Leopold von Sacher-Masoch, whose novels often included characters who were obsessed with the combination of sex and pain.

In the broader sense, masochism refers to any experience of receiving pleasure or satisfaction from suffering pain. The psychoanalytic view is that masochism is aggression turned inward, onto the self, when a person feels too guilty or is afraid to express it outwardly.

Sexual sadism

A sadistic individual achieves sexual gratification by inflicting pain on another person.

In psychoanalytic theory, sadism is related to the fear of castration, while the behaviorist explanation of sadomasochism (the deviant sexual practice combining sadism and masochism) is that its constituent feelings are physiologically similar to sexual arousal. Separate but parallel descriptions are given for sexual sadism and sexual masochism in the *DSM-IV-TR*. The clinical diagnostic criteria for both are recurrence of the behavior over a period of at least six months, and significant distress or impairment of the ability to function as a result of the behavior or associated urges or fantasies. Either type of behavior may be limited to fantasies (sometimes while one is engaged in outwardly nondeviant sex) or acted out with a consenting partner, a non-consenting partner, or in the case of masochism, alone. Sadomasochism occurs in both males and females, and in both heterosexual and homosexual relationships.

Transvestic fetishism

This disorder is characterized by heterosexual males who dress in women's clothing to achieve a sexual response. The activity may begin in adolescence, and in secret; later, as an adult, the man may dress as a woman completely and in public. Not all men who cross-dress are unhappy with their gender, but some are. In a small minority of men with transvestic fetishism, gender dysphoria (unhappiness with original gender) may emerge, and those men may eventually seek hormonal treatments or surgical sex reassignment to enable them to live permanently as women.

Voyeurism

Voyeurism is a paraphilia in which a person finds sexual excitement in watching unsuspecting people who are nude, undressing, or having sex. Voyeurs are almost always male, and the victims are usually strangers. A voyeur may fantasize about having sex with the victim but almost never actually pursues this. The voyeur may return to watch the same stranger repeatedly, but there is rarely any physical contact.

Voyeurs are popularly known as "peeping Toms," based on the eleventh-century legend of Lady Godiva. According to the story, Tom was a tailor who "peeped" at Lady Godiva as she rode naked through the streets of Coventry, England, in a sacrificial act to get her husband to lower taxes. Tom was struck with blindness for not looking away like everyone else.

Uncommon paraphilias

BESTIALITY. Bestiality is a term that describes sexual feelings or behaviors involving animals. Termed zoophilia by *DSM-IV* this is an uncommon disorder. The disorder does not specify an animal or category of animals; the person with zoophilia may focus sexual feelings on domesticated animals, such as dogs, or farm animals, such as sheep or goats.

NECROPHILIA. Necrophilia is a term that describes sexual feelings or behaviors involving corpses.

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Parent management training

Definition

Parent management training (PMT) is an adjunct to treatment that involves educating and coaching parents to change their child's problem behaviors using principles of learning theory and **behavior modification**.

Purpose

The aim of PMT is to decrease or eliminate a child's disruptive or inappropriate behaviors at home or school and to replace problematic ways of acting with positive interactions with peers, parents and such authority figures as teachers. In order to accomplish this goal, PMT focuses on enhancing parenting skills. The PMT therapist coaches parents in applying such strategies as rewarding positive behavior, and responding to negative behavior by removing rewards or enforcing undesirable consequences (punishments). Although PMT focuses on specific targeted behaviors rather than on the child's **diagnosis** as such, it has come to be associated with the treatment of certain disorders. PMT is used in treating **oppositional defiant disorder**, **conduct disorder**, **intermittent explosive disorder** (age-inappropriate tantrums), and attention deficit disorder with hyperactivity (**attention-deficit/hyperactivity disorder**). Such antisocial behaviors as firesetting and truancy can also be addressed through PMT.

KEY TERMS

Behavior modification—An approach to therapy based on the principles of operant conditioning. Behavior modification seeks to replace undesirable behaviors with preferable behaviors through the use of positive or negative reinforcement.

Positive reinforcement—A procedure or response that rewards a desired behavior.

Response-contingent—An approach to treatment in which rewards or punishments are given in response to a particular behavior to be encouraged or corrected.

Social learning theory—A subset of learning theories based on the concept that human behavior originates in and is affected by the interplay among the person's learned experiences, previous behaviors, and environmental influences.

Description

In PMT, the therapist conducts initial teaching sessions with the parent(s), giving a short summary of foundational concepts in behavior modification; demonstrating interventions for the parents; and coaching parents in carrying out the techniques of PMT. Early meetings with the therapist focus on training in the principles of behavior modification, response-contingent learning, and ways to apply the techniques. Parents are instructed to define the behavior(s) to be changed concretely and specifically. In addition, they learn how to observe and identify relevant behavior and situational factors, and how to chart or otherwise record the child's behavior. Defining, observing and recording behavior are essential to the success of this method, because when such behaviors as fighting or tantrums are highlighted in concrete, specific ways, techniques of **reinforcement** and punishment can be put to use. Progress or its absence is easier to identify when the description of the behavior is defined with enough clarity to be measurable, and when responses to the PMT interventions are tracked on a chart. After the child's parents grasp the basic interventions as well as when and how to apply them, the techniques that the parents practiced with the therapist can be carried out at home.

Learning theory, which is the conceptual foundation of PMT, deals with the ways in which organisms learn to respond to their environment, and the factors that affect the frequency of a specific behavior. The core of learning theory is the notion that actions increase or decrease in frequency in response to the consequences that occur immediately after the action. Research in parent-child interactions in families with disruptive, difficult or defiant children shows that parental responses are unintentionally reinforcing the unwanted behavior. PMT trains parents to become more careful in their reactions to a child's behavior. The parents learn to be more discerning: to provide attention, praise and increased affection in reaction to the child's behaving in desired ways; and to withdraw attention, to suspend displays of affection, or to withdraw privileges in instances of less desirable behavior.

The most critical element of PMT is offering positive reinforcement for socially appropriate (or at least nondeviant) behaviors. An additional component involves responding to any undesired behaviors by removing rewards or applying punishment. These two types of response to the child must be carried out with great consistency. Consistent responding is important because erratic responses to unwanted behavior can actually cause the behavior to increase in frequency. For instance, if a child consistently throws tantrums in stores, hoping to be given something to end the tantrum, inconsistent parent responses can worsen the situation. If a parent is occasionally determined not to give in, but provides a candy bar or a toy to end the tantrum on other occasions, the child learns either to have more tantrums, or to have more dramatic tantrums. The rise in the number or intensity of tantrums occurs because the child is trying to increase the number of opportunities to obtain that infrequent parental reward for the behavior. Planning responses ahead of time to predefined target behaviors by rewarding desired actions and by withdrawing rewards or applying punishment for undesirable behavior is a funda-

mental principle of PMT. Consistent consequences, which are contingent on (in response to) the child's behavior, result in behavior change. Parents practice therapeutic ways of responding to their child's behavior in the PMT sessions with the therapist.

Through PMT, parents learn that positive rewards for appropriate behaviors can be offered in a variety of ways. Giving praise, providing extra attention, earning points toward obtaining a reward desired by the child, earning stickers or other small indicators of positive behavior, earning additional privileges, hugging (and other affectionate gestures) are all forms of reward. The technical term for the rewarding of desired behavior is *positive reinforcement*. Positive reinforcement refers to consequences that cause the desired target behavior to increase.

PMT instructs parents to cancel rewards or give punishments when the child behaves in undesirable ways. The removal of rewards usually entails time away from the circumstances and situations in which the child can do desired activities or receive attention. The concept of a "time out" is based on this notion of removal of rewards. Time out from rewards customarily means that the child is removed from people and stimulation for a certain period of time; it can also include deprivation of privileges.

Punishment in PMT is not necessarily what parents typically refer to as punishment; it most emphatically is *not* the use of physical punishment. A punishment in PMT involves a response to the child's negative behavior by exposing the child to something he or she regards as unpleasant. Examples of punishments might include having to redo the correct behavior so many times that it becomes annoying; verbal reproaches; or the military standby—"drop and give me fifty"—having to do pushups or situps or laps around a playing field to the point of discomfort.

The least challenging problems, which have the greatest likelihood of successful change, are tackled first, in hope of giving the family a "success experience." The success experience is a positive reinforcement for the family, increasing the likelihood that they will continue using PMT in efforts to bring about change. In addition, lower-level behavioral problems provide opportunities for parents to become skilled in intervening and to learn consistency in their responses. After the parents have practiced using the skills learned in PMT on the less important problems, more severe issues can be tackled.

In addition to face-to-face sessions with the parents, some PMT therapists make frequent telephone calls to the parents between sessions. The purposes of the calls are to remind parents to continue to be consistent in applying the techniques; to answer questions about the work at home; and to praise the parents' attempts to correct the child's behavior. In addition, ongoing support in sessions and on the telephone helps parents feel less isolated and thus more likely to continue trying to use learning principles in managing their child. Troubleshooting any problems that arise regarding the application of the behavioral techniques is handled over the telephone and in the office sessions.

An additional aspect of learning theory is that rewarding subunits of the ultimately desired behavior can lead to developing more complex new actions. The subunits are finally linked together by changing the ways in which the rewards are given. This process is called "chaining." Sometimes, if the child shows no elements of the desired response, then the desired behavior is demonstrated for the child and subsequent "near hits" or approximations are rewarded. To refine "close but not quite" into the targeted response, rewards are given in a slightly "pickier" manner. Rewarding successive approximations of the desired behavior is also called "shaping."

Risks

The best way to learn to alter parental responses to child behaviors is with the support and assistance of a behavioral health professional (**psychologist**, **psychiatrist**, clinical social worker). As noted earlier, parents often inadvertently reinforce the problem behaviors, and it is difficult for a parent to see objectively the ways in which he or she is unintentionally supporting the defiant or difficult behavior. Furthermore, inappropriate application of such behavioral techniques as those used in PMT can actually make the problem situation worse. Families should seek therapists with valid credentials, skills, training and experience in PMT.

Normal results

Typically, the parents should notice a decrease in the unwanted behaviors after they implement the techniques learned in PMT at home. Of the various therapies used to treat childhood disorders, PMT is among those most frequently researched. PMT has shown effectiveness in changing children's behavior in very well-designed and rigorous studies. PMT has a greater effect on behavior than many other treatments, including **family therapy** or **play therapy**. Furthermore, the results— improved child behavior and reduction or elimination of undesirable behavior— are sustained over the long term. When a group of children whose families had used PMT were examined one to fourteen years later, they had maintained higher rates of positive behavior and lower levels of problem behavior.

See also: Pyromania

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Association for the Advancement of Behavior Therapy. 305 Seventh Avenue, 16th Floor, New York, NY 10001-60008. Telephone: (212) 647-1890. Web site: <www.aabt.org>.

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Parents & Teachers of Explosive Kids. <www.explosivekids.org>.

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Parnate *see* **Tranylcypromine**

Paroxetine

Definition

Paroxetine is an antidepressant of the type known as selective serotonin reuptake inhibitors (SSRI). It is sold in the United States under the brand name Paxil.

KEY TERMS

Bioavailability—Medication that is available in the body. If the bioavailability of a drug is increased, more is available to the body for use, and if it is decreased, less is available for use.

Bipolar syndrome—An abnormal mental condition characterized by periods of intense elation, energy and activity followed by periods of inactivity and depression; formerly called manic-depression.

Hyponatremia—A condition characterized by an abnormally low concentration of sodium in the blood.

Manic—Referring to mania, a state characterized by excessive activity, excitement or emotion.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Tryptophan—An essential amino acid released from proteins during the process of digestion. Tryptophan is an important ingredient in the body's production of serotonin.

Purpose

Paroxetine is approved by the United States Food and Drug Administration (FDA) for treatment of depression and for the following anxiety disorders: **obsessive-compulsive disorder**, **panic disorder**, **generalized anxiety disorder**, **post-traumatic stress disorder**, and social anxiety disorder.

Description

Paroxetine increases the amount of serotonin (also called 5-HT) available in the **brain**. Serotonin is a neurotransmitter, or chemical in the brain that carries nerve impulses from a sending neuron (nerve cell) to a receiving neuron. The sending neuron releases serotonin into a little gap between neurons, called the synapse. The receiving neuron picks up the serotonin from the synapse, allowing the nerve impulse to continue on its way.

Researchers think that depression and certain other disorders may be caused, in part, because there is not enough available serotonin in the brain. Normally, once a nerve impulse has crossed the synapse, serotonin is reabsorbed by the sending neuron that released it. Once reabsorbed, this serotonin is no longer available and cannot interact with a receiving neuron. Paroxetine blocks the

reabsorption, or re-uptake, of serotonin, leaving it available to stimulate receiving neurons. Therefore, paroxetine facilitates the transmission of nerve impulses by increasing available serotonin in the brain and thus increasing its effectiveness.

Paroxetine is an antidepressant that is virtually completely absorbed via oral administration. Food does not reduce its absorption.

The benefits of paroxetine develop slowly over a period of up to four weeks. Patients should be aware of this and continue to take the drug as directed, even if they feel no immediate improvement.

Recommended dosage

The recommended dosage of paroxetine is 20–50 mg per day. The drug should be taken only once per day. An appropriate initial dosage is 20 mg. Dosage changes should not be made more frequently than once per week.

The recommended dosage for older persons or individuals with liver or kidney disease is 10 mg per day. The total dosage for such persons should not exceed 40 mg per day.

Precautions

Paroxetine should never be taken with monoamine oxidase inhibitors (MAOIs)(see interactions below).

Paroxetine may lower the threshold for a **manic episode** among people with bipolar (manic-depressive) disorders. For this reason, the drug should be used only with caution and under close supervision in these patients. It may also increase the change of having a seizure in people with a history of seizure disorders.

The possibility of **suicide** is a component of depression. The minimum number of doses of paroxetine should be dispensed at any one time to minimize the potential for use as a suicide agent.

Hyponatremia (abnormally low concentration of sodium in the blood) has been associated with the use of paroxetine. In all cases, this condition resolved when the drug was discontinued. Most of these instances occurred among older individuals who were also taking diuretics (water pills).

Side effects

Common side effects associated with paroxetine include headache, weakness, chills, malaise, nausea, and sleepiness. Other complaints included dry mouth, dizziness, tremors, constipation, diarrhea, and problems with ejaculation. Adverse reactions to paroxetine have been reported for all organ systems of the body, but all of these side effects are uncommon.

In general, the incidence of side effects increases as the dosage of paroxetine increases.

Interactions

There is the potential for a fatal interaction with another class of antidepressant drugs called monoamine-oxidase (MAO) inhibitors. There have been reports of dangerously elevated body temperature, muscle rigidity, and rapid changes in vital signs such as heart rate and blood pressure. Mental changes ranging from extreme agitation to **delirium** and coma have also been reported. Because of this, paroxetine should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting paroxetine or any other antidepressant. The same holds true when discontinuing paroxetine and starting an MAO inhibitor.

The combination of paroxetine with the antipsychotic drug **thioridazine** has the potential to cause fatal cardiac arrhythmias (irregular heartbeat). The use of paroxetine in combination with tryptophan may result in unwanted reactions including agitation, restlessness, and gastrointestinal distress. Paroxetine may also increase the change of having a seizure in people with a history of seizure disorders. People taking anticonvulsants to control **seizures** should be closely monitored and a physician may need to adjust the dosage of their seizure medication.

People with **bipolar disorder** are commonly treated with lithium. No interactions between paroxetine and lithium have been reported, nor have are there any reported interactions with the common anti-anxiety drug **diazepam** (Valium).

Phenobarbital at dosages greater than 100 mg per day decreases the bioavailability of paroxetine in some persons. Paroxetine has been reported to increase the systemic bioavailability of procyclidine.

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American Academy of Clinical Toxicology. 777 East Park Drive, PO Box 8820, Harrisburg, PA 17105-8820. Telephone: (717) 558-7750. Fax: (717) 558-7845. Web site: <http://www.clintox.org/index.html>.

American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

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American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

L. Fleming Fallon, Jr., M.D., Dr.P.H.

KEY TERMS

Antispasmodic—A medication or preparation given to relieve muscle or digestive cramps.

Anxiolytic—A preparation or substance given to relieve anxiety; a tranquilizer.

Chrysin—A flavonoid found in passionflower that may be the source of its anxiolytic properties.

Flavonoids—Plant pigments that have a variety of effects on human physiology. Some of these pigments have anti-inflammatory, anti-carcinogenic, and antioxidant effects, for example.

Gastritis—Inflammation of the lining of the stomach.

Infusion—The most potent form of extraction of a herb into water. Infusions are steeped for a longer period of time than teas.

Tincture—An alcohol-based herbal extract prepared by soaking parts of the plant in a mixture of alcohol and water. Established ratios and dilutions are followed.

Topical—A type of medication or preparation intended for use on the skin or external suface of the body.

Passionflower

Definition

Passionflower (*Passiflora incarnata*) is a vine whose leaves and flowers are widely used in Europe to make a herbal remedy for anxiety and **insomnia**. The plant, which is native to the tropical regions of North America, was first used by the Aztecs of Mexico as a folk remedy for these conditions. Passionflower is also known as maypop, apricot vine, passion vine, and granadilla. It grows as much as 30 ft (10 m) tall, with a thick, woody stem.

Passionflower received its name from the sixteenth-century conquistadors who claimed Mexico for the Spanish Empire. The priests and soldiers who accompanied Hernando Cortez thought that the whitish-purple flowers of the vine symbolized certain features of the passion of Christ. The corona in the center of the flower reminded them of Christ's crown of thorns, the five stamens of the number of Christ's wounds, and the tendrils of the whips that were used to scourge Christ.

Purpose

Passionflower is still used as a sedative and anxiolytic, although far more frequently in Great Britain and Europe than in the United States. In Britain, passionflower is the single most common ingredient in herbal sedatives, and the German Commission E approved it for use as a tranquilizer. It is also used in homeopathic remedies. In addition to its long-standing uses as a remedy for anxiety and insomnia, passionflower has also been recommended for the treatment of gastrointestinal disorders related to anxiety; asthma; tachycardia (an abnormally rapid heartbeat); menstrual cramps; **seizures**; **attention-deficit/hyperactivity disorder**; and hysteria. A topical

preparation made from passionflower has been used to treat hemorrhoids.

The parts of the plant that grow above the ground are gathered to make passionflower preparations. They may be used either fresh or dried. The most common sources of the passionflower that is used today are India, the West Indies, and the southern United States, even though the vine can also be grown in Mexico and Latin America.

Description

Passionflower preparations may be made from the flowers, leaves, or shoots of the plant. After the first fruits of the plant have matured, younger shoots growing 12.7–17.8 cm. above the ground are harvested and air-dried. The plant material is then used to prepare infusions, teas, liquid extracts, and tinctures of passionflower. In Europe, passionflower is often combined with lemon balm or **valerian** to make a sedative tea. The standardized formula approved by the German Commission E contains 30% passionflower, 40% valerian root, and 30% lemon balm. Passionflower is also used to make a special sedative tea for children, which typically includes 30% passionflower, 30% lemon balm, 30% **lavender** flower, and 10% **St. John's wort**. Passionflower is sometimes combined with hawthorn to make a remedy for stomach cramps associated with gastritis.

Although passionflower has been shown in animal studies to have sedative and antispasmodic effects, researchers are not yet certain which compounds in the plant have these properties. Passionflower is known to contain flavonoids and a group of alkaloid compounds that include harman, harmine, harmaline, and harmalol. Some researchers have hypothesized that the medicinal effects of passionflower derive from a combination of these substances rather than from any of them in isolation. A recent Swiss study, however, appears to indicate that a flavonoid called chrysin may be the source of passionflower's anxiolytic properties.

Recommended dosage

As the German recipe indicates, passionflower is considered safe for children. Dosages for children should be calculated on the basis of the child's weight. Since most adult dosages of herbal remedies assume an average adult weight of 150 lb (70 kg), a child weighing 50 lb (23 kg) can be given 1/3 of the adult dose.

Recommended adult doses of passionflower are as follows:

- Infusion: 2–5 g of dried herb, up to three times daily
- Fluid extract (1:1 ratio in a solution of 25% alcohol): 0.5–1.0 mL up to three times daily
- Tincture (1:5 ratio in a solution of 45% alcohol): 0.5–2.0 mL up to three times daily.

Precautions

Passionflower should not be used in doses higher than the recommended levels. Because it has a sedative effect, it should not be combined with alcoholic beverages or prescription sedatives. Passionflower should not be used by pregnant or lactating women, or for children under six months old.

Side effects

As of 2002, passionflower has not been reported to cause any significant side effects when taken at recommended dosage levels.

Interactions

The alkaloids found in passionflower, especially harman and harmaline, may increase the effects of a class of prescription antidepressants called monoamine oxidase inhibitors (MAOIs). These drugs are most often prescribed for depression, panic attacks, and eating disorders. Passionflower may also increase the effects of over-the-counter sedatives as well as prescription sedatives.

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American Botanical Council. PO Box 144345. Austin, TX 78714-4345. <www.herbalgram.org>.

Herb Research Foundation. 1007 Pearl Street. Suite 200. Boulder, CO 80302. <www.herbs.org>.

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Pathological gambling disorder

Definition

Pathological gambling disorder occurs when a person gambles compulsively to such an extent that the wagering has a severe negative effect on his or her job, relationships, mental health, or other important aspects of life. The person may continue to gamble even after they have developed social, economic, interpersonal, or legal problems as a result of the gambling.

Description

Pathological gambling disorder is characterized by uncontrollable gambling well beyond the point of a social or recreational activity, such that the gambling has a major disruptive effect on the gambler's life. People who are pathological gamblers may lose their life savings, and may even commit crimes (stealing, embezzling, or forging checks) to get money for their "habit." Relationships and jobs may also be lost as a result of the disorder.

Pathological gambling disorder is an example of a process, or behavioral, **addiction**, as distinct from an addiction to such substances as food, drugs, tobacco, or alcohol. In process addictions, the characteristic "rush" or "high" comes from the series of steps or actions that are involved in the addictive behavior. With gambling, the "high" may be stimulated by the social atmosphere or group setting of the casino, race track, or bingo hall as well as by the excitement of risk-taking. Some gamblers have a "lucky" outfit, item of clothing, or accessory that they wear or take along when gambling; sometimes putting on the outfit or item in question is enough to start the "rush."

People with pathological gambling disorder may engage in many different types of gambling activities. These may include games of chance that are found in casinos, such as slot machines, card games, and roulette. Many of these games are now available on the Internet, the chief difference being that the bettor uses a credit card instead of cash or chips. Other gambling activities may include the state lottery, horse or dog racing, or even bingo. The person may place bets on the outcome of an election, baseball or football games, or even the weather on a particular day. Pathological gambling usually develops slowly over time; people tend to begin with acceptable levels of social or recreational gambling and slowly progress to pathological gambling. In most cases the disorder develops slowly over a period of years; however, there are cases of patients who gambled socially for decades and then began to gamble compulsively under the impact of a major life stressor, such as divorce or being laid off from work.

KEY TERMS

Aversion therapy—An approach to treatment in which an unpleasant or painful stimulus is linked to an undesirable behavior in order to condition the patient to dislike or avoid the behavior.

Chasing—Betting larger and larger sums of money, or taking greater risks, in order to make up for money previously lost in gambling.

Denial—A psychological defense mechanism that reduces anxiety by excluding recognition of an addiction or similar problem from the conscious mind.

Process addiction—An addiction to a mood-altering behavior or series of behaviors rather than a substance.

Reinforcement—A term that refers to the ability of a drug, substance, or behavior to produce effects that will make the user want to take the substance or perform the behavior again.

Rush—The initial intensely pleasurable sensation experienced from injecting a narcotic or stimulant drug. The term has also been applied to the feeling of excitement experienced from the behaviors involved in process addictions.

Causes and symptoms

Causes

There are no known biological causes of pathological gambling disorder. Some studies have found interesting differences between compulsive gamblers and the general population on the biological level, but none that are thought to be an actual cause of pathological gambling. Many people, however, have significant psychological causes for excessive gambling. They may use gambling as an emotional escape from depression; this pattern appears more often in females with the disorder than in males. Some people who are pathologic gamblers are seeking the mood alteration associated with gambling— specifically the excitement and energy that they find in the activity— more than the money involved. In other words, the person with the disorder is reinforced by an emotional "high" rather than by the money itself. Some researchers have found that males diagnosed with pathological gambling disorder were more likely to have

Pathological gambling disorder is characterized by uncontrollable gambling well beyond the point of a social or recreational activity, such that the gambling has a major disruptive effect on the gambler's life. People who are pathological gamblers may lose their life savings, and may even commit crimes to get money for their "habit." *(AP/Wide World Photos, Inc. Reproduced by permission.)*

been diagnosed with **attention-deficit/hyperactivity disorder** as children than males in the general population. Other researchers have described compulsive gamblers in general as highly competitive people who are restless and easily bored.

Other theories about the causes of pathological gambling emphasize cognitive distortions rather than mood problems. Pathological gambling has been associated with dysfunctional thinking patterns; many people with this disorder are highly superstitious or believe that they can control the outcome of events when they are gambling. Many people diagnosed with the disorder also have distorted beliefs about money, tending to see it at the same time as the source of all their problems and the answer to all their problems. Patients diagnosed with pathological gambling disorder have an increased risk of either having or developing histrionic, narcissistic, or **borderline personality disorder**.

One social change that has been linked with the rise in the number of adults diagnosed with pathological gambling disorder in the United States is the increased availability of legalized gambling.

Symptoms

The symptoms of pathological gambling include preoccupation with gambling activity, often to the extent of interfering with the person's occupational or social functioning. The person is often unable to control the gambling behavior, continuing to place bets or go to casinos in spite of attempts to cut back or stop. A common behavior in persons with pathological gambling disorder is "chasing," which refers to betting larger sums of money or taking greater risks in order to undo or make up for previous losses. The person may also lie about their gambling or engage in such antisocial behaviors as stealing, credit card fraud, check forgery, embezzling from an employer, or similar dishonest behaviors in order to obtain more money for gambling.

Demographics

More males than females in the United States are diagnosed with pathological gambling disorder; the sex ratio is thought to be about 2:1. Relatively few women, however, are in treatment programs for the disorder, most probably because of the greater social **stigma** attached to women who gamble. As a rule, men diagnosed with pathological gambling disorder began gambling as teenagers, whereas women tend to start compulsive gambling at a later age. Pathological gambling disorder tends to be more common in minority groups and in people with lower socioeconomic status. About 25% of people diagnosed as pathological gamblers had a parent with the disorder. People who smoke tobacco or abuse alcohol are more likely to have pathological gambling disorder than people who do not use these substances.

As many as 4% of the general population in the United States may meet criteria for pathological gambling disorder at some point in their lives. In some countries such as Australia the number is thought to be as high as 7%.

Diagnosis

Pathological gambling disorder is more likely to be diagnosed when the affected person's spouse or family becomes concerned than to be self-reported. **Denial** is common among persons with the disorder. The professional handbook, the ***Diagnostic and Statistical Manual of Mental Disorders****, fourth edition, text revision, or* DSM-IV-TR, specifies that the patient must have at least five of the following symptoms to meet criteria for the disorder:

- thinks about gambling all the time
- uses larger and larger amounts of money when gambling
- has tried to stop gambling but failed
- is moody or cranky when trying to stop gambling
- uses gambling as a way to escape problems
- keeps gambling to try to make back money that had previously been lost ("chasing")
- lies about the extent of gambling

- has tried to make money for gambling by engaging in illegal or immoral behavior
- has problems at work or home caused by the gambling
- relies on other people to get him or her out of financial problems caused by the gambling

Pathological gambling disorder is distinguished from social gambling, in which the person is typically socializing with friends, gambling for a limited period of time, and gambling with a limited sum of money that they can afford to lose. Pathological gambling disorder is also distinguished from professional gambling, in which participants limit their risks and discipline their behavior. Lastly, pathological gambling disorder must be distinguished from a **manic episode**; in most cases, the distinguishing feature of the disorder is that the manic-like behavior disappears after the person leaves the gambling setting.

Treatments

There are a number of different treatments for pathological gambling disorder. **Psychodynamic psychotherapy** attempts to uncover any underlying psychological factors that trigger the gambling. For people who are gambling to escape, such as those who are depressed, this approach may be very successful. Treating any substance abuse problems that may coexist with the pathological gambling can also be helpful. Other types of treatments involve behavioral techniques used to teach relaxation and avoidance of stimuli associated with gambling. **Aversion therapy** appears to be successful in treating pathological gambling disorder in highly motivated patients with some insight into the problem, but is not helpful for patients who are less educated or resistant to behavioral methods of treatment.

Gamblers Anonymous, or GA, is a Twelve-Step program patterned on the model of Alcoholics Anonymous (AA). The gambler's admission that she or he does have a gambling problem and a willingness to go to meetings are considered the first steps in treating pathological gambling disorder. Looking realistically at what gambling has done to a person's life, and a willingness to work hard to stop gambling are also important parts of the GA program. People involved in this program are expected to attend meetings regularly, try to make amends for wrongs that their gambling has caused, and find a sponsor (usually of the same sex) to help them through the program. Gamblers Anonymous also expects that people who stop gambling to understand that they probably will never be able to gamble again socially, just as recovering alcoholics cannot drink socially.

Prognosis

There are very few statistics on the number of people successfully treated for pathological gambling disorder. Treatment for any underlying psychological disorders or substance abuses can be very helpful. Sometimes **family therapy** is recommended. Some types of relaxation or behavioral therapy can also be helpful. Gamblers Anonymous can help in many cases, although the program has a high dropout and recurrence rate. For many people, a combination of more than one of these approaches is probably the most effective. Even when a person has successfully stopped compulsive gambling, it is unlikely that he or she will ever be able to gamble socially again, or even spend time in places where he or she once gambled.

Prevention

Prevention of pathological gambling disorder is very difficult because it is impossible to predict when someone will react to gambling in a way that leads to compulsive gambling. If a person begins to feel, however, that he or she may have a problem, immediate treatment can prevent the development of a disorder that affects all areas of life and may have legal as well as economic consequences.

See also Internet addiction disorder; Self-help groups

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Gamblers Anonymous. P. O. Box 17173, Los Angeles, CA 90017. (219) 386-8789. <www.gamblersanonymous.org>.

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Paxil *see* **Paroxetine**

Paxipam *see* **Galantamine**

PCP *see* **Phencyclidine and related disorders**

Pedophilia

Definition

Pedophilia is a paraphilia that involves an abnormal interest in children. A paraphilia is a disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving: nonhuman objects; the suffering or humiliation of oneself or one's partner (not merely simulated); or animals, children, or other nonconsenting persons. Pedophilia is also a psychosexual disorder in which the fantasy or actual act of engaging in sexual activity with prepubertal children is the preferred or exclusive means of achieving sexual excitement and gratification. It may be directed toward children of the same sex or children of the other sex. Some pedophiles are attracted to both boys and girls. Some are attracted only to children, while others are attracted to adults as well as to children.

Pedophilia is defined by mental health professionals as a mental disorder, but the American legal system defines acting on a pedophilic urge as a criminal act.

Description

The focus of pedophilia is sexual activity with a child. Many courts interpret this reference to age to mean children under the age of 18. Most mental health professionals, however, confine the definition of pedophilia to sexual activity with prepubescent children, who are generally age 13 or younger. The term *ephebophilia*, derived from the Greek word for "youth," is sometimes used to describe sexual interest in young people in the first stages of puberty.

The sexual behaviors involved in pedophilia cover a range of activities and may or may not involve the use of force. Some pedophiles limit their behaviors to exposing themselves or masturbating in front of the child, or fondling or undressing the child, but without genital contact. Others, however, compel the child to participate in oral sex or full genital intercourse.

The most common overt aspect of pedophilia is an intense interest in children. There is no typical pedophile. Pedophiles may be young or old, male or female, although the great majority are males. Unfortunately, some pedophiles are professionals who are entrusted with educating or maintaining the health and well-being of young persons, while others are entrusted with children to whom they are related by blood or marriage.

Causes and symptoms

Causes

A variety of different theories exist as to the causes of pedophilia. A few researchers attribute pedophilia along with the other **paraphilias** to biology. They hold that testosterone, one of the male sex hormones, predisposes men to develop deviant sexual behaviors. As far as genetic factors are concerned, as of 2002 no researchers have claimed to have discovered or mapped a gene for pedophilia.

Most experts regard pedophilia as resulting from psychosocial factors rather than biological characteristics. Some think that pedophilia is the result of having been sexually abused as a child. Still others think that it derives from the person's interactions with parents during their early years of life. Some researchers attribute pedophilia to arrested emotional development; that is, the pedophile is attracted to children because he or she has never matured psychologically. Some regard pedophilia as the result of a distorted need to dominate a sexual partner. Since children are smaller and usually weaker than adults, they may be regarded as nonthreatening potential partners. This drive for domination is sometimes thought to explain why most pedophiles are males.

Symptoms

A pedophile is often very attractive to the children who are potential victims. Potential pedophiles may volunteer their services to athletic teams, Scout troops, or religious or civic organizations that serve youth. In some cases, pedophiles who are attracted to children within their extended family may offer to baby-sit for their relatives. They often have good interpersonal skills with children and can easily gain the children's trust.

Some pedophiles offer rationalizations or excuses that enable them to avoid assuming responsibility for their actions. They may blame the children for being too attractive or sexually provocative. They may also maintain that they are "teaching" the child about "the facts of life" or "love"; this rationalization is frequently offered by pedophiles who have molested children related to them. All these rationalizations may be found in pornography with pedophilic themes.

Demographics

Pedophilia is one of the more common paraphilias; the large worldwide market for child pornography sug-

gests that it is more frequent in the general population than prison statistics would indicate. Together with **voyeurism** and **exhibitionism**, pedophilia is one of the three paraphilias most commonly leading to arrest by the police.

The onset of pedophilia usually occurs during adolescence. Occasional pedophiles begin their activities during middle age but this late onset is uncommon. In the United States, about 50% of men arrested for pedophilia are married.

The frequency of behavior associated with pedophilia varies with psychosocial **stress**. As the pedophile's stress levels increase, the frequency of his or her acting out generally rises also.

Pedophilia is more common among males than among females. In addition, the rate of recidivism for persons with a pedophilic preference for males is approximately twice that of pedophiles who prefer females.

Little is known about the incidence of pedophilia in different racial or ethnic groups.

Diagnosis

According to the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition text revised, the following criteria must be met to establish a **diagnosis** of pedophilia.

- Over a period of at least six months, the affected person experiences recurrent, intense and sexually arousing fantasies, sexual urges or actual behaviors involving sexual activity with a prepubescent child or children aged 13 or younger.
- The fantasies, sexual urges or behaviors cause clinically significant distress or impairment in social, occupational or other important areas of daily functioning.
- The affected person must be at least age sixteen and be at least five years older than the child or children who are the objects or targets of attention or sexual activity.

A diagnosis of pedophilia cannot be assigned to an individual in late adolescence (age 17 to 19) who is involved in an ongoing sexual relationship with a 12- or 13-year-old person.

In establishing a diagnosis of pedophilia, it is important for a mental health professional to determine if the patient is attracted to males, females or both. It is also important to determine whether incest is a factor in the relationship. Finally, the doctor must determine whether the pedophilia is exclusive or nonexclusive; that is, whether the patient is attracted only to children (exclusive pedophilia) or to adults as well as to children (nonexclusive pedophilia).

KEY TERMS

Aversion therapy—An approach to treatment in which an unpleasant or painful stimulus is linked to an undesirable behavior in order to condition the patient to dislike or avoid the behavior.

Castration—Desexing a person or animal by surgical removal of the testes (in males) or ovaries (in females). Castration is sometimes offered as a treatment option to pedophiles who are violent rapists and/or repeat offenders.

Comorbidity—Association or presence of two or more mental disorders in the same patient. A disorder that is said to have a high degree of comorbidity is likely to occur in patients diagnosed with other disorders that may share or reinforce some of its symptoms.

Ephebophilia—Sexual desire on the part of an adult for youths in the early stages of puberty, as distinct from prepubertal children.

Incest—Unlawful sexual contact between persons who are biologically related. Many therapists, however, use the term to refer to inappropriate sexual contact between any members of a family, including stepparents and stepsiblings.

Paraphilia—A disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Recidivism—A tendency to return to a previously treated activity, or repeated relapse into criminal or deviant behavior.

Voyeurism—A paraphilia that involves watching unsuspecting people, usually strangers, undress or engage in sexual activity.

One difficulty with the diagnosis of the disorder is that persons with pedophilia rarely seek help voluntarily from mental health professionals. Instead, counseling and treatment is often the result of a court order. An interview that establishes the criteria for diagnosis listed above may be enough to diagnose the condition, or surveillance or Internet records obtained through the criminal investigation may also be used.

An additional complication in diagnosis is that the paraphilias as a group have a high rate of comorbidity

with one another and an equally high rate of comorbidity with major depression, anxiety disorders, and substance abuse disorders. A person diagnosed with pedophilia may also meet the criteria for exhibitionism or for a substance abuse or mood disorder.

Treatments

In the earliest stages of **behavior modification** therapy, pedophiles may be narrowly viewed as being attracted to inappropriate persons. Such aversive stimuli as electric shocks have been administered to persons undergoing therapy for pedophilia. This approach has not been very successful.

In 2002, the most common form of treatment for pedophilia is **psychotherapy**, often of many years' duration. It does not have a high rate of success in inducing pedophiles to change their behavior.

Pedophilia may also be treated with medications. The three classes of medications most often used to treat pedophilia (and other paraphilias) are: female hormones, particularly medroxyprogesterone acetate, or MPA; luteinizing hormone-releasing hormone (LHRH) agonists, which include such drugs as triptorelin (Trelstar), leuprolide acetate, and goserelin acetate; and anti-androgens, which block the uptake and metabolism of testosterone as well as reducing blood levels of this hormone. Most clinical studies of these drugs have been done in Germany, where the legal system has allowed their use in treating repeat sexual offenders since the 1970s. The anti-androgens in particular have been shown to be effective in reducing the rate of recidivism.

Surgical castration is sometimes offered as a treatment to pedophiles who are repeat offenders or who have pleaded guilty to violent rape.

Increasingly, pedophiles are being prosecuted under criminal statutes and being sentenced to prison terms. Imprisonment removes them from society for a period of time but does not usually remove their pedophilic tendencies. In 2002, many states have begun to publish the names of persons being released from prison after serving time for pedophilia. Legal challenges to this practice are pending in various jurisdictions.

Prognosis

The prognosis of successfully ending pedophilic habits among persons who practice pedophilia is not favorable. Pedophiles have a high rate of recidivism; that is, they tend to repeat their acts often over time.

The rate of prosecution for pedophiles through the criminal justice system has increased in recent years. Pedophiles are at high risk of being beaten or killed by other prison inmates. For this reason, they must often be kept isolated from other members of a prison population. Knowledge of the likelihood of abuse by prison personnel and inmates is not, however, an effective deterrent for most pedophiles.

Prevention

The main method for preventing pedophilia is avoiding situations that may promote pedophilic acts. Children should never be allowed to in one-on-one situations with any adult other than their parents or trustworthy family members. Having another youth or adult as an observer provides some security for all concerned. Conferences and other activities can be conducted so as to provide privacy while still within sight of others.

Children should be taught to yell or run if they are faced with an uncomfortable situation. They should also be taught that it is acceptable to scream or call for help in such situations.

Another basis of preventing pedophilia is education. Children must be taught to avoid situations that make them vulnerable to pedophiles. Adults who work with youth must be taught to avoid situations that may be construed as promoting pedophilia.

Many states have adopted legislation that requires periodic background investigations of any adult who works with children. These persons may be paid, such as teachers, or they may be volunteers in a youth-serving organization.

The Boy Scouts of America has tried to address the problem of pedophilia by creating a training program that is required for all adults in the organization. All applications for volunteers are reviewed and approved by several persons. Adults and youth are required to use separate facilities on all activities. Secret meetings and one-on-one interactions between adults and youth are prohibited. This program has received several national awards.

See also Abuse; Aversion therapy

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ORGANIZATIONS

American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Phone: (913) 906-6000. Web site: <http://www.aafp.org>.

American Academy of Pediatrics. 141 Northwest Point Boulevard, Elk Grove Village, IL 60007-1098. Telephone: (847) 434-4000. Fax: (847) 434-8000. Web site: <http://www.aap.org/default.htm>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax: (202) 682-6850.

American Psychological Association. 750 First Street NW, Washington, DC, 20002-4242. Phone: (800) 374-2721 or (202) 336-5500. Web site: <http://www.apa.org>.

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Peer groups

Definition

Peer groups are an important influence throughout one's life, but they are more critical during the developmental years of childhood and adolescence. There is often controversy about the influence of a peer group versus parental influence, particularly during adolescence. Recent studies show that parents continue to have significant influence, even during adolescence, a reassuring finding for many parents. It appears that the power of the peer group becomes more important when the family relationships are not close or supportive. For example, if the parents work extra jobs and are largely unavailable, their children may turn to their peer group for emotional support. This also occurs when the conflict between parents and children during adolescence, or at any time during a child's development, becomes so great that the child feels pushed away and seeks closeness elsewhere. Most children and adolescents in this situation are not discriminating about the kind of group they join. They will often turn to a group simply because that group accepts them, even if the group is involved in illegal or negative activities. Gang involvement, for example, is a common form of organized—often antisocial—peer interaction. Gangs may be based on ethnicity, sex, and/or common activity. Most youths who join gangs come from families where drug and alcohol use, financial burdens, and broken relationships are common. The need for affiliation or closeness is often greater than the need to "do the right thing" for some adolescents who feel isolated and abandoned by members of their own family. Being part of a gang provides such individuals with acceptance and security not available at home or in other peer groups.

KEY TERMS

Cluster suicide—Refers to the phenomenon of additional suicides being attempted or completed after one suicide has occurred within a small community, such as a group of high school students.

Membership in peer groups

Despite significant gains in diversity training, current studies continue to show that children are less likely to accept those who are different from themselves. The differences can be as obvious as physical impairments, or as subtle as differences in academic motivation. These rigid standards may create an atmosphere of exclusion for some children and adolescents that pushes them toward peer acceptance of any type.

Peer groups offer children and adults alike the opportunity to develop various social skills, such as leadership, sharing or teamwork, and empathy. Peer groups also offer the opportunity to experiment with new roles and interactions, similar to treatment groups, although they are less structured. It is for this reason that many children and adolescents drift from one group to another as they "find themselves," or work toward formation of their relatively permanent identity.

Aggression in peer groups

Although bullying and teasing have long been part of peer group interactions, these negative behaviors have increased over the last decade, resulting in school violence in many instances. As children and adolescents feel

Peer groups offer children and adults alike the opportunity to develop various social skills, such as leadership, sharing or teamwork, and empathy. Peer groups also offer the opportunity to experiment with new roles and interactions, and can have positive or negative influences on an individual. *(Bill Varie/ CORBIS. Reproduced by permission.)*

marginalized from their peers, anger builds to a point of rage at times. It is at those times that violence erupts within the school or community setting.

Negative peer interactions also occur more frequently following friendships or romantic relationships that have gone sour. The level of harassment that many of these children—often young women—experience is great enough for parents to become involved. In some cases, it may be necessary to move the child to another school district. A potential remediation for these negative interactions includes more active teacher involvement when negative social interactions are observed.

Influence of peer groups

Peer groups can also have a positive influence—a fact many parents have known for years. Studies support parent's perceptions that the influence of friends can have a positive effect on academic motivation and performance. Conversely, experimentation with drugs, drinking, vandalism, and stealing may also be increased by interaction with the peer group.

Interventions

Since schools are often the site of negative peer interactions, school personnel have a unique opportunity for effective **intervention**. Many schools have peer-mediation programs, in which students are encouraged to resolve conflicts on their own without the use of violence or aggression. School counselors also organize groups within the school to handle various problems, including providing **social skills training** and empathy training.

Risks

Peer groups often provide an example for negative and harmful behaviors. Cluster **suicide** is one such example. When a teen realizes that someone he or she knew has attempted or has committed suicide, the teen may see suicide as a viable option for him- or herself as well. For this reason, schools and local media should exercise caution when reporting such tragedies. Care must be taken not to portray the suicide glamorously or mythically.

When parents try to protect their children by telling them to stay away from certain friends, they should realize that sometimes this only encourages them to seek out negative role models. Parents should be supportive of their child and redirect their child's activities to more positive and prosocial peers and events. A trusted adult friend, such as a scout leader or a respected coach, may be an important part of the redirection effort.

As noted, children and adolescents without strong family connections, or at least a positive connection with other adults in their life, face a higher risk of negative influence from peer groups. If the child or adolescent has not been able to form bonds with positive peer groups, it is more likely they will be perceived as distant and different from their peers, making them feel more like outsiders. Lower standards of acceptance often exist in less positive peer groups, making it easier for people to join. Unfortunately, many such groups often engage in self-destructive and anti-social activities.

See also: Family therapy

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Deanna Pledge, Ph.D.

Pemoline

Definition

Pemoline is classified as a central nervous system (CNS) stimulant. It is sold in the United States under the brand names Cylert and PemADD and is also available under its generic name.

Purpose

Pemoline is used in combination with psychological, educational, and social support for the treatment of **attention-deficit/hyperactivity disorder** (ADHD).

Description

Pemoline is a central nervous system stimulant that derives at least some of its effects by increasing levels of dopamine in the **brain**. Dopamine is one of several **neurotransmitters** in the brain. Neurotransmitters are naturally occurring chemicals that regulate the transmission of nerve impulses from one cell to another. Mental and physical well-being are partially dependent on maintaining the proper balance among the various neurotransmitters in the brain.

Pemoline is similar in its effects to dextroamphetamine and **methylphenidate**, two other drugs used to treat ADHD, although it is not chemically related to these drugs. The mechanism of action of CNS stimulants in the treatment of ADHD is not totally clear, but probably includes increased mental alertness, decreased mental **fatigue**, and an increased sense of well-being.

Pemoline should not be used as a substitute for psychological, educational, and social support in treating ADHD. Because pemoline may be associated with liver toxicity (poisoning causing liver damage), it should be used only after other drugs to treat ADHD have been tried. Patients should try dextroamphetamine or methylphenidate first.

Pemoline is available in 18.75-mg, 37.5-mg, and 75-mg oral tablets and in 37.5-mg chewable tablets.

KEY TERMS

Attention-deficit/hyperactivity disorder (ADHD)—A learning and behavioral disorder characterized by difficulty in sustaining attention, impulsive behavior, and excessive activity.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Recommended dosage

The dose of pemoline should be carefully adjusted to patient need. The initial dose of pemoline in children six years of age or older is 37.5 mg each morning. The dose may be increased by 18.75 mg each week to as much as 75 mg daily. Most people respond to doses ranging from 56.25 mg to 75 mg daily, although some people may require as much as 112.5 mg daily.

There is no need to continue pemoline indefinitely. Rather, patients should be evaluated both during therapy and during periods in which the medication is voluntarily stopped. In many situations, the drug may be safely discontinued altogether when the child reaches adolescence.

Precautions

Pemoline is associated with liver toxicity. Symptoms range from mild reversible changes in liver function tests to acute liver failure. The risk of liver damage should be weighed against any therapeutic benefit derived from treatment with pemoline. Therefore, if no therapeutic benefit is observed within three to four weeks of starting the drug, pemoline should be discontinued. In order to detect the early signs of liver damage, liver function tests should be performed before starting the drug and every two weeks while taking pemoline.

Because pemoline is a central nervous stimulant, physical or psychological **addiction** is possible in people who are emotionally unstable.

Side effects

Loss of appetite accompanied by weight loss generally occurs during the first few weeks after starting pemoline. With continued treatment, appetite and body weight usually stabilize.

Because it is a central nervous system stimulant, **insomnia** is a common side effect of pemoline.

The most serious side effect is liver toxicity. Liver toxicity is usually characterized by changes in liver function tests without obvious liver damage, but in rare cases, liver failure resulting in death or requiring a liver transplant has occurred.

Interactions

There are no scientific data concerning drugs that negatively interact with pemoline. However, because pemoline is considered a stimulant, other drugs with stimulant properties (caffeine, over-the-counter decongestants, **amphetamines**, antidepressants) may theoretically and inappropriately increase CNS stimulation.

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Pentobarbitol *see* **Barbiturates**

Permitil *see* **Fluphenazine**

Perphenazine

Definition

Perphenazine is a phenothiazine antipsychotic used to treat serious mental disorders. It has also been used to treat severe nausea and vomiting. It is sold in the United States under the brand name Trilafon and is also available under its generic name.

Purpose

Perphenazine is used to treat psychotic disorders and severe nausea and vomiting.

Description

Perphenazine is one of many drugs in the class called phenothiazine derivatives. Phenothiazines work by inhibiting the actions of the **brain** chemicals, dopamine and norepinephrine, which are overproduced in individuals with **psychosis**.

Recommended dosage

For the treatment of psychosis, adults usually receive a total of 4 mg to 16 mg taken as tablets in three or four doses daily, up to a maximum of 64 mg each day. Injections of perphenazine are also available and are typically given in 5 mg doses every six hours, up to 15 mg per day. Hospitalized patients can receive up to 30 mg per day in the injectable form of perphenazine.

Adult patients with serious nausea and vomiting receive 8 mg to 16 mg per day as tablets in divided into several doses up to a maximum of 24 mg per day. Injections are typically given in 5 mg to 10 mg doses every six hours, up to 15 mg per day in patients who are not confined to bed. Hospitalized patients can receive up to a maximum of 30 mg per day. Intravenous perphenazine can be given to nausea and vomiting patients up to 1 mg every one to two minutes to a maximum of 5 mg.

The correct dosage of perphenazine must be carefully determined for each patient. Physicians try to find a dose that controls symptoms of the disease without causing intolerable side effects. Dosage guidelines for the treatment of psychosis have not been established for children under the age of 12 years. In children over age 12, the lowest adult dosage is generally used to treat psychosis. Children with severe nausea and vomiting are usually given 5 mg injections every six hours.

Precautions

Persons who take perphenazine should not stop taking the drug abruptly. Instead, the dose should be decreased gradually, then stopped. People who take perphenazine often have develop sunburn more easily than Sunscreen should be used by people, especially fair-skinned individuals, taking perphenazine.

People who are known to have severe central nervous system depression should not take perphenazine or any other drug in its class. In addition, those with a prior history of brain damage, coma, or bone marrow depression should not receive perphenazine without a thorough evaluation by a doctor.

Children under the age of 12 years, the elderly (over age 65), those with a history of epilepsy, glaucoma,

prostate problems, severe asthma, and other severe breathing problems should receive perphenazine only with great caution and under close supervision of a physician. In addition, persons with a history of heart or blood vessel disease and those with a history of liver or kidney disease should take perphenazine only after a thorough evaluation. Perphenazine should also be used cautiously when taken over a long period. Rarely should perphenazine be taken by pregnant or nursing women.

Side effects

Serious or life-threatening side effects due to perphenazine are rare. However, if any of these occur, patients should contact their doctors or get immediate medical attention: **seizures**, irregular heartbeat, significant changes in blood pressure, muscle stiffness, weakness, pale skin color, and increased sweating. The treating doctor should be contacted immediately if any of these common side effects develop: rapid movements of the tongue, uncontrolled chewing movement, unusual amounts of lip smacking, and frequent movement of the arms or legs. The treating doctor should be contacted relatively soon if any of the following common side effects develop: reduced balance control, muscle spasms, restlessness, trembling, weakness in the limbs, blurred vision, and decreased night vision.

Less common side effects that need to be reported to the doctor include severe sunburn, skin rashes, and urination problems. Rare side effects that should be reported to the doctor include abdominal pain, muscle aches, joint aches, fever, chills, muscle weakness, and vomiting. Common and not serious side effects include constipation, drowsiness, decreased sweating, mouth dryness, and nasal congestion. Uncommon and not typically serious side effects include decreased sexual desire, increased susceptibility to sunburn, menstrual cycle changes, swelling or pain in the breasts, and weight gain.

Interactions

Combining perphenazine with drugs such as the anti-mallarials amodiaquine, chloroquine, and sulfadoxine-pyrimethamine (Fansidar) can increase the concentrations within the body of these three latter drugs.

Perphenazine combined with **barbiturates** tends to lower the concentrations of perphenazine in the body. Combining perphenazine with **clonidine** (Catapres), guanadrel (Hylorel), and guanethidine (Ismelin) can produce dangerously low blood pressure.

Perphenazine should not be combined with alcohol, because alcohol increases the drug's depressive effect on the central nervous system. Perphenazine inhibits the effects of levodopa in Parkinson patients when the two are combined. Lithium combined with perphenazine lowers the levels of both drugs.

Perphenazine should not be combined with analgesics (pain killers) containing narcotics because of the combination increases depressive effects on the central nervous system. Orphenadrine (Norflex) combined with perphenazine can reduce the beneficial effects of perphenazine.

KEY TERMS

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

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Personality disorders

Definition

Long-standing, deeply ingrained patterns of social behavior that are detrimental to those who display them or to others.

Description

Personality disorders constitute a separate diagnostic category (Axis II) in the American Psychiatric Association's ***Diagnostic and Statistical Manual of Mental Disorders*** (DSM). Unlike the major mental disorders (Axis I), which are characterized by periods of illness and remission, personality disorders are generally ongoing. Often, they first appear in childhood or adolescence and persist throughout a person's lifetime. Aside from their persistence, the other major characteristic of personality disorders is inflexibility. Persons affected by these disorders have rigid personality traits and coping styles, are unable to adapt to changing situations, and experience impaired social and/or occupational functioning. A further difference between personality disorders and the major clinical syndromes listed in Axis I of *DSM-IV-TR* (*DSM*, fourth edition, text revised) is that people with personality disorders may not perceive that there is anything wrong with their behavior and are not motivated to change it. Although the *DSM-IV-TR* lists specific descriptions of 10 personality disorders, these conditions are often difficult to diagnose. Some characteristics of the various disorders overlap. In other cases, the complexity of human behavior makes it difficult to pinpoint a clear dividing line between pathology and normality in the assessment of personality. In still other cases, persons may have more than one personality disorder, complicating the **diagnosis**. There also has been relatively little research done on some of the personality disorders listed in *DSM-IV-TR*.

The 10 personality disorders listed in *DSM-IV-TR* include:

- **Paranoid personality disorder**. The individual affected with this disorder believes in general that people will exploit, harm, or deceive him or her, even if there is no evidence to support this belief.
- **Schizoid personality disorder**. The individual with this disorder seems to lack desire for intimacy or belonging in a social group, and often chooses being alone to being with others. This individual also tends not to show a full range of emotions.
- **Schizotypal personality disorder**. With this disorder, the affected person is uncomfortable with (and may be unable to sustain) close relationships, and also has odd behaviors and thoughts that would typically be viewed by others as eccentric, erratic, and bizarre.
- **Antisocial personality disorder**. Individuals with this disorder have no regard for the rights of others. Other, recent names associated with this personality type are psychopath and sociopath. Unable to base their actions on anything except their own immediate desires, persons with this disorder demonstrate a pattern of impulsive, irresponsible, thoughtless, and sometimes criminal behavior. They are often intelligent, articulate individuals with an ability to charm and manipulate others; at their most dangerous, they can become violent criminals who are particularly dangerous to society because of their ability to gain the trust of others combined with their lack of conscience or remorse.
- **Borderline personality disorder**. People with this disorder are unstable in their relationships, decisions, moods, and self-perceptions. These individuals are often impulsive and insecure.
- **Histrionic personality disorder**. The behavior of individuals of this personality type is characterized by persistent attention-seeking, exaggerated emotional displays (such as tantrums), and overreaction to trivial problems and events.
- **Narcissistic personality disorder**. This disorder consists primarily of an inflated sense of self-importance coupled with a lack of empathy for others. Individuals with this disorder display an exaggerated sense of their own importance and abilities and tend to fantasize about them. Such persons also have a sense of entitlement, expecting (and taking for granted) special treatment and concessions from others. Paradoxically, individuals with narcissistic personality disorder are generally very insecure and suffer from low self-esteem.
- **Avoidant personality disorder**. This disorder has characteristics that resemble those of **social phobia**, including hypersensitivity to possible rejection and the resulting social withdrawal in spite of a strong need for love and acceptance. Individuals with this disorder are inhibited and feel inadequate in social situations.
- **Dependent personality disorder**. Persons with dependent personality disorder are extremely passive and tend to subordinate their own needs to those of others. Due to their lack of self-confidence, they avoid asserting themselves and allow others to take responsibility for their lives.
- **Obsessive-compulsive personality disorder**. This disorder is characterized by a preoccupation with orderliness, perfectionism, and control.

An additional category for personality disorders exists—personality disorder not otherwise specified. This category is reserved for clinicians' use when they encounter a patient with symptoms similar to one of the above disorders, but the exact criteria for a specific disorder are not met.

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Person-centered therapy

Definition

Person-centered therapy, which is also known as client-centered, non-directive, or Rogerian therapy, is an approach to counseling and **psychotherapy** that places much of the responsibility for the treatment process on the client, with the therapist taking a nondirective role.

Purpose

Two primary goals of person-centered therapy are increased self-esteem and greater openness to experience. Some of the related changes that this form of therapy seeks to foster in clients include closer agreement between the client's idealized and actual selves; better self-understanding; lower levels of defensiveness, guilt, and insecurity; more positive and comfortable relationships with others; and an increased capacity to experience and express feelings at the moment they occur.

Description

Background

Developed in the 1930s by the American **psychologist** Carl Rogers, client-centered therapy departed from the typically formal, detached role of the therapist emphasized in **psychoanalysis** and other forms of treatment. Rogers believed that therapy should take place in a supportive environment created by a close personal relationship between client and therapist. Rogers's introduction of the term "client" rather than "patient" expresses his rejection of the traditionally hierarchical relationship between therapist and client and his view of them as equals. In person-centered therapy, the client determines the general direction of therapy, while the therapist seeks to increase the client's insight and self-understanding through informal clarifying questions.

Beginning in the 1960s, person-centered therapy became associated with the human potential movement. This movement, dating back to the beginning of the 1900s, reflected an altered perspective of human nature. Previous psychological theories viewed human beings as inherently selfish and corrupt. For example, Freud's theory focused on sexual and aggressive tendencies as the primary forces driving human behavior. The human potential movement, by contrast, defined human nature as inherently good. From its perspective, human behavior is motivated by a drive to achieve one's fullest potential.

Self-actualization, a term derived from the human potential movement, is an important concept underlying person-centered therapy. It refers to the tendency of all human beings to move forward, grow, and reach their fullest potential. When humans move toward self-actualization, they are also pro-social; that is, they tend to be concerned for others and behave in honest, dependable, and constructive ways. The concept of self-actualization focuses on human strengths rather than human deficiencies. According to Rogers, self-actualization can be blocked by an unhealthy self-concept (negative or unrealistic attitudes about oneself).

Rogers adopted terms such as "person-centered approach" and "way of being" and began to focus on personal growth and self-actualization. He also pioneered the use of encounter groups, adapting the sensitivity training (T-group) methods developed by Kurt Lewin (1890-1947) and other researchers at the National Training Laboratories in the 1950s. More recently, two major variations of person-centered therapy have developed: experiential therapy, developed by Eugene Gendlin in 1979; and process-experiential therapy, developed by Leslie Greenberg and colleagues in 1993.

While person-centered therapy is considered one of the major therapeutic approaches, along with psychoanalytic and **cognitive-behavioral therapy**, Rogers's influence is felt in schools of therapy other than his own. The concepts and methods he developed are used in an eclectic fashion by many different types of counselors and therapists.

Process

Rogers believed that the most important factor in successful therapy was not the therapist's skill or training, but rather his or her attitude. Three interrelated attitudes on the part of the therapist are central to the success of person-centered therapy: congruence; unconditional positive regard; and empathy. Congruence refers to the therapist's openness and genuineness—the willingness to relate to clients without hiding behind a professional facade. Therapists who function in this way have all their

KEY TERMS

Congruence—A quality of the client-centered therapist, consisting of openness to the client.

Empathy—A quality of the client-centered therapist, characterized by the therapist's conveying appreciation and understanding of the client's point of view.

Encounter groups—A term coined by Carl Rogers for therapist-run groups that focus on personal exploration, experiencing in the here-and-now (that is, feelings and interpersonal exchanges occurring in the group setting), and genuine concern and honesty among the members.

Experiential therapy—An approach to therapy that focuses on experiencing inner feelings, rather than talking about problems in a disconnected, intellectual way. Although it is based on person-centered therapy, experiential therapy is more directive because it uses techniques from a variety of therapeutic approaches to draw out a person's inner experiences.

Human potential movement—A movement dating back to the beginning of the 1900s that reflected an altered perspective of human nature from inherently corrupt to inherently good.

Nondirective therapy—An approach to therapy in which the therapist actively attempts to avoid giving advice, making interpretations, or otherwise influencing the focus of the individual's thoughts or statements.

Play therapy—A type of psychotherapy for young children involving the use of toys and games to build a therapeutic relationship and encourage the child's self-expression.

Process-experiential therapies—A group of therapies based on a person-centered approach that incorporate elements of cognitive and Gestalt therapies.

Self-actualization—The belief that all human beings have an inborn tendency toward growth and self-improvement.

Self-concept—Attitudes about oneself.

Sensitivity training—Training conducted in T-groups to reduce tensions and racial prejudice among the public.

T-groups—Short for "basic skills training groups" that were focused on education and discussion regarding social issues, personal problems experienced outside the group setting, and problems from one's past.

Unconditional positive regard—A quality of the client-centered therapist, characterized by the therapist's acceptance of the client without judgment.

feelings available to them in therapy sessions and may share significant emotional reactions with their clients. Congruence does *not* mean, however, that therapists disclose their own personal problems to clients in therapy sessions or shift the focus of therapy to themselves in any other way.

Unconditional positive regard means that the therapist accepts the client totally for who he or she is without evaluating or censoring, and without disapproving of particular feelings, actions, or characteristics. The therapist communicates this attitude to the client by a willingness to listen without interrupting, judging, or giving advice. This attitude of positive regard creates a nonthreatening context in which the client feels free to explore and share painful, hostile, defensive, or abnormal feelings without worrying about personal rejection by the therapist.

The third necessary component of a therapist's attitude is empathy ("accurate empathetic understanding"). The therapist tries to appreciate the client's situation from the client's point of view, showing an emotional understanding of and sensitivity to the client's feelings throughout the therapy session. In other systems of therapy, empathy with the client would be considered a preliminary step to enabling the therapeutic work to proceed; but in person-centered therapy, it actually constitutes a major portion of the therapeutic work itself. A primary way of conveying this empathy is by active listening that shows careful and perceptive attention to what the client is saying. In addition to standard techniques, such as eye contact, that are common to any good listener, person-centered therapists employ a special method called reflection, which consists of paraphrasing and/or summarizing what a client has just said. This technique shows that the therapist is listening carefully and accurately, and gives clients an added opportunity to examine their own thoughts and feelings as they hear them repeated by another person. Generally, clients respond by elaborating further on the thoughts they have just expressed.

According to Rogers, when these three attitudes (congruence, unconditional positive regard, and empathy) are conveyed by a therapist, clients can freely express themselves without having to worry about what the therapist thinks of them. The therapist does not attempt to change the client's thinking in any way. Even negative expressions are validated as legitimate experiences. Because of this nondirective approach, clients can explore the issues that are most important to them—not those considered important by the therapist. Based on the principle of self-actualization, this undirected, uncensored self-exploration allows clients to eventually recognize alternative ways of thinking that will promote personal growth. The therapist merely facilitates self-actualization by providing a climate in which clients can freely engage in focused, in-depth self-exploration.

Applications

Rogers originally developed person-centered therapy in a children's clinic while he was working there; however, person-centered therapy was not intended for a specific age group or subpopulation but has been used to treat a broad range of people. Rogers worked extensively with people with **schizophrenia** later in his career. His therapy has also been applied to persons suffering from depression, anxiety, alcohol disorders, cognitive dysfunction, and **personality disorders**. Some therapists argue that person-centered therapy is not effective with nonverbal or poorly educated individuals; others maintain that it can be successfully adapted to any type of person. The person-centered approach can be used in individual, group, or **family therapy**. With young children, it is frequently employed as **play therapy**.

There are no strict guidelines regarding the length or frequency of person-centered therapy. Generally, therapists adhere to a one-hour session once per week. True to the spirit of person-centered therapy, however, scheduling may be adjusted according to the client's expressed needs. The client also decides when to terminate therapy. Termination usually occurs when he or she feels able to better cope with life's difficulties.

Normal results

The expected results of person-centered therapy include improved self-esteem; trust in one's inner feelings and experiences as valuable sources of information for making decisions; increased ability to learn from (rather than repeating) mistakes; decreased defensiveness, guilt, and insecurity; more positive and comfortable relationships with others; an increased capacity to experience and express feelings at the moment they occur; and openness to new experiences and new ways of thinking about life.

Outcome studies of humanistic therapies in general and person-centered therapy in particular indicate that people who have been treated with these approaches maintain stable changes over extended periods of time; that they change substantially compared to untreated persons; and that the changes are roughly comparable to the changes in clients who have been treated by other types of therapy. Humanistic therapies appear to be particularly effective in clients with depression or relationship issues. Person-centered therapy, however, appears to be slightly less effective than other forms of humanistic therapy in which therapists offer more advice to clients and suggest topics to explore.

Abnormal results

If therapy has been unsuccessful, the client will not move in the direction of self-growth and self-acceptance. Instead, he or she may continue to display behaviors that reflect self-defeating attitudes or rigid patterns of thinking.

Several factors may affect the success of person-centered therapy. If an individual is not interested in therapy (for example, if he or she was forced to attend therapy), that person may not work well together with the therapist. The skill of the therapist may be another factor. In general, clients tend to overlook occasional therapist failures if a satisfactory relationship has been established. A therapist who continually fails to demonstrate unconditional positive regard, congruence, or empathy cannot effectively use this type of therapy. A third factor is the client's comfort level with nondirective therapy. Some studies have suggested that certain clients may get bored, frustrated, or annoyed with a Rogerian style of therapeutic interaction.

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Association for the Development of the Person-Centered Approach. <http://www.adpca.org>.

Center for Studies of the Person. 1150 Silverado, Suite 112, La Jolla, California 92037. (858) 459-3861. <http://www.centerfor theperson.org>.

World Association for Person-Centered and Experiential Psychotherapy and Counseling (WAPCEPC). c/o SGGT Office, Josefstrasse 79, CH-8005 Zürich, Switzerland. +41 1 2717170. <http://pce-world.org>.

Sandra L. Friedrich, M.A.

Pervasive developmental disorders

Definition

Pervasive developmental disorders are a group of conditions originating in childhood that involve serious impairment in several areas, including physical, behavioral, cognitive, social, and language development.

Description

Pervasive developmental disorders (PDDs) are thought to be genetically based, with no evidence linking them to environmental factors; their incidence in the general population is estimated at 1%. The most serious PDD is **autism**, a condition characterized by severely impaired social interaction, communication, and abstract thought, and often manifested by stereotyped and repetitive behavior patterns. Many children who are diagnosed with PDDs today would have been labeled psychotic or schizophrenic in the past.

The handbook used by mental health professionals to diagnose mental disorders such as PDDs is the ***Diagnostic and Statistical Manual of Mental Disorders***. The 2000 edition of this manual (fourth edition, text revised) is known as the *DSM-IV-TR*. Published by the American Psychiatric Association, the *DSM* contains diagnostic criteria, research findings, and treatment information for mental disorders. It is the primary reference for mental health professionals in the United States.

Besides autism, the *DSM* lists several other conditions as PDDs:

Rett's disorder

Characterized by physical, mental, and social impairment, this syndrome appears between the ages of five months and four years in children whose development has been normal up to that point. Occurring only in girls, it involves impairment of coordination, repetitive movements, a slowing of head growth, and severe or profound **mental retardation**, as well as impaired social and communication skills.

Childhood disintegrative disorder

This disorder is marked by the deterioration of previously acquired physical, social, and communication skills after at least two years of normal development. More common in males than females, it first appears between the ages of two and 10 (usually at three or four years of age), and many of its symptoms resemble those of autism. Other names for this disorder are Heller's syndrome, **dementia** infantilis, and disintegrative **psychosis**. It sometimes appears in conjunction with a medical condition such as Schilder's disease, but usually no organic cause can be found.

Asperger's disorder

Children with this disorder have many of the same social and behavioral impairments as autism, except for difficulties with language. They lack normal tools of social interaction, such as the ability to meet someone

else's gaze, use appropriate body language and gestures, or react to another person's thoughts and feelings. Behavioral impairments include the repetitive, stereotyped motions and rigid adherence to routines that are characteristic of autism. Like **childhood disintegrative disorder**, **Asperger's disorder** is more common in males than females.

Prognosis

In general, the prognosis in each of these conditions is tied to the severity of the illness.

The prognosis for Asperger's syndrome is more hopeful than the others in this cluster. These children are likely to become functional, independent adults, but will always have problems with social relationships. They are also at greater risk for developing serious mental illness than the general population.

The prognosis for autistic disorder is not as good, although great strides have been made in recent years in its treatment. The higher the patient's intelligence quotient (IQ) and ability to communicate, the better the prognosis. However, many patients will always need some level of custodial care. In the past, most of these individuals were confined to institutions, but many are now able to live in **group homes** or supervised apartments. The prognosis for childhood disintegrative disorder is the least favorable. These children will require intensive and long-term care.

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Autism National Committee (AUTCOM). P.O. Box 6175, North Plymouth, MA 02362-6175. Web site: <http://www.autcom.org/>.

Autism Research Institute. 4182 Adams Avenue, San Diego, CA 92116. Telephone: (619) 281-7165. Web site: <http://www.autism.com/ari>.

New Jersey Center for Outreach and Services for the Autism Community (COSAC). 1450 Parkside Avenue Suite 22, Ewing, NJ 08638. Telephone: (609) 883-8100 or (800) 4-AUTISM (428-8476). Web site: <http://www.njcosac.org>.

PET *see* **Positron emission tomography**

Phencyclidine and related disorders

Definition

Phencyclidine (PCP) is a street drug known as "angel dust" that causes physiological changes to the nervous and circulatory system, disturbances in thinking and behavior, and can cause **hallucinations**, psychotic disorder, mood disorder, and anxiety disorder.

Description

Phencyclidine (PCP) is the best known of several related drugs including ketamine, cyclohexamine, and dizocilpine. PCP was first synthesized by a pharmaceutical company in the 1950s and sold under the brand names Sernyl and Sernylan until 1967. It was hoped that PCP could be used as a dissociative anesthetic, because it produced a catatonic state in which patients were dissociated from their environment and from pain, but not unconscious. Problems with side effects as the drug wore off, including agitated behavior and hallucinations made PCP unsuitable for medical use. Ketamine (Ketlar, Ketaject) is less potent, has fewer side effects and is approved for use as a human anesthetic.

PCP became an illicit street drug in the mid-1960s. It was most commonly found in large cities such as New York and San Francisco, and even today, most users tend to live in urban areas. Into the 1970s, PCP appeared mainly as a contaminant of other illict drugs, especially marijuana and cocaine. This complicated **diagnosis** of PCP use, as many people did not know that they had ingested the drug.

PCP is easy to manufacture and is inexpensive. By the late 1970s, in some urban areas its use equaled that of crack cocaine. Use of PCP peaked between 1973 and 1979. Since 1980, PCP use has declined, although as with most illicit drugs, its popularity increases and decreases in cycles.

People who use PCP exhibit both behavioral and physiological signs. The effects of PCP are erratic, and serious complications can occur at relatively low doses. It is often difficult to distinguish PCP use from the use of other illicit drugs, and many people who use PCP also abuse other substances. According to the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*), which presents guidelines used by the American Psychiatric Association for diagnosis of mental disorders, phencyclidine can induce mood disorder, psychotic disorder, and anxiety disorder—but these classifications are somewhat controversial and not all are recognized by

Phencyclidine, also known as "angel dust." *(Custom Medical Stock Photo, Inc. Reproduced by permission.)*

international psychiatric organizations. No human studies have been done on PCP tolerance and withdrawal. Animal studies suggest that both conditions occur, just as they do with many other abused drugs.

PCP is a Schedule II drug under the Controlled Substances Act. In its pure form, it is a white powder that dissolves easily in water. Once dissolved, the solution can be sprayed on tobacco or marijuana cigarettes. Less pure forms range from yellowish-tan to brown and can be a sticky mass. On the street PCP has many names including angel dust, devil dust, tranq, hog, crazy Eddie, rocket fuel, embalming fluid, wack, and ozone. Ketamine, which is legal and not regulated as a Schedule III controlled substance, also used illicitly, is known on the street as K, special K, and cat valium. Crack cocaine combined with PCP is sometimes called tragic magic. Marijuana laced with PCP is called love boat, killer weed, or crystal supergrass.

Causes and symptoms

Causes

PCP is easy to manufacture and is inexpensively available on the street in most cities, especially East Coast cities. It can be eaten, smoked, injected, snorted, and is readily soluble and will cross the skin barrier if liquid PCP is spilled on skin or clothing. The most common methods of ingestion are eating and smoking marijuana or tobacco on which liquid PCP has been sprayed. PCP is long acting. It accumulates in body fat, and flashbacks can occur as it is released from fat during exercise.

PCP binds to receptors in the **brain** and interferes with the chemical reactions that mediate the transmission of nerve impulses. It is deactivated slowly by the liver and excreted in urine. Although there are no controlled human studies on PCP intoxication, monkeys allowed free use of PCP will dose themselves repeatedly and maintain an almost continuous state of intoxication. They exhibit withdrawal symptoms if their supply of the drug is restricted. PCP is considered to be psychologically and possibly physically addictive in humans.

Symptoms

PCP produces both physiological and psychological symptoms. Effects of the drug are erratic and not always dose-dependent. Physical symptoms include:

- involuntary rapid movements of the eyes vertically or horizontally
- high blood pressure
- racing heartbeat
- dizziness and shakiness
- drooling
- increased body temperature
- reduced response to pain
- slurred speech
- excessive sensitivity to sound
- lack of muscle coordination
- muscle rigidity or frozen posture
- **seizures**
- breakdown of muscle and excretion of muscle proteins in urine
- coma
- death

Psychiatric and social symptoms include:

- disordered thinking and confusion
- impaired judgment
- belligerence
- aggressiveness
- agitation
- impulsiveness and unpredictability
- schizophrenic-like psychoses
- hallucinations of sight, sound, or touch
- memory impairment
- difficulty in social-emotional relationships
- chaotic lifestyle including difficulty functioning at work or school, legal and financial problems

PCP is known for its variability of symptoms, which change both from person to person and from exposure to exposure. In addition, symptoms come and go throughout a period of intoxication that can last from one to two hours for low dose exposure to one to four days for high

dose exposure. Severity of symptoms is not always related to the size of the dose as measured by blood levels of the drug.

Three rough phases of intoxication have been established: behavioral toxicity, stuperous stage, and comatose stage. Many patients fluctuate between phases, and some present symptoms that do not fit neatly into any phase. In the behavioral toxicity stage, people tend to gaze blankly while their eyes dart horizontally or vertically. Muscle control is poor, and the person may make repetitive movements, grind the teeth, or grimace. Body temperature, heart rate, and respiration are mildly elevated. Vomiting and drooling may occur.

In the stuperous phase the eyes are wide open, and the person appears wide awake, but in a stupor. Seizures may occur if the person is stimulated. The eyes may dart in any direction while the gaze remains fixed. Body temperature is increased substantially. Heart and respiration rate are increased by about 25%. Muscles are rigid with twitching.

In the comatose stage, which may last from one to four days, the person is in a deep coma. The pupils are dilated and the eyes drift. Body temperature is elevated to the point of being life-threatening. The heart rate is dangerously high, increasing to about twice the normal level and blood pressure is dangerously low. Breathing may stop for brief periods (apnea). There is no response to pain, and the person sweats heavily. Death is possible, although most deaths with PCP occur in earlier stages through accidents or **suicide**.

Demographics

In the 1970s, PCP was used mainly by adolescents. Today the largest regular users are between the ages of 26 and 35. Men outnumber women users two to one, and men account for about three-quarters of PCP-related emergency room visits. Most users live in cities. About 90% of people who use PCP use other drugs as well, usually marijuana and alcohol. About 3% of substance abuse deaths are caused by PCP. Studies by the National Institute of Drug Abuse show that PCP use by high school students has declined steadily from about 13% in 1979 to about 4% in 1997.

Diagnosis

Diagnosis of PCP abuse or dependence is often complicated by the fact that symptoms are variable. Most people who use PCP use other drugs; and PCP can be a contaminant in other street drugs or can itself be contaminated with other chemicals. PCP use is also found among people with psychiatric disorders. In many ways, PCP mimics the symptoms of **schizophrenia**.

The American Psychiatric Association recognizes two levels of PCP disorders: PCP dependence and PCP abuse. In addition, it recognizes seven other PCP-induced psychiatric disorders.

PCP dependence is characterized by a psychological dependence or craving for the drug, as well as withdrawal symptoms if it is discontinued. Although physical dependence has been shown in animal studies with suggestions that physical dependence is present in heavy human users, no human studies have confirmed this. Heavy users may take the drug several times a day. They continue to use it despite experiencing psychological or physical problems. People with psychiatric disorders are more likely to have bad side effects from PCP than those without psychiatric problems. Adverse effects of PCP dependence can continue for weeks after the drug is discontinued.

Individuals with PCP abuse use the drug less regularly than those with PCP dependence. They experience both physical and psychological symptoms of PCP intoxication and often are unable to meet the normal demands of society (work, school, family responsibilities). Because PCP use impairs judgment and increases aggressiveness, they often are involved in accidents while under the drug's influence.

Phencyclidine-induced disorders include:

- PCP intoxication with or without perceptual disturbances
- PCP intoxication **delirium**
- PCP-induced psychotic disorder
- PCP-induced mood disorder
- PCP-induced anxiety disorder
- PCP-induced disorders not otherwise specified

PCP intoxication and delirium are diagnosed by a history of recent PCP use, behavioral changes and physical changes that are not accounted for by any other substance use, medical condition, or psychiatric condition. PCP is present in the blood and urine. With PCP intoxication, a patient may have hallucinations but be aware that these are caused by PCP use.

PCP delirium is diagnosed when a patient exhibits muddled thinking, hostility, bouts of hyperactivity and aggressiveness, and schizophrenic-like symptoms, as well as the more severe physical symptoms listed above. PCP delirium can last for hours or days.

It may be difficult initially to separate PCP intoxication or delirium from other mental disorders, as symp-

toms may mimic depression, schizophrenia, mood disorders, **conduct disorder**, and **antisocial personality disorder**. People with PCP intoxication also have physical and psychological symptoms similar to those that occur with the use of other illicit drugs, complicating diagnosis. A complete physical and psychological history helps rule out these other conditions.

Treatments

People experiencing PCP intoxication or delirium often hurt themselves or others. They are generally kept in an environment where there is as little stimulation as possible. They are restrained only as much as is necessary to keep them from hurting themselves or others until the level of PCP in their bodies can be reduced. Antipsychotic medications may be used to calm patients in cases of PCP delirium.

There are no quick ways to rid the body of PCP. If the PCP has been eaten, stomach pumping or feeding activated charcoal may help keep the drug from being absorbed into the bloodstream. Physical symptoms such as high body temperature are treated as needed.

Most people recover from PCP intoxication or delirium without major medical complications. Many are habitual users who return to use almost immediately. There are no specific behavioral therapies to treat PCP use. Antidepressants are sometimes prescribed. Long-term residential treatment or intensive outpatient treatment along with urine monitoring offers some chance of success. Narcotics Anonymous, a self-help group, may be helpful for some patients.

Prognosis

Relapse and return to PCP use is common, even among people who have experienced severe medical and psychiatric complications from the drug. Since many users also abuse other drugs, their success in renouncing PCP is tied to their successful treatment for other addictions. Successful treatment takes persistence, patience, and a functional support system, all of which many users lack.

Prevention

PCP intoxication and related disorders can be prevented by not using the drug.

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National Clearinghouse for Alcohol and Drug Information. P. O. Box 2345, Rockville, MD 20852. (800) 729-6686. <http://www.health.org>.

National Institute on Drug Abuse. 5600 Fishers Lane, Room 10 A-39, Rockville, MD 20857. (888) 644-6432. <http://niad.nih.gov>.

Tish Davidson, A.M.

Phenelzine

Definition

Phenelzine is classified as a monoamine oxidase (MAO) inhibitor. In the United States, phenelzine is sold under the brand name Nardil.

Purpose

Phenelzine is used to treat certain types of serious depression and severe depression complicated by severe anxiety that do not respond to other antidepressant drugs.

Description

Phenelzine is a member of a class of drugs called monoamine oxidase inhibitors. Monoamine oxidase, or MAO, is an enzyme found throughout the body. In the **brain**, MAO breaks down norepinephrine and serotonin, two naturally occurring chemicals that are important in maintaining mental well-being and preventing depression. Monoamine oxidase inhibitors, such as phenelzine, reduce the activity of MAO. Less norepinephrine and serotonin are broken down, so their levels rise. This helps to lift depression.

Phenelzine is effective for treating depression, especially complicated types of depression that have not responded to more traditional antidepressants. However, phenelzine also affects the MAO enzyme in many other areas of the body. This accounts for the large number of serious side effects and drug interactions it causes.

Recommended dosage

Adults are usually started on 15 mg of phenelzine three times per day. This dosage can be increased to a maximum of 90 mg per day if lower doses are not effective, and the patient can tolerate the higher dose without excessive side effects. After the maximum benefits are achieved, the dosage is usually lowered over several weeks to the lowest level that is effective. This could be as little as 15 mg daily or every other day.

In general, phenelzine is not recommended for people over the age of 60. When it is used by the elderly, the starting dosage is usually 15 mg taken in the morning. This dose may be gradually increased over time to a maximum of 60 mg. Phenelzine is not frequently given to children under the age of 16, and recommended dosage in such cases has not been established.

Phenelzine can be taken with food or on an empty stomach. It should not be taken close to bedtime, because it can interfere with sleep. The benefits of this drug may not become apparent for as long as four to eight weeks. Patients should be aware of this and continue taking the drug as directed even if they do not see an immediate improvement.

Precautions

People with a history of congestive heart failure, high blood pressure, cardiovascular disease, headache, kidney disease, or liver disease should not take phenelzine or, if they do take it, they should be under careful medical supervision and monitoring. Children under the age of 16 and people with a history of low blood pressure, **bipolar disorders**, angina, hyperactivity, diabetes mellitus, **seizures**, suicidal thoughts, and overactive thyroid should discuss the risks and benefits of this drug with their physician, and a decision to treat should be made on an individual basis. If these patients receive phenelzine, it should be taken only under the careful supervision of a doctor. Evidence suggests that phenelzine should not be used during pregnancy or while nursing.

People taking phenelzine should get up slowly from a reclining position to prevent dizziness. Those who use phenelzine should use caution when operating heavy machinery or performing hazardous activities that require alertness.

It is very important for the doctor to determine the lowest dosage of phenelzine that produces benefits. When this dosage is exceeded, side effects and interactions increase substantially. Over-the-counter medications that contain decongestants or dextromethorphan (for example, some cough syrups and cold remedies) should not be taken while using phenelzine (see "Interactions," below). In addition, foods and beverages that contain tyramine should not be eaten while using this medication. These foods include yeast or meat extracts, fermented sausage, overripe fruit, sauerkraut, cheese, and fava beans. Phenelzine should not be used within two weeks of undergoing surgery that requires anesthesia.

KEY TERMS

Amphetamines—A group of powerful and highly addictive substances that stimulate the central nervous system. May be prescribed for various medical conditions, but are often purchased illicitly and abused.

Angina—Severe pain and a feeling of constriction around the heart.

Bipolar affective disorder—A disorder in which a person alternates manic and depressive episodes.

Hepatitis—An inflammation of the liver that can be caused by a variety of factors.

Jaundice—A yellowing of the skin caused by excess bilirubin in the blood; a liver disorder.

Nonendogenous—A factor that arises or is produced outside of the organism.

Tyramine—Intermediate product between the chemicals tyrosine and epinephrine in the body and a substance normally found in many foods. Found especially in protein-rich foods that have been aged or fermented, pickled, or bacterially contaminated, such as cheese, beer, yeast, wine, and chicken liver.

Side effects

The enzyme monoamine oxidase regulates functions throughout the body. Phenelzine decreases the activity of monoamine oxidase in all the areas of the body where it exists, not just in the brain. This is why phenelzine is capable of causing a wide variety of side effects in many different organ systems.

The most common and unavoidable side effects associated with phenelzine use are swelling of the feet and ankles, low blood pressure upon arising from a reclining position, and **insomnia** if taken near bedtime. Mild side effects and ones that are not frequent include skin rash, headache, dizziness, confusion, memory impairment, drowsiness, weakness, shakiness, muscle

twitching, constipation, indigestion, appetite changes, and dry mouth. Although these side effects are considered mild, they should be reported to the treating doctor.

More serious side effects include hepatitis coupled with jaundice, high blood pressure crisis, excessive nervousness, and changes in heart rate. The high blood pressure crisis involves significantly increased blood pressure, severe headache, heart palpitations, nausea, vomiting, and sweating. These symptoms need immediate medical attention. Sexual function can be affected in both men and women.

Interactions

Phenelzine interacts with a long list of drugs. Some of these interactions can cause death. This section is not a complete list of interactions, but it includes the most serious ones. Patients must make sure that every health care professional who takes care of them (for example, doctors, dentists, podiatrists, optometrists, pharmacists, nurses) knows that they take phenelzine, as well as all of the other prescription, nonprescription, and herbal drugs they take.

All foods and beverages containing tyramine need to be avoided while taking this medication. Coffee, tea, and cola beverages should be restricted to one serving per day. Alcohol should not be used while taking phenelzine, because it can significantly increase blood pressure.

Any type of amphetamine and other stimulant should not be used, because this combination can increase blood pressure to dangerously high levels. Phenelzine should not be combined with other antidepressants, because of increased risk of dangerously high blood pressure and manic episodes. Patients taking phenelzine should stop the drug, then wait at least 14 days before starting any other antidepressant. The same holds true when discontinuing another antidepressant and starting phenelzine. Phenelzine combined with **barbiturates** can prolong the effects of barbiturates.

Phenelzine combined with **clomipramine** (Anafranil) can cause death. Diet drugs and decongestants containing compounds such as dextromethorphan should not be combined with phenelzine because of an increased risk of seizures and agitation. Phenelzine can decrease the effectiveness of high blood pressure drugs, such as guanadrel (Hylorel) and guanethidine (Ismelin). Phenelzine combined with the Parkinson disease drug levodopa (Dopar, Larodopa) can produce severely high blood pressure. Lithium should not be used with phenelzine because of the risk of developing extremely high fever. Phenelzine can prolong the effects of muscle relaxants when the two are combined.

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Mark Mitchell, M.D.

Phobias *see* **Agoraphobia, Social phobias, and Specific phobias**

Phonological disorder

Definition

Phonological disorder occurs when a child does not develop the ability to produce some or all sounds necessary for speech that are normally used at his or her age.

Description

Phonological disorder is sometimes referred to as articulation disorder, developmental articulation disorder, or speech sound production disorder. If there is no known cause, it is sometimes called "developmental phonological disorder." If the cause is known to be of neurological origin, the names "dysarthria" or "dyspraxia" are often used. Phonological disorder is characterized by a child's inability to create speech at a level expected of his or her age group because of an inability to form the necessary sounds.

There are many different levels of severity of phonological disorder. These range from speech that is completely incomprehensible, even to a child's immediate family members, to speech that can be understood by everyone but in which some sounds are slightly mispronounced. Treatment for phonological disorder is important not only for the child's development to be able to

form speech sounds, but for other reasons, as well. Children who have problems creating speech sounds may have academic problems in subject areas such as spelling or reading. Also, children who sound different than their peers may find themselves frustrated and ridiculed, and may become less willing to participate in play or classroom activities.

Causes

Phonological disorder is often divided into three categories, based on the cause of the disorder. One cause is structural problems, or abnormalities in the areas necessary for speech sound production, such as the tongue or the roof of the mouth. These abnormalities make it difficult for children to produce certain sounds, and in some cases make it impossible for a child to produce the sounds at all. The structural problem causing the phonological disorder generally needs to be treated before the child goes into language therapy. This therapy is especially useful, because, in many of these cases, correction of the structural problem results in correction of the speech sound problem.

The second category of phonological disorder is problems caused by neurological problems or abnormalities. This category includes problems with the muscles of the mouth that do not allow the child sufficient fine motor control over the muscles to produce all speech sounds. The third category of phonological disorder is phonological disorder of an unknown cause. This is sometimes called "developmental phonological disorder." Although the cause is not known, there is much speculation. Possible causes include slight **brain** abnormalities, causes rooted in the child's environment, and immature development of the neurological system. As of 2002, there is research pointing to all of these factors, but no definitive cause has been found.

Symptoms

The symptoms of phonological disorder differ significantly depending on the age of the child. It is often difficult to detect this disorder, as the child with phonological disorder develops speech sounds more slowly than his or her peers; generally, however, he or she develops them in the same sequence. Therefore, speech that may be normal for a four-year-old child may be a sign of phonological disorder in a six-year-old.

Nearly all children develop speech sounds in the same sequence. The consonant sounds are grouped into three main groups of eight sounds each: the early eight, the middle eight, and the late eight. The early eight include consonant sounds such as "m," "b,", and "p." The middle eight include sounds such as "t," "g", and "chi," and the late eight include more complicated sounds such as "sh," "th," "z," and "zh." Many children do not normally finish mastering the late eight until they are seven or eight years old. As children normally develop speech sound skills, there are some very common mistakes that are made. These include the omission of sounds, (i.e., frequently at the end of words), the distortion of sounds, or the substitution of one sound for another. Often the substitution is of a sound that the child can more easily produce for one that he or she cannot.

KEY TERMS

Dysarthria—A group of speech disorders caused by disturbances in the strength or coordination of the muscles of the speech mechanism as a result of damage to the brain or nerves. Difficulty talking and speaking.

Dyspraxia—Developmental dyspraxia is an impairment or immaturity of the organization of movement. It is a defect in the way the brain processes information, resulting in messages not being correctly or fully transmitted. The term dyspraxia comes from the word "praxis", meaning "doing" or "acting". Dyspraxia is associated with problems of perception, language, and thought.

Diagnosis

The **diagnosis** for phonologic disorder depends greatly on the age of the child in question. Children who are four years old may have speech production difficulties that show normal development for their age, while children who are eight years old and making the same mistakes may have phonological disorder. In children with phonological disorder, the pattern and order of speech sound acquisition is usually similar to that of normally developing children. However, the speech sound skills develop more slowly, so age is an important factor in determining a diagnosis of phonological disorder. Children with phonological disorder may make the same speech sound mistakes as younger, normally developing children. In some cases, however, children with phonological disorder have demonstrated more instances of omissions, substitutions, and distortions in their speech.

When exploring a diagnosis of phonological disorder, it is generally recommended that a physician check for other possible causes of the signs and symptoms. A child's hearing should be checked, because speech sounds that are not heard well by a child cannot be imi-

tated and learned well. In school-age children, reading comprehension should be checked to discover any other language disorders, which are sometimes present in addition to phonological disorder. Any general developmental delays should also be checked by the physician. It is important to remember that for some children whose native language is one other than English, the problems with speech sounds may result from poor crossover of sounds between the child's languages. Therefore, when diagnosing a child with a different native language, it is recommended that tests involve the child's first language, as well as English. Also, it must be remembered that in some parts of the country, normal pronunciation of some words is different from pronunciation in other parts of the country. Therefore a child's background and history can be very important in making a diagnosis.

The ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*) states that for a diagnosis of phonological disorder to be made, three general criteria must be met. The first criterion is that the child is not developing speech sounds skills considered to be appropriate for his or her age group. Also, this lack of speech sound acquisition must be causing problems for the child at home, at school, or in other important aspects of the child's life. If the child is mentally retarded, has problems with his or her speech muscles or hearing, or if there is environmental deprivation, a diagnosis of phonological disorder may still be appropriate. The diagnosis can only be made, however, if the lack of speech sounds skill is considered greater than the child's other problems.

Demographics

Phonological disorder of unknown cause is considered significantly more common than phonological disorder that is caused by neurological or structural abnormalities. It has been estimated that 7–8% children who are five years old have phonological disorder with any cause (developmental phonological disorder). About 7.5% of children between the ages of three and eleven are thought to have development phonological disorder. Phonological disorder is more common in boys than it is in girls. Estimates suggest that two to four times as many boys as girls have the disorder. Children who have phonological disorder are more likely to have other language problems and disorders. Children with one or more family members who have this or similar language disorders are also considered to be more likely to have phonological disorders.

Treatment

Treatment by a speech-language pathologist is generally recommended for children with phonological disorders. The therapy will differ depending on an individual child's needs, but generally takes the form of practicing sounds. Sometimes the child is shown the physical ways that the sound is made, such as where to place the tongue and how to form the lips. Repetition of the difficult sounds with the therapist is an integral part of treatment. There is debate, however, over the way that children with more severe forms of the disorder should be treated. Some therapists believe that the sounds that are learned later in development should be addressed first, even if the child has not developed the more simple sound skills. Other therapists believe that simple sounds should be treated first, as it is easier for children with phonological disorder to master them. One other school of thought is that when the child develops a sense of accomplishment when these sounds are mastered, and he or she will more willingly continue with treatment. There is ongoing research on this debate, and the results as of 2002 are still mixed.

Children who have phonological disorder because of neurological or structural problems that do not allow them to produce some sounds are often helped to find approximate alternatives for the sounds within the range of sounds that they are able to produce.

Prognosis

The prognosis for children with phonological disorder is generally good. For many children, the problem resolves spontaneously. It is reported that in 75% of children with mild-or-moderate forms of the disorder, and whose problems do not stem from a medical condition, the symptoms resolve before age six. In many other cases, children who receive treatment eventually develop normal or close to normal speech. In some cases, there may be mild effects that last until adulthood, but speech is completely understandable. For children with phonological disorder due to a neurological or structural cause, the outcome generally rests on how well the cause of the problem is treated.

Prevention

There is no known way to prevent phonological disorder. A healthy diet during pregnancy and regular prenatal care may help to prevent some of the neurological or structural problems that can result in the disorder.

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American Academy of Child and Adolescent Psychiatry. 3615 Wisconsin Ave. NW, Washington, DC 20016-3007. (202) 966- 7300. <www.aacap.org>.

The American Academy of Pediatrics. 141 Northwest Point Boulevard, Elk Grove Village, IL 60007-1098. (847) 434-4000 <www.aap.org>.

American Speech-Language-Hearing Association. 10801 Rockville Pike, Rockville, MD 20852. (800) 638-8355. <http://www.asha.org>.

Tish Davidson, A.M.

KEY TERMS

Behavior modification—An approach to therapy based on the principles of operant conditioning. Behavior modification seeks to replace undesirable behaviors with preferable behaviors through the use of positive or negative reinforcement.

Bezoar—A hard ball of hair or vegetable fiber that may develop in the stomach of humans as the result of ingesting nonfood items.

Chelation—A method of treating lead or mercury poisoning by giving medications that remove heavy metals from the bloodstream. The medications that are used are called chelating agents.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Toxocariasis—Infection with roundworm larvae, commonly transmitted by the feces of dogs and cats.

Toxoplasmosis—A parasitic infection caused by the intracellular protozoan *Toxoplasmosis gondii*. Humans are most commonly infected by swallowing the oocyte form of the parasite in soil (or kitty litter) contaminated by feces from an infected cat; or by swallowing the cyst form of the parasite in raw or undercooked meat.

Trichuriasis—Infection with the larvae of roundworms. These parasites may live for 10–20 years in humans.

Pica

Definition

Pica is a term that refers to cravings for substances that are not foods. Materials consumed by patients with pica include dirt, ice, clay, glue, sand, chalk, beeswax, chewing gum, laundry starch, and hair.

Description

Pica is the craving or ingestion of nonfood items. The cravings found in patients diagnosed with pica may be associated with a nutritional deficiency state, such as iron-deficiency anemia; with pregnancy; or with **mental retardation** or mental illness. The word *pica* is derived from the Latin word for magpie, a species of bird that feeds on whatever it encounters.

The mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders****, fourth edition, text revision (2000), which is abbreviated as* DSM-IV-TR, classifies pica under the heading of "Feeding and Eating Disorders of Infancy or Early Childhood." A **diagnosis** of pica requires that the patient must persist in eating nonfood substances for at least one month. This behavior must be inappropriate for the child's stage of development. Further, it must not be approved or encouraged by the child's culture.

Causes and symptoms

Causes

The cause of pica is not known. Many hypotheses have been developed to explain the behavior. These have included a variety of such factors as cultural influences; low socioeconomic status; deficiency diseases; and psychological disorders.

Malnutrition is often diagnosed at the same time as pica. A causal link has not been established. Eating clay has been associated with iron deficiency; however,

whether decreased iron absorption is caused by eating clay or whether iron deficiency prompts people to eat clay is not known. Some cultural groups are said to teach youngsters to eat clay. Persons with iron deficiency anemia have also been reported to chew on ice cubes. Again, the mechanism or causal link is not known.

Eating paint is most common among children from families of low socioeconomic status. It is often associated with lack of parental supervision. Hunger also may result in pica.

Among persons with mental retardation, pica has been explained as the result of an inability to tell the difference between food and nonfood items. This explanation, however, is not supported by examples of nonfood items that were deliberately selected and eaten by persons with limited mental faculties.

Pica, iron deficiency, and a number of other physiological disturbances in humans have been associated with decreased activity of the dopamine system in the **brain**. Dopamine is a neurotransmitter, or chemical that helps to relay the transmission of nerve impulses from one nerve cell to another. This association has led some researchers to think that there may be a connection between abnormally low levels of dopamine in the brain and the development of pica. No specific underlying biochemical disorders have been identified, however.

Risk factors for pica include the following:

- parental/child psychopathology
- family disorganization
- environmental deprivation
- pregnancy
- epilepsy
- brain damage
- mental retardation
- pervasive developmental disorders

Symptoms

Infants and children diagnosed with pica commonly eat paint, plaster, string, hair, and cloth. Older children may eat animal droppings, sand, insects, leaves, pebbles and cigarette butts. Adolescents and adults most often ingest clay or soil.

The symptoms of pica vary with the item ingested.

- Sand or soil is associated with gastric pain and occasional bleeding.
- Chewing ice may cause abnormal wear on teeth.
- Eating clay may cause constipation.
- Swallowing metal objects may lead to bowel perforation.
- Eating fecal material often leads to such infectious diseases as toxocariasis, toxoplasmosis, and trichuriasis.
- Consuming lead can lead to kidney damage and mental retardation.

Demographics

Pica tends to taper off as children grow older. The disorder occasionally continues into adolescence but is rarely observed in adults who are not disabled.

Pica is observed more commonly during the second and third years of life and is considered to be developmentally inappropriate in children older than 18–24 months. Research findings indicate that the disorder occurs in 25%–33% of young children and 20% of children in mental health clinics. Among individuals with mental retardation, pica occurs most often in those between the ages of 10–20 years. Among young pregnant women, the onset of pica is frequently associated with a first pregnancy in late adolescence or early adulthood. Although pica usually stops at the end of the pregnancy, it may continue intermittently for years.

Pica usually occurs with equal frequency among males and females. It is relatively uncommon, however, among adolescent and adult males of average intelligence who live in developed countries.

Diagnosis

Pica is often diagnosed in a hospital emergency room, when the child or adolescent develops symptoms of lead poisoning, bowel perforation, or other medical complications caused by the nonfood items that have been swallowed. Laboratory studies may be used to assess these complications. The choice of imaging or laboratory studies depends on the characteristics of the ingested materials and the resultant medical problems.

The examining doctor may order a variety of **imaging studies** in order to identify the ingested materials and treat the gastrointestinal complications of pica. These imaging studies may include the following:

- abdominal x rays
- barium examinations of the upper and lower gastrointestinal (GI) tracts
- upper GI endoscopy to diagnose the formation of bezoars (solid masses formed in the stomach) or to identify associated injuries to the digestive tract

Films and studies may be repeated at regular intervals to track changes in the location of ingested materials.

Treatments

As of 2002, there is no standard treatment for pica. Currently, the most effective strategies are based on **behavior modification**, but even these treatments have achieved limited success. Pica associated with a nutritional deficiency often clears up when the missing nutrient is added to the patient's diet.

Few studies have examined the efficacy of drug treatments for pica. Ongoing research, however, is exploring the relationship between pica and abnormally low levels of the neurotransmitter dopamine. This line of research may help to identify new medications for the treatment of pica. There is some evidence that medications used to manage severe behavioral problems in children may be useful in treating coexisting pica.

Lead poisoning resulting from pica may be treated by chelating medications, which are drugs that remove lead or other heavy metals from the bloodstream. The two medications most often given for lead poisoning are dimercaprol, which is also known as BAL or British Anti-Lewisite; and edetate calcium disodium (EDTA). A medical toxicologist (a doctor who specializes in treating poisoning cases) may be consulted regarding children's dosages of these drugs.

In some cases, surgery may be required to remove metal objects from the patient's digestive tract or to repair tissue injuries. It is particularly important to remove any objects made of lead (fishing weights, lead shot, pieces of printer's type, etc.) as quickly as possible because of the danger of lead poisoning.

Prognosis

Pica frequently ends spontaneously in young children and pregnant women. Untreated pica, however, may persist for years, especially in persons with mental retardation and developmental disabilities.

Prevention

There is no known way to prevent pica at the present time. Educating people, particularly young couples with children, about healthy nutritional practices is the best preventive strategy.

Resources

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org>.

American Academy of Pediatrics. 141 Northwest Point Boulevard, Elk Grove Village, IL 60007-1098. Telephone: (847) 434-4000. Fax: (847) 434-8000. Web site: <http://www.aap.org/default.htm>.

American College of Physicians, 190 N Independence Mall West, Philadelphia, PA 19106-1572, Phone: (800) 523-1546, x2600 or (215) 351-2600, Web site: <http://www.acponline.org>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org>.

OTHER

Anorexia Nervosa and Related Eating Disorders, Inc.: <http://www.anred.com/pica.html>.

Support, Concern and Resources For Eating Disorders: <http://www.eating-disorder.org/pica.html>.

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Pimozide

Definition

Pimozide is an atypical antipsychotic drug used to treat serious motor and verbal tics associated with Tourette's syndrome. It is sold under the brand name Orap.

KEY TERMS

Parkinsonian—Related to symptoms associated with Parkinson's disease, a nervous system disorder characterized by abnormal muscle movement of the tongue, face, and neck, inability to walk or move quickly, walking in a shuffling manner, restlessness, and/or tremors.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tic—A sudden involuntary behavior that is difficult or impossible for the person to suppress. Tics may be either motor (related to movement) or vocal, and may become more pronounced under stress.

Tourette syndrome—Neurological disorder characterized by multiple involuntary movements and uncontrollable vocalizations called tics that come and go over years, usually beginning in childhood and becoming chronic. Sometimes the tics include inappropriate language.

Purpose

Pimozide is classified as an atypical antipsychotic drug. It is structurally similar to another drug, **haloperidol**, which was the first drug to be used in Tourette's syndrome. Pimozide is most often used to treat symptoms of Tourette's syndrome, although it has also been used for treating **schizophrenia** mania, and other behavioral disorders.

Description

Excess dopamine activity in the **brain** is associated with the verbal and physical tics observed in Tourette's syndrome. Like haloperidol, pimozide is believed to inhibit the actions of the brain chemical, dopamine.

Pimozide is broken down by the liver and eliminated from the body by the kidneys. Because pimozide is associated with health risks, it should not be used for tics that are simply annoying or cosmetic. Pimozide should be used only in patients with severe symptoms after other drug therapy has been tried and failed.

Pimozide is available in 1-mg and 2-mg tablets.

Recommended dosage

The common starting dose of pimozide in adults is 1-2 mg per day. The dose may be increased every other day until 0.2 mg per kg (or 0.9 mg per pound) of body weight per day or 10 mg per day is reached, whichever is less. Doses that exceed 0.2 mg per kg per day or 10 mg daily are not recommended.

In children, the usual initial dose is 0.05 mg per kg daily, and increased every three days to a maximum dose of 0.2 mg per kg (or 10 mg) per day.

Periodically, the dosage of pimozide should be reduced to determine if tics are still present. Patients should be maintained on the lowest dose that is effective in treating their disorder.

Precautions

Pimozide may alter the rhythm of the heart. As a result, it should be used with caution in people with heart disease, and these patients should be observed carefully while receiving the drug.

Pimozide should not be taken with grapefruit juice.

Pimozide should be used with close physician supervision by people who have a history of seizure disorders, because it may increase the tendency to have **seizures**.

Pimozide may cause extreme drowsiness and should be used carefully by people who need to be mentally alert.

Patients should not take pimozide while pregnant or breast-feeding.

Pimozide should not be used by people with mild tics, by individuals taking stimulants such as **methylphenidate** (Ritalin), **pemoline** (Cylert), or dextroamphetamine (Dexedrine) since these drugs may cause tics.

Side effects

The most common side effects associated with pimozide are sleepiness, headache, stomach upset, muscle tightness, muscle weakness, difficulty moving, tremor, abnormal behavior, visual disturbances, and impotence.

Other side effects that might also occur with pimozide involve rapid heart rates or irregular heart rhythms, low blood pressure, constipation, dry mouth and eyes, rash, breast pain, breast milk production, loss of bladder control, or low blood cell counts.

Pimozide use may lead to the development of symptoms that resemble Parkinson's disease. These symptoms may include a tight or mask-like expression on the face,

drooling, tremors, pill-rolling motions in the hands, cogwheel rigidity (abnormal rigidity in muscles characterized by jerky movements when the muscle is passively stretched), and a shuffling gait. Taking anti-Parkinson drugs **benztropine** mesylate or **trihexyphenidyl** hydrochloride along with the pimozide usually controls these symptoms.

Pimozide has the potential to produce a serious side effect called **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements that may appear late in therapy and not disappear even after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage of pimozide. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period.

An occasionally reported side effect of pimozide is neuroleptic malignant syndrome. This is a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia (fast heartbeat), and arrhythmias (irregular heartbeat). People who think they may be experiencing any side effects from this or any other medication should talk to their physician promptly.

Interactions

If pimozide is used with bethanechol (Urecholine), **clonidine** (Catapres), **fluoxetine** (Prozac), indomethacin (Indocin), meperidine (Demerol), **paroxetine** (Paxil), quinidine, or **trazodone** (Desyrel), the side effects associated with pimozide may be increased.

There is an increased risk of irregular heart rhythms if pimozide is used with other antipsychotics, certain antidepressants, some heart drugs, and antibiotics like erythromycin.

The beneficial effects of pimozide may be reduced if used with bromocriptine (Parlodel), **carbamazepine** (Tegretol), levodopa (Larodopa, Sinemet), lithium, or phenobarbital.

Some antibiotics, antifungals, antidepressants, and drugs used for AIDS may prevent the breakdown of pimozide by the liver and thus, increase the amount of pimozide in the body. The combination of pimozide and the above classes of drugs should be used cautiously if at all.

Pimozide may interact with other central nervous system depressants such as alcohol, sleeping pills, antihistamines, and antidepressants.

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Play therapy

Definition

Play therapy refers to a method of **psychotherapy** with children in which a therapist uses a child's fantasies and the symbolic meanings of his or her play as a medium for understanding and communication with the child.

Purpose

The aim of play therapy is to decrease those behavioral and emotional difficulties that interfere significantly with a child's normal functioning. Inherent in this aim is improved communication and understanding between the child and his parents. Less obvious goals include improved verbal expression, ability for self-observation, improved impulse control, more adaptive ways of coping with anxiety and frustration, and improved capacity to trust and to relate to others. In this type of treatment, the therapist uses an understanding of cognitive development and of the different stages of emotional development as well as the conflicts common to these stages when treating the child.

Play therapy is used to treat problems that are interfering with the child's normal development. Such difficulties would be extreme in degree and have been occurring for many months without resolution. Reasons for treatment include, but are not limited to, temper tantrums, aggressive behavior, non-medical problems with bowel or bladder control, difficulties with sleeping or having nightmares, and experiencing worries or fears. This type of treatment is also used with children who have experienced sexual or physical **abuse**, **neglect**, the loss of a family

Children communicate their thoughts and feelings through play more naturally than they do through verbal communication. As the child plays, the therapist begins to recognize themes and patterns or ways of using the materials that are important to the child. Over time, the clinician helps the child begin to make meaning out of the play. *(S. Villeger/Explorer. Photo Researchers, Inc. Reproduced by permission.)*

member, medical illness, physical injury, or any experience that is traumatic.

At times, children in play therapy will also receive other types of treatment. For instance, youngsters who are unable to control their attention, impulses, tendency to react with violence, or who experience severe anxiety may take medication for these symptoms while participating in play therapy. The play therapy would address the child's psychological symptoms. Other situations of dual treatment include children with **learning disorders**. These youngsters may receive play therapy to alleviate feelings of low self-esteem, excessive worry, helplessness, and incompetency that are related to their learning problems and academic struggles. In addition, they should receive a special type of tutoring called **cognitive remediation**, which addresses the specific learning issues.

Precautions

Play therapy addresses psychological issues and would not be used to alleviate medical or biological problems. Children who are experiencing physical problems should see a physician for a medical evaluation to clarify the nature of the problem and, if necessary, receive the appropriate medical treatment. Likewise, children who experience academic difficulties need to receive a neuropsychological or in-depth psychological evaluation in order to clarify the presence of a biologically based learning disability. In both of these cases, psychological problems may be present in addition to medical ailments and learning disabilities, but they may not be the primary problem and it would not be sufficient to treat only the psychological issues. Alternatively, evaluations may show that medical or biological causes are not evident, and this would be important information for the parents and therapist to know.

Description

In play therapy, the clinician meets with the child alone for the majority of the sessions and arranges times to meet with parents separately or with the child, depending on the situation. The structure of the sessions is maintained in a consistent manner in order to provide a feeling of safety and stability for the child and parents. Sessions are scheduled for the same day and time each week and occur for the same duration. The frequency of sessions is typically one or two times per week, and meetings with parents occur about two times per month, with some variation. The session length will vary depending on the environment. For example, in private settings, sessions usually last 45 to 50 minutes while in hospitals and mental health clinics the duration is typically 30 minutes. The number of sessions and duration of treatment varies according to treatment objectives of the child.

During the initial meeting with parents, the therapist will want to learn as much as possible about the nature of the child's problems. Parents will be asked for information about the child's developmental, medical, social and school history, whether or not previous evaluations and interventions were attempted and the nature of the results. Background information about parents is also important since it provides the therapist with a larger context from which to understand the child. This process of gathering information may take one to three sessions, depending on the style of the therapist. Some clinicians gather the important aspects of the child's history during the first meeting with parents and will continue to ask relevant questions during subsequent meetings. The clinician also learns important information during the initial sessions with the child.

Sessions with parents are important opportunities to keep the therapist informed about the child's current functioning at home and at school and for the therapist to offer

some insight and guidance to parents. At times, the clinician will provide suggestions about parenting techniques, about alternative ways to communicate with their child, and will also serve as a resource for information about child development. Details of child sessions are not routinely discussed with parents. If the child's privacy is maintained, it promotes free expression in the therapist's office and engenders a sense of trust in the therapist. Therapists will, instead, communicate to the parents their understanding of the child's psychological needs or conflicts.

For the purposes of explanation, treatment can be described as occurring in a series of initial, middle and final stages. The initial phase includes evaluation of the problem and teaching both child and parents about the process of therapy. The middle phase is the period in which the child has become familiar with the treatment process and comfortable with the therapist. The therapist is continuing to evaluate and learn about the child, but has a clearer sense of the youngster's issues and has developed, with the child, a means for the two to communicate. The final phase includes the process of ending treatment and saying goodbye to the therapist.

During the early sessions, the therapist talks with the child about the reason the youngster was brought in for treatment and explains that the therapist helps make children's problems go away. Youngsters often deny experiencing any problems. It is not necessary for them to acknowledge having any since they may be unable to do so due to normal cognitive and emotional factors or because they are simply not experiencing any problems. The child is informed about the nature of the sessions. Specifically, the child is informed that he or she can say or play or do anything desired while in the office as long as no one gets hurt, and that what is said and done in the office will be kept private unless the child is in danger of harming himself.

Children communicate their thoughts and feelings through play more naturally than they do through verbal communication. As the child plays, the therapist begins to recognize themes and patterns or ways of using the materials that are important to the child. Over time, the clinician helps the child begin to make meaning out of the play. This is important because the play reflects issues which are important to the child and typically relevant to their difficulties.

When the child's symptoms have subsided for a stable period of time and when functioning is adequate with peers and adults at home, in school, and in extracurricular activities, the focus of treatment will shift away from problems and onto the process of saying goodbye. This last stage is known as the termination phase of treatment and it is reflective of the ongoing change and loss that human beings experience throughout their lives. Since this type of therapy relies heavily on the therapist's relationship with the child and also with parents, ending therapy will signify a change and a loss for all involved, but for the child in particular. In keeping with the therapeutic process of communicating thoughts and feelings, this stage is an opportunity for the child to work through how they feel about ending therapy and about leaving the therapist. In addition to allowing for a sense of closure, it also makes it less likely that the youngster will misconstrue the ending of treatment as a rejection by the therapist, which would taint the larger experience of therapy for the child. Parents also need a sense of closure and are usually encouraged to process the treatment experience with the therapist. The therapist also appreciates the opportunity to say goodbye to the parents and child after having become involved in their lives in this important way, and it is often beneficial for parents and children to hear the clinician's thoughts and feelings with regards to ending treatment.

Preparation

It is recommended that parents explain to the child that they will be going to see a therapist, that they discuss, if possible, the particular problem that is interfering with the child's growth and that a therapist is going to teach both parents and child how to make things better. As described earlier, the child may deny even obvious problems, but mainly just needs to agree to meet the therapist and to see what therapy is like.

Aftercare

Children sometimes return to therapy for additional sessions when they experience a setback that cannot be easily resolved.

Normal results

Normal results include the significant reduction or disappearance of the main problems for which the child was initially seen. The child should also be functioning adequately at home, in school, with peers and should be able to participate in and enjoy extracurricular activities.

Abnormal results

Sometimes play therapy does not alleviate the child's symptoms. This situation can occur if the child is extremely resistant and refuses to participate in treatment or if the child's ways of coping are so rigidly held that it is not possible for them to learn more adaptive ones.

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ORGANIZATIONS

American Psychological Association. 750 First Street, NE, Washington D.C. 20002. < http://www.apa.org>.

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KEY TERMS

Apnea—A brief suspension or interruption of breathing.

Arrhythmia—Any disturbance in the normal rhythm of the heartbeat.

Bruxism—Habitual, often unconscious, grinding of the teeth.

Hypopnea—Breathing that is too shallow to maintain adequate levels of oxygen in the blood.

Narcolepsy—A disorder characterized by frequent and uncontrollable attacks of deep sleep.

Oximetry—The measurement of blood oxygen levels.

Parameter—A characteristic or factor that is measured during a test of a complex process or activity like sleep.

Parasomnia—A type of sleep disorder characterized by abnormal changes in behavior or body functions during sleep, specific stages of sleep, or the transition between sleeping and waking.

Thermistor—An electrical device whose resistance decreases with rises in temperature.

Polysomnography

Definition

Polysomnography is a series of tests performed on patients while they sleep. Polysomnography is a comprehensive overnight procedure that evaluates **sleep disorders**. It generally includes monitoring of the patient's airflow through the nose and mouth, blood pressure, heartbeat as measured by an electrocardiograph, blood oxygen level, **brain** wave patterns, eye movements, and the movements of respiratory muscles and limbs. The word *polysomnography* is derived from the Greek root *poly* meaning "many," the Latin noun *somnus* meaning "sleep," and the Greek verb *graphein* meaning "to write."

Purpose

Polysomnography is used to help diagnose and evaluate a number of sleep disorders. For instance, it can help diagnose sleep apnea, a common disorder in middle-aged and elderly obese men, in which the muscles of the soft palate in the back of the throat relax and close off the airway during sleep. Sleep apnea may cause the person to snore loudly and gasp for air at night. It may also cause the person to be excessively drowsy and likely to fall asleep during the day. Another syndrome often uncovered by polysomnography is **narcolepsy**. Persons with narcolepsy have sudden attacks of sleep and/or cataplexy (temporary loss of muscle tone caused by moments of emotion, such as fear, anger, or surprise, which causes people to slump or fall over), sleep paralysis or **hallucinations** while they are falling asleep.

Polysomnography is often used to evaluate such parasomnias (abnormal behaviors or movements during sleep) as sleepwalking; talking in one's sleep; nightmares; and bed-wetting (**enuresis**). It can also be used to detect or evaluate **seizures** that occur in the middle of the night, when the patient and his or her family are unlikely to be aware of them.

Other problems uncovered by polysomnography include sleep-related psychiatric depression, asthma, and **panic disorder**. Polysomnography is generally not used if the sleep disorder has been clearly identified by the treating physician. It is also not used in cases of **insomnia** that have simple and obvious causes.

Precautions

Polysomnography is extremely safe, and no special precautions need to be taken.

Description

Polysomnography requires an overnight stay in a sleep laboratory. While the patient sleeps, he or she is monitored in a number of ways that can provide useful information.

A polysomnograph collects data on the electrical activity of the heart and brain, and muscle activity in the face and neck of a sleeping subject. *(Photograph by Philippe Plailly. Science Source/ Photo Researchers, Inc. Reproduced by permission.)*

One form of monitoring is **electroencephalography** (EEG), which involves the attachment of electrodes to the patient's scalp to record his or her brain wave activity. The electroencephalograph records brain wave activity from different parts of the brain and charts them on a graph. The EEG not only helps doctors establish what stage of sleep the patient is in, but may also detect seizures.

Another form of monitoring is continuous electrooculography (EOG), which records eye movements. EOG is used to determine the time periods during which the patient is going through a stage of sleep called rapid-eye-movement (REM) sleep. Both EEG and EOG can be helpful in determining sleep latency (the time period between getting into bed and the onset of sleep); total sleep time; the time spent in each sleep stage; and the number of arousals from sleep.

The airflow through the patient's nose and mouth are measured by heat-sensitive devices called thermistors. The thermistors can help detect episodes of apnea (stopped breathing), or hypopnea (inadequate or too-shallow breathing). Another test called pulse oximetry measures the amount of oxygen in the patient's blood. Pulse oximetry can be used to assess the degree of oxygen starvation during episodes of hypopnea or apnea.

The electrical activity of the patient's heart is also measured on an electrocardiogram, or EKG. Electrodes are attached to the patient's chest. The electrodes pick up electrical activity from various areas of the heart. They help to detect cardiac arrythmias (abnormal heart rhythms), which may occur during periods of sleep apnea. The patient's blood pressure is also measured, because some episodes of sleep apnea can raise blood pressure to dangerously high levels.

In some cases, sleep laboratories monitor the movement of the patient's arms and legs during sleep. This measurement can be helpful in detecting such sleep disorders as periodic limb movements. Some sleep laboratories perform an additional test called multiple sleep latency testing (MSLT), which records several naps throughout the day. In addition, many sleep researchers prefer to evaluate the patient over a period of a few days rather than just one night. This approach is based on the recognition that the patient may need more than one night to adjust to the unfamiliar surroundings of the sleep laboratory.

Preparation

The patient may be asked to discontinue taking any medications, and avoid alcohol and strenuous exercise the day before the sleep analysis is performed. Before the patient goes to sleep, the technician hooks him or her up to all of the monitors being used.

Aftercare

After the test is completed, the monitors are detached from the patient. No special measures need to be taken after polysomnography. On occasion, skin irritation from the adhesive can develop in the areas where the electrodes have been attached to the patient.

Normal results

A normal result in polysomnography shows normal results for all parameters (EEG, EKG, blood pressure, eye movement, air flow, pulse oximetry, etc.) that were monitored throughout all stages of sleep.

Abnormal results

Polysomnography may yield a number of abnormal results, indicating one or more potential sleep disorders. For instance, abnormal transitions into and out of various stages of sleep, as documented by the EEG and the EOG, may be signs of narcolepsy. Reduced air flow through the nose and mouth, along with a fall in blood oxygen levels, may indicate apnea or hypopnea. If apnea is accompanied by abnormal patterns on the EKG or elevations in blood pressure, then the sleep apnea may be producing harmful effects. Frequent movements of the patient's arms and legs may suggest a sleep disorder called periodic limb movement. A related condition that affects sleep as well as daytime movement is called restless legs syndrome. Polysomnography can also be used to diagnose bruxism, which is the chronic grinding of the teeth during sleep.

See also Breathing-related sleep disorder

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American Sleep Disorders Association. 6301 Bandel Road NW, Suite 101, Rochester, MN 55901. <http://www.asda.org>.

National Heart, Lung and Blood Institute. P.O. Box 30105, Bethesda, MD 20824-0105. (301) 251-1222. <http://www.nhlbi.nih.gov>.

Robert Scott Dinsmoor

Polysubstance dependence

Definition

Polysubstance dependence refers to a type of substance dependence disorder in which an individual uses at least three different classes of substances indiscriminately and does not have a favorite drug that qualifies for dependence on its own.

Description

Polysubstance dependence is listed as a substance disorder in the ***Diagnostic and Statistical Manual of Mental Disorders*** published in 2000 (also known as the *DSM-IV-TR*). The *DSM-IV-TR* is the latest revision of the manual that it is used by mental health professionals to diagnose mental disorders. When an individual meets criteria for dependence on a group of substances (at least three different types used in the same 12-month period) he or she is given the **diagnosis** of polysubstance dependence. For example, an individual may use cocaine, sedatives, and hallucinogens indiscriminately (i.e., no single drug predominated; there was no "drug of choice") for a year or more. The individual may not meet criteria for cocaine dependence, sedative dependence, or hallucinogen dependence, but may meet criteria for substance dependence when all three drugs are considered as a group.

Causes and symptoms

Causes

There is very little documented regarding the causes of polysubstance dependence.

Symptoms

The *DSM-IV-TR* specifies that three or more of the following symptoms must occur at any time during a 12-month period (and cause significant impairment or distress) in order to meet diagnostic criteria for substance dependence:

- Tolerance: The individual either has to use increasingly higher amounts of the drugs over time in order to achieve the same drug effect or finds that the same amount of the drug has much less of an effect over time than before. After using several different drugs regularly for a while, an individual may find that he or she needs to use at least 50% more of the amount they began using in order to get the same effect.
- Withdrawal: The individual either experiences the withdrawal symptoms when he or she stops using the drugs or the individual uses drugs in order to avoid or relieve withdrawal symptoms.
- Loss of control: The individual either repeatedly uses more drugs than planned or uses the drugs over longer periods of time than planned. For instance, an individual may begin using drugs (any combination of three or more types of drugs) on weekdays in addition to weekends.
- Inability to stop using: The individual has either unsuccessfully attempted to cut down or stop using the drugs or has a persistent desire to stop using. An individual may find that, despite efforts to stop using drugs on weekdays, he or she is unable to do so.
- Time: The individual spends a lot of time obtaining drugs, using drugs, being under the influence of drugs, and recovering from the effects of drugs.
- Interference with activities: The individual either gives up or reduces the amount of time involved in recreational activities, social activities, and/or occupational activities because of the use of drugs. An individual may use drugs instead of engaging in hobbies, spending time with friends, or going to work.
- Harm to self: The individual continues to use drugs despite having either a physical or psychological problem that is caused by or made worse by the use of drugs.

Demographics

Young adults (i.e., between the ages of 18 and 24) have the highest rates of use for all substances. Generally, males tend to be diagnosed with more substance use disorders.

Diagnosis

Individuals who abuse alcohol and other drugs usually meet criteria for substance abuse and/or dependence for each individual substance used. Multiple diagnoses are given in this situation (cocaine dependence, hallucinogen dependence, and sedative dependence, for example). Polysubstance dependence is reserved only for those situations when an individual uses multiple substances indiscriminately and meets criteria for dependence on these substances, taken as a whole.

Treatments

There is very little documented regarding the treatment of polysubstance dependence. However, several treatments have been tried. Psychological evaluation and tests may be used to assess the affected individual. The person may be admitted into a hospital or treatment center as an inpatient, and/or he or she may receive **cognitive-behavioral therapy**.

Prognosis

The course of substance dependence varies from short-lived episodes to chronic episodes lasting years. The individual with substance dependence may alternate between periods of heavy use with severe problems, periods of no use at all, and periods of use with few problems.

Prevention

The best single thing an individual can do to prevent polysubstance dependence is to avoid using drugs including alcohol altogether. On a larger scale, comprehensive prevention programs that utilize family, schools, communities, and the media (such as television) can be effective in reducing substance abuse.

Resources

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Positive symptoms

Definition

Positive symptoms are thoughts, behaviors, or sensory perceptions present in a person with a mental disorder, but not present in people in the normal population.

Description

Examples of positive symptoms are **hallucinations** (seeing, hearing, or smelling things not really there), **delusions** (belief in ideas not based on reality), disorganized speech (loose association between ideas, derailment of sentences, incoherence, illogical statements, excessive detail, and rhyming of words), or bizarre behavior. In other disorders, positive symptoms are primarily associated with **schizophrenia** or **psychosis**.

See also Negative symptoms

Sandra L. Friedrich, M.A.

Positron emission tomography

Definition

Positron emission tomography (PET) is a highly specialized imaging technique using short-lived radiolabeled substances to produce extremely high resolution images of the body's biological function.

Purpose

Besides being used to investigate the metabolism of normal organs, PET has also become the technique of choice to investigate various neurological diseases, including **stroke**, epilepsy, **Alzheimer's disease**, Parkinson's disease, and Huntington's disease. Various psychiatric disorders, such as **schizophrenia**, depression, **obsessive-compulsive disorder**, **attention-deficit/hyperactivity disorder**, and Tourette syndrome, are also imaged by PET, because these disorders have changes in specific areas of the **brain**. Additionally, PET scanning is a powerful research tool to detect changes or abnormalities in areas that may be difficult to visualize using other radiological procedures. In the field of mental health, a PET scan may be used when a patient seeks medical help for symptoms that could possibly be caused by a brain tumor. These symptoms may include headaches, emotional abnormalities, or intellectual or memory problems. In these cases, a PET scan may be performed to "rule out" a tumor, so that other tests can be performed in order to establish an accurate **diagnosis**.

PET is especially utilized in persons affected by cancer because it can detect metastatic tumors that may not be visualized by other imaging techniques. It is also being increasingly used not only as a cancer diagnostic tool, but also to help researchers design the most beneficial therapies. For example, it may be used to assess response to chemotherapy. PET imaging is very accurate in differentiating malignant from benign cell growths, and in assessing the spread of malignant tumors. PET is also used to detect recurrent brain tumors and cancers of the lung, colon, breast, lymph nodes, skin, and other organs.

KEY TERMS

Benign growth—A noncancerous cell growth that does not metastasize and does not recur after treatment or removal.

Cancer screening—A procedure designed to detect cancer even though a person has no symptoms, usually performed using an imaging technique.

CT scan—An imaging technique that uses a computer to combine multiple x-ray images into a two-dimensional cross-sectional image.

Electron—One of the small particles that make up an atom. An electron has the same mass and amount of charge as a positron, but the electron has a negative charge.

Gamma ray—A high-energy photon, emitted by radioactive substances.

Half-life—The time required for half of the atoms in a radioactive substance to disintegrate.

Malignant growth—A cell growth or tumor that becomes progressively worse and that can metastasize elsewhere in the body.

Metabolism—The group of biochemical processes within the body that release energy in support of life.

MRI—Magnetic resonance imaging. A special imaging technique used to image internal parts of the body, especially soft tissues.

Photon—A light particle.

Positron—One of the small particles that make up an atom. A positron has the same mass and amount of charge as an electron, but the positron has a positive charge.

Precautions

In some cases, patients may be allergic to the radioactive agents used for PET. A patient with known

allergies should discuss this with their specialist before undergoing the PET scan.

Description

PET is used in conjunction with compounds that closely resemble a natural substance used by the body, such as a simple sugar (glucose, for example), labeled with a radioactive atom and injected into the patient. These compounds (radionuclides or radiopharmaceuticals) emit particles called positrons. As positrons emitted from the radionuclides encounter electrons in the body, they produce high-energy photons (gamma rays) that can be recorded as a signal by detectors surrounding the body. The radionuclides move through the body and accumulate in the organs targeted for examination. A computer collects the distribution of radioactivity and reassembles them into actual images.

By further defining a lesion seen on other imaging modalities, PET may enhance assessment of tumors exceedingly well. This is because of its operating principle. The radiolabeled sugars injected into the patient will be used by all body cells, but more sugar will be used by cells that have an increased metabolism. Cancer cells are highly metabolic, meaning that they use more sugar than healthy nearby cells, and they are easily seen on the PET scan. PET images thus show the chemical functioning of an organ or tissue, unlike x ray, **computed tomography**, or **magnetic resonance imaging**, which show only body structure.

Preparation

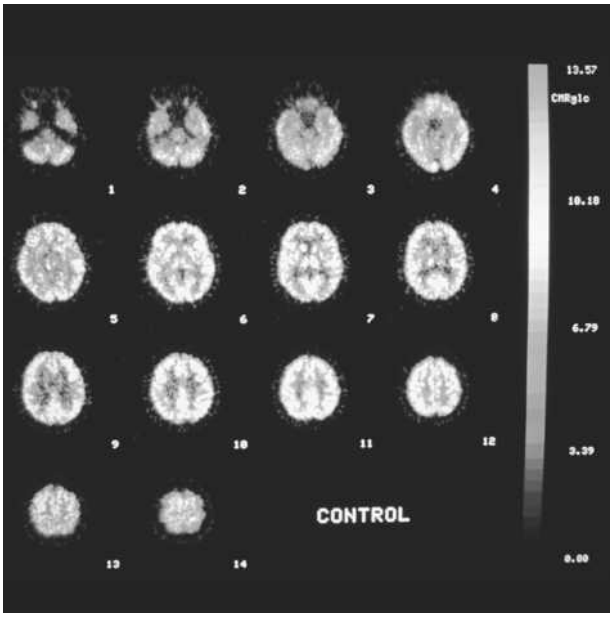
The radiopharmaceutical is given by intravenous injection or inhaled as a gas a few minutes before the PET procedure. How it is administered depends on the radiopharmaceutical used and which one is selected depends on what organ or body part is being scanned. During the scan, the patient lies comfortably; the only discomfort involved may be the pinprick of a needle used to inject the radiopharmaceutical.

Aftercare

No special aftercare measures are indicated for PET.

Risks

Some of radioactive compounds used for PET scanning can persist for a long time in the body. Even though only a small amount is injected each time, the long half-lives of these compounds can limit the number of times a patient can be scanned. However, PET is a relatively safe procedure. PET scans using radioactive fluorine result in patients receiving exposures comparable to (or less than) those from other medical procedures, such as the taking of x rays. Other scanning radiopharmaceuticals—for instance, 6-F-dopa or radioactive water—normally cause even less exposure.



A positron emission tomography (PET) scan of the human brain. *(Jon Meyer. Cutom Medical Stock Photo. Reproduced by permission.) See color insert for color version of photo.*

Normal results

The PET scan of a healthy organ or body part will yield images without contrasting regions, because the radiolabeled sugar will have been metabolized at the same rate.

Abnormal results

The PET scan of a diseased organ or body part, however, will yield images showing contrasting regions, because the radiolabeled sugar will not have been metabolized (breaking down a large molecule to a smaller molecule that can be used by the body) at the same rate by the healthy and diseased cells.

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Postpartum depression

Definition

Postpartum depression is a depression that can range from mild to suicidal and can occur anytime after delivery up to one year later.

Description

Postpartum depression is an affective disorder (any mental disorder characterized by a consistent change in mood that affects thoughts and behaviors) that can occur after pregnancies of all duration, from spontaneous (not induced) abortions, also called miscarriages, to full-term deliveries. The depression can take a mild clinical course or it can range to suicidal ideations (thoughts). The depression can occur anytime post-delivery to one year after delivery. Symptoms commonly start within four to six weeks after delivery. Differentiating postpartum depression from "maternity blues" or the **stress** from the pregnancy and delivery can be difficult. Postpartum depression can be differentiated from other types of depression if the mother exhibits signs of ambivalence to the infant and **neglect** of other family members.

Causes and symptoms

Causes

The cause of postpartum depression has been extensively studied. Alterations of hormone levels for prolactin, progesterone, estrogen, and cortisol are not significantly different from those of patients who do not suffer from postpartum depression. However, some research indicates a change in a **brain** chemical that controls the release of cortisol.

Research seems to indicate that postpartum depression is unlikely to occur in a patient with an otherwise psychologically uncomplicated pregnancy and past history. There is no association of postpartum depression with marital status, social class, or the number of live children born to the mother. However, there seems to be an increased chance to develop this disorder after pregnancy loss.

Certain characteristics have been associated with increased risk of developing postpartum depression. These risk factors include:

- medical indigence— being in need of health care and not being able to receive it, possibly due to lack of medical insurance
- being younger than 20 years old at time of delivery
- being unmarried
- having been separated from one or both parents in childhood or adolescence
- receiving poor parental support and attention in childhood
- having had limited parental support in adulthood
- poor relationship with husband or boyfriend
- economic problem with housing or income
- dissatisfaction with amount of education
- low self-esteem
- past or current emotional problem(s)
- family history of depression

Symptoms

The symptoms can range from mild depression to a severe depression with thoughts of ending one's life (**suicide**). The disorder should be suspected during its peak (four to six weeks after delivery) in a patient who demonstrates signs and symptoms of clinical depression (feelings of worthlessness and hopelessness, changes in eating and sleeping patterns, irritability, difficulty with motivation, and difficulty getting out of bed in the morning). Additionally, patients may be emotionally detached from the infant and unable to display loving affection towards family members. Physical and emotional stress during delivery in conjunction with great demands for infant care may cause the patient to neglect other family members, increasing the woman's feelings of self-worthlessness, isolation, and being trapped. Patients may also feel as if they are inadequate mothers, causing them guilt and embarrassment.

Demographics

There is a 20% to 30% risk of postpartum depression for women who had a previous depressive episode that was not associated with pregnancy. Additionally, there is an increased risk of recurrence in subsequent pregnancies since 50–100% of patients will have more than one episode.

Diagnosis

Patients should undergo careful clinical assessment from a **psychologist** or **psychiatrist**, who can determine the risk factors and diagnose the condition. A careful, comprehensive psychological assessment interview could reveal a previous depressive cycle or a family history of depression—important risk factors. The most widely used standard for **diagnosis** is the Edinburgh Postnatal Depression Scale (EPDS). This is a simple and short 10-question scale. A score of 12 or greater on the EPDS is considered high risk for postpartum depression.

Treatments

Treatment should begin as soon as the diagnosis is established. A typical treatment plan includes **psychotherapy** and medications. Recent studies have found that a group of medications known as the selective serotonin reuptake inhibitors (SSRIs) are effective in treating postpartum depression. These antidepressants have fewer side effects than other antidepressants and can be taken by breast-feeding mothers. SSRIs are secreted into breast milk, however, in varying amounts. Some studies indicate that **paroxetine** secretes the least amount of medication into breast milk. Breast-feeding women considering taking an antidepressant should discuss medication choices with their doctor. SSRIs can be given two to three weeks before delivery to patients who had a previous episode to avoid recurrence. Some SSRIs include: **fluoxetine** (Prozac), paroxetine (Paxil), **sertraline** (Zoloft), and **citalopram** (Celexa).

When medications are combined with psychological therapy, the rates for successful treatment are increased. **Interpersonal therapy** and **cognitive-behavioral therapy** have been found to be effective.

Prognosis

The prognosis for postpartum depression varies because this disorder is usually implicated with difficult social factors, a personal history of emotional problems, and adverse pregnancy outcomes, such as miscarriage. The prognosis is better if depression is detected early during its clinical course and a combination of SSRIs and psychotherapy is available and inititated.

Care for a newborn can be overwhelming, and some women experience postpartum depression shortly after birth. *(Layne Kennedy/CORBIS. Photo reproduced by permission.)*

Prevention

The best method to prevent the disorder is through education. Mothers should be advised prior to hospital discharge that if the "maternity blues" last longer than two weeks or pose tough difficulties with family interactions, they should call the hospital where their baby was delivered and pursue a referral for a psychological evaluation. Education concerning risk factors and reduction of these is important. Prophylactic (preventive) use of SSRIs is indicated two to three weeks before delivery to prevent the disorder in a patient with a past history of depression, since recurrence rates are high if the mother had a previous depressive episode.

Resources

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ORGANIZATIONS

Online PPD Support Group.
<http://www.ppdsupportpage.com>.

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Post-traumatic stress disorder

Definition

Post-traumatic stress disorder, often abbreviated as PTSD, is a complex disorder in which the affected person's memory, emotional responses, intellectual processes, and nervous system have all been disrupted by one or more traumatic experiences. It is sometimes summarized as "a normal reaction to abnormal events." The *DSM-IV-TR* (the professional's diagnostic manual) classifies PSTD as an anxiety disorder.

Description

PTSD has a unique position as the only psychiatric **diagnosis** (along with **acute stress disorder**) that depends on a factor outside the individual, namely, a traumatic stressor. A patient cannot be given a diagnosis of PTSD unless he or she has been exposed to an event that is considered traumatic. These events include such obvious traumas as rape, military combat, torture, genocide, natural disasters, and transportation or workplace disasters. In addition, it is now recognized that repeated traumas or such traumas of long duration as child **abuse**, domestic violence, stalking, cult membership, and hostage situations may also produce the symptoms of PTSD in survivors.

A person suffering from PTSD experiences flashbacks, nightmares, or daydreams in which the traumatic event is experienced again. The person may also experience abnormally intense startle responses, **insomnia**, and may have difficulty concentrating. Trauma survivors with PTSD have been effectively treated with **group therapy** or individual psychological therapy, and other therapies have helped individuals, as well. Some affected individuals have found **support groups** or peer counseling groups helpful. Treatment may require several years, and in some cases, PTSD may affect a person for the rest of his or her life.

Demographics

General United States population

PTSD is much more widespread in the general population than was thought when it was first introduced as a diagnostic category. The National Comorbidity Survey, a major epidemiological study conducted between 1990 and 1992, estimates that the lifetime prevalence among adult Americans is 7.8%, with women (10.4%) twice as likely as men (5%) to be diagnosed with PTSD at some point in their lives. These figures represent only a small proportion of adults who have experienced at least one traumatic event—60.7% of men and 51.2% of women respectively. More than 10% of the men and 6% of the women reported experiencing four or more types of trauma in their lives. The most frequently mentioned traumas are:

- witnessing someone being badly hurt or killed
- involvement in a fire, flood, earthquake, severe hurricane, or other natural disaster
- involvement in a life-threatening accident (workplace explosion or transportation accident)
- military combat

The traumatic events most frequently mentioned by men diagnosed with PTSD are rape, combat exposure, childhood **neglect**, and childhood physical abuse. For women diagnosed with PTSD, the most common traumas are rape, sexual molestation, physical attack, being threatened with a weapon, and childhood physical abuse.

High-risk populations

Some subpopulations in the United States are at greater risk of developing PTSD. The lifetime prevalence of PTSD among persons living in depressed urban areas or on Native American reservations is estimated at 23%. For victims of violent crimes, the estimated rate is 58%.

Military veterans

Information about PTSD in veterans of the Vietnam era is derived from the National Vietnam Veterans Readjustment Survey (NVVRS), conducted between 1986 and 1988. The estimated lifetime prevalence of PTSD among American veterans of this war is 30.9% for men and 26.9% for women. An additional 22.5% of the men and 21.2% of the women have been diagnosed with partial PTSD at some point in their lives. The lifetime prevalence of PTSD among veterans of World War II and the Korean War is estimated at 20%.

Cross-cultural issues

Further research needs to be done on the effects of ethnicity and culture on post-traumatic symptoms. As of 2001, most PTSD research has been done by Western clinicians working with patients from a similar background. Researchers do not yet know whether persons from non-Western societies have the same psychological reactions to specific traumas or whether they develop the same symptom patterns.

Causes and symptoms

Causes

When PTSD was first suggested as a diagnostic category for *DSM-III* in 1980, it was controversial precisely because of the central role of outside stressors as causes of the disorder. Psychiatry has generally emphasized the internal weaknesses or deficiencies of individuals as the source of mental disorders; prior to the 1970s, war veterans, rape victims, and other trauma survivors were often blamed for their symptoms and regarded as cowards, moral weaklings, or masochists. The high rate of psychiatric casualties among Vietnam veterans, however, led to studies conducted by the Veterans Administration. These studies helped to establish PTSD as a legitimate diagnostic entity with a complex set of causes.

BIOCHEMICAL/PHYSIOLOGICAL CAUSES. Present neurobiological research indicates that traumatic events cause lasting changes in the human nervous system, including abnormal secretions of stress hormones. In addition, in PTSD patients, researchers have found changes in the amygdala and the hippocampus—the parts of the **brain** that form links between fear and memory. Experiments with ketamine, a drug that inactivates one of the neurotransmitter chemicals in the central nervous system, suggest that trauma works in a similar way to damage associative pathways in the brain. **Positron emission tomography** (PET) scans of PTSD patients suggest that trauma affects the parts of the brain that govern speech and language.

SOCIOCULTURAL CAUSES. Studies of specific populations of PTSD patients (combat veterans, survivors of rape or genocide, former political hostages or prisoners, etc.) have shed light on the social and cultural causes of PTSD. In general, societies that are highly authoritarian, glorify violence, or sexualize violence have high rates of PTSD even among civilians.

OCCUPATIONAL FACTORS. Persons whose work exposes them to traumatic events or who treat trauma survivors may develop secondary PTSD (also known as compassion **fatigue** or burnout). These occupations include specialists in emergency medicine, police officers, firefighters, search-and-rescue personnel, psychotherapists, disaster investigators, etc. The degree of risk for PTSD is related to three factors: the amount and intensity of exposure to the suffering of trauma victims; the worker's degree of empathy and sensitivity; and unresolved issues from the worker's personal history.

PERSONAL VARIABLES. Although the most important causal factor in PTSD is the traumatic event itself, individuals differ in the intensity of their cognitive and emotional responses to trauma; some persons appear to be more vulnerable than others. In some cases, this greater vulnerability is related to temperament or natural disposition, with shy or introverted people being at greater risk. In other cases, the person's vulnerability results from chronic illness, a physical disability, or previous traumatization—particularly abuse in childhood. As of 2001, researchers have not found any correlation between race and biological vulnerability to PTSD.

KEY TERMS

Acute stress disorder—Symptoms occuring in an individual following a traumatic event to oneself or surrounding environment. Symptoms include a continued response of intense fear, helplessness, or terror within four weeks of the event, extreme nervousness, sleep disorders, increased anxiety, poor concentration, absence of emotional response to surroundings, and sometimes a dissociative amnesia—not recalling the significance of the trauma. Symptoms last a minimum of two days and maximum of four weeks. Can become post-traumatic stress disorder.

Adjustment disorder—A disorder defined by the development of significant emotional or behavioral symptoms in response to a stressful event or series of events. Symptoms may include depressed mood, anxiety, and impairment of social and occupational functioning.

Borderline personality disorder—A severe and usually life-long mental disorder characterized by violent mood swings and severe difficulties in sustaining interpersonal relationships.

Somatoform—Referring to physical symptoms with a psychological origin.

Substance abuse disorder—Disorder that is characterized by: an individual's need for more of a drug or alcohol than intended, an inability to stop using by choice, and an ongoing difficulty in recovering from the effects of the substance.

Symptoms

DSM-IV-TR specifies six diagnostic criteria for PTSD:

- Traumatic stressor: The patient has been exposed to a catastrophic event involving actual or threatened death or injury, or a threat to the physical integrity of the self or others. During exposure to the trauma, the person's emotional response was marked by intense fear, feelings of helplessness, or horror. In general, stressors caused intentionally by human beings (genocide, rape, torture, abuse, etc.) are experienced as more traumatic than accidents, natural disasters, or "acts of God."
- Intrusive symptoms: The patient experiences flashbacks, traumatic daydreams, or nightmares, in which he or she relives the trauma as if it were recurring in the present. Intrusive symptoms result from an abnormal process of memory formation. Traumatic memories have two distinctive characteristics: 1) they can be triggered by stimuli that remind the patient of the traumatic event; 2) they have a "frozen" or wordless quality, consisting of images and sensations rather than verbal descriptions.
- Avoidant symptoms: The patient attempts to reduce the possibility of exposure to anything that might trigger memories of the trauma, and to minimize his or her reactions to such memories. This cluster of symptoms includes feeling disconnected from other people, psychic numbing, and avoidance of places, persons, or things associated with the trauma. Patients with PTSD are at increased risk of substance abuse as a form of self-medication to numb painful memories.
- Hyperarousal: Hyperarousal is a condition in which the patient's nervous system is always on "red alert" for the return of danger. This symptom cluster includes hypervigilance, insomnia, difficulty concentrating, general irritability, and an extreme startle response. Some clinicians think that this abnormally intense startle response may be the most characteristic symptom of PTSD.
- Duration of symptoms: The symptoms must persist for at least one month.
- Significance: The patient suffers from significant social, interpersonal, or work-related problems as a result of the PTSD symptoms. A common social symptom of PTSD is a feeling of disconnection from other people (including loved ones), from the larger society, and from spiritual or other significant sources of meaning.

Diagnosis

The diagnosis of PTSD is complicated by several factors.

Time of onset/symptom duration

In the case of a known trauma of recent occurrence—most often a civilian disaster or war—the diagnosis of PTSD is relatively straightforward, based on the criteria listed above.

DSM-IV introduced a new diagnostic category, acute stress disorder, to differentiate between time-limited and longer-term stress reactions. In acute stress disorder, the hyperarousal and intrusive symptoms last between two days and four weeks. If the symptoms last beyond four weeks, and all of the above criteria are met, the diagnosis is changed to PTSD.

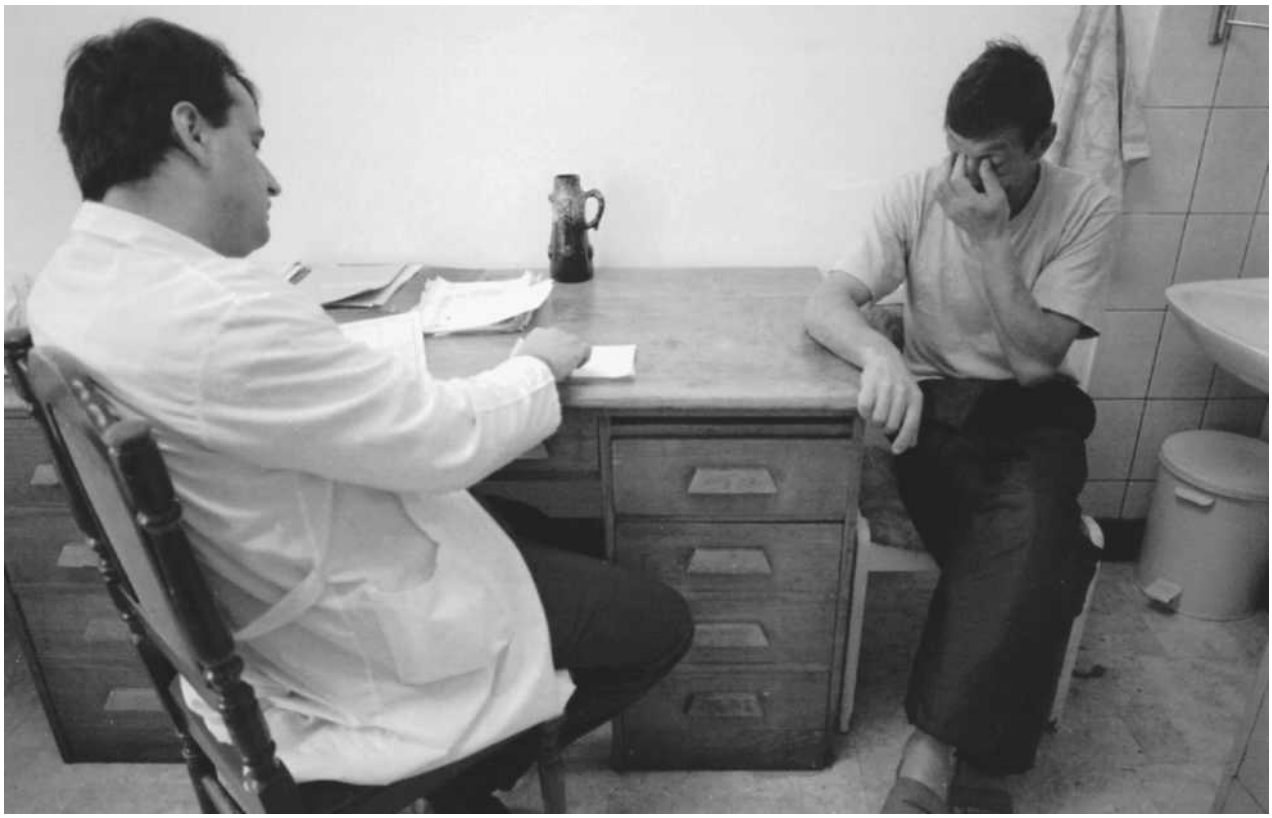
The diagnosis of PTSD is more difficult in cases of delayed reaction to trauma. Some individuals do not develop symptoms of PTSD until months or even years after the traumatic event. *DSM-IV-TR* specifies an interval of at least six months between the event and the development of symptoms for a diagnosis of PTSD with delayed onset. Delayed symptoms are often triggered by a situation that resembles the original trauma, as when a person raped in childhood experiences workplace sexual harassment.

Individual variations in response to stressors

DSM-III and its successors included the category of **adjustment disorder** to differentiate abnormal reactions to such painful but relatively common life events ("ordinary stressors") as divorce, job loss, or bereavement from symptoms resulting from overwhelming trauma. The differential diagnosis (the process of determining that the diagnosis is one disorder although it may resemble another) is complicated, however, by the fact that "ordinary stressors" sometimes reawaken unresolved childhood trauma, producing the delayed-reaction variant of PTSD.

Dual diagnoses

Most patients with PTSD (as many as 80%) have been diagnosed with one of the anxiety (30–60%), dissociative, mood (26–85%), or somatoform disorders as well as with PTSD. Between 40–60% of persons with delayed-reaction PTSD are diagnosed with a personality disorder, most often **borderline personality disorder**. Another common **dual diagnosis** is PTSD/substance abuse disorder. Between 60%–80% of patients who develop PTSD turn to alcohol or narcotics in order to avoid or numb painful memories. According to the NVVRS, the estimated lifetime prevalence of alcohol abuse among male Vietnam veterans is 39.2%, and the estimated lifetime prevalence of drug abuse is 5.7%. Dual diagnoses complicate treatment because the thera-



A Bosnian man with post-traumatic stress disorder talks with a therapist. *(AP/Wide World Photos. Reproduced by permission.)*

pist must decide whether to treat the disorders in sequence or concurrently. PTSD patients diagnosed with **personality disorders** are regarded as the most difficult to treat.

Psychological measures

As of 2002, there are no physical tests to establish a diagnosis of PTSD. The diagnosis is usually made on the basis of the patient's history and results from one or more short-answer interviews or symptom inventories. The instruments most often used to evaluate patients for PTSD include the Anxiety Disorders Interview Scale (ADIS), the **Beck Depression Inventory**, the Clinician-Administered PTSD Scale (CAPS), the Disorders of Extreme Stress Inventory (DESI), the Dissociative Experiences Scale (DES), the **Hamilton Anxiety Scale**, and the Impact of Event Scale (IES).

Treatments

Psychological and social interventions

In general, there have been few well-controlled clinical trials of treatment options for PTSD, particularly for severely affected patients.

Critical incident stress debriefing (CISD) is a treatment offered to patients within 48 hours following a civilian disaster or war zone trauma. It is intended to weaken the acute symptoms of the trauma and to forestall the development of full-blown PTSD. CISD usually consists of four phases:

- description of the traumatic event
- sharing of survivors' emotional reactions to the event
- open discussion of symptoms caused by the event
- reassurance that the symptoms are normal responses to trauma, followed by discussion of coping strategies

Critical incident stress management is a system of interventions designed to help emergency/disaster response workers, public safety personnel, and therapists deal with stress reactions before they develop secondary PTSD.

Other mainstream treatment methods used with patients who have already developed PTSD include:

- **Cognitive-behavioral therapy**. There are two treatment approaches to PTSD included under this heading: exposure therapy, which seeks to desensitize the patient to reminders of the trauma; and anxiety management training, which teaches the patient strategies for reduc-

ing anxiety. These strategies may include relaxation training, **biofeedback**, **social skills training**, distraction techniques, or cognitive restructuring.

- **Psychodynamic psychotherapy**. This method helps the patient recover a sense of self and learn new coping strategies and ways to deal with intense emotions related to the trauma. Typically, it consists of three phases: 1) establishing a sense of safety for the patient; 2) exploring the trauma itself in depth; 3) helping the patient re-establish connections with family, friends, the wider society, and other sources of meaning.
- Discussion groups or peer-counseling groups. These groups are usually formed for survivors of specific traumas, such as combat, rape/incest, and natural disasters. They help patients to recognize that other survivors of the shared experience have had the same emotions and reacted to the trauma in similar ways. They appear to be especially beneficial for patients with guilt issues about their behavior during the trauma (such as submitting to rape to save one's life, or surviving the event when others did not).
- **Family therapy**. This form of treatment is recommended for PTSD patients whose family life has been affected by the PTSD symptoms.

Medications

In general, medications are used most often in patients with severe PTSD to treat the intrusive symptoms of the disorder as well as feelings of anxiety and depression. These drugs are usually given as one part of a treatment plan that includes **psychotherapy** or **group therapy**. As of 2002, there is no single medication that appears to be a "magic bullet" for PTSD. The selective serotonin reuptake inhibitors (SSRIs) appear to help the core symptoms when given in higher doses for five to eight weeks, while the tricyclic antidepressants (TCAs) or the monoamine oxidase inhibitors (MAOIs) are most useful in treating anxiety and depression.

Alternative therapies

Some alternative therapies for PTSD include:

- Spiritual/religious counseling. Because traumatic experiences often affect patients' spiritual views and beliefs, counseling with a trusted religious or spiritual advisor may be part of a treatment plan. A growing number of pastoral counselors in the major Christian and Jewish bodies have advanced credentials in trauma therapy.
- **Yoga** and various forms of bodywork are often recommended as ways of releasing physical tension or muscle soreness caused by anxiety or hypervigilance.
- Martial arts training can be helpful in restoring the patient's sense of personal effectiveness and safety. Some martial arts programs, such as Model Mugging, are designed especially for survivors of rape and other violent crimes.
- Art therapy, journaling, dance therapy, and creative writing groups offer safe outlets for the strong emotions that follow traumatic experiences.

Recent controversial therapies

Since the mid-1980s, several controversial methods of treatment for PTSD have been introduced. Some have been developed by mainstream medical researchers while others are derived from various forms of alternative medicine. They include:

- Eye Movement Desensitization and Reprocessing. This is a technique in which the patient reimagines the trauma while focusing visually on movements of the therapist's finger. It is claimed that the movements of the patient's eyes reprogram the brain and allow emotional healing.
- Tapas Acupressure Technique (TAT). TAT was derived from traditional Chinese medicine (TCM), and its practitioners maintain that a large number of **acupuncture** meridians enter the brain at certain points on the face, especially around the eyes. Pressure on these points is thought to release traumatic stress.
- Thought Field Therapy. This therapy combines the acupuncture meridians of TCM with analysis of the patient's voice over the telephone. The therapist then provides an individualized treatment for the patient.
- Traumatic Incident Reduction. This is a technique in which the patient treats the trauma like a videotape and "runs through" it repeatedly with the therapist until all negative emotions have been discharged.
- Emotional Freedom Techniques (EFT). EFT is similar to TAT in that it uses the body's acupuncture meridians, but it emphasizes the body's entire "energy field" rather than just the face.
- Counting Technique. Developed by a physician, this treatment consists of a preparation phase, a counting phase in which the therapist counts from 1 to 100 while the patient reimagines the trauma, and a review phase. Like Traumatic Incident Reduction, it is intended to reduce the patient's hyperarousal.

Prognosis

Trauma survivors who receive critical incident stress debriefing as soon as possible after the event have the best prognosis for full recovery. For patients who devel-

op full-blown PTSD, a combination of peer-group meetings and individual psychotherapy are often effective. Treatment may require several years, however, and the patient is likely to experience relapses.

There are no studies of untreated PTSD, but long-term studies of patients with delayed-reaction PTSD or delayed diagnosis of the disorder indicate that treatment of patients in these groups is much more difficult and complicated.

In some patients, PTSD becomes a chronic mental disorder that can persist for decades, or the remainder of the patient's life. Patients with chronic PTSD often have a cyclical history of symptom remissions and relapses. This group has the poorest prognosis for recovery; some patients do not respond to any of the currently available treatments for PTSD.

Prevention

Some forms of trauma, such as natural disasters and accidents, can never be completely eliminated from human life. Traumas caused by human intention would require major social changes to reduce their frequency and severity, but given the increasing prevalence of PTSD around the world, these long-term changes are worth the effort. In the short term, educating people—particularly those in the helping professions—about the signs of critical incident stress may prevent some cases of exposure to trauma from developing into full-blown PTSD.

See also Anxiety reduction techniques; Bodywork therapies; Creative therapies; Exposure treatment; Somatization and Somatoform disorders

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International Critical Incident Stress Foundation, Inc. 10176 Baltimore National Pike, Unit 201, Ellicott City, MD 21042. (410) 750-9600. Emergency: (410) 313-2473. <http://www.icisf.org>.

International Society for Traumatic Stress Studies. 60 Revere Drive, Suite 500, Northbrook, IL 60062. (847) 480-9028. <http://www.istss.org>.

National Center for PTSD. 1116D V.A. Medical Center, 215 N. Main Street, White River Junction, VT 05009-0001. (802) 296-5132. <http://www.ncptsd.org>.

National Institute of Mental Health. 6001 Executive Boulevard, Rm. 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

Rebecca J. Frey, Ph.D.

Premature ejaculation

Definition

Premature ejaculation (PE) refers to the persistent or recurrent discharge of semen with minimal sexual stimulation before, on, or shortly after penetration, before the person wishes it, and earlier than he expects it. In making the **diagnosis** of PE, the clinician must take into account factors that affect the length of time that the man feels sexually excited. These factors include the age of the patient and his partner, the newness of the sexual partner, and the location and recent frequency of sexual activity.

Causes

Premature ejaculation (PE) is a common complaint. The available evidence supports the notion that control and modulation of sexual excitement is learned behavior. If someone has learned it incorrectly or inadequately, they can relearn it. PE is only rarely caused by a physical or structural problem; in these cases it is usually associated with other physical symptoms, usually pain. In rare cases, PE may be associated with a neurological condition; infection of the prostate gland; or urethritis (inflammation of the duct that carries urine and semen to the outside of the body). With the rising prevalence of substance abuse, an increasing number of cases of PE are being diagnosed in patients withdrawing from drugs, especially opioids.

PE may be of lifelong duration or develop in later life, especially if a difficult interpersonal relationship is

KEY TERMS

Abstinence—Refraining from sexual intercourse for a period of time.

Ejaculation—The discharge of semen by the male reproductive organs.

Glans—The tip of the penis.

Orgasm—Another word for sexual climax. In the male, orgasm is usually accompanied by ejaculation but may be experienced as distinct from ejaculation.

Prostate—A muscular gland surrounding the urethra in males at the base of the urinary bladder. The prostate gland secretes the fluid that combines with the male sperm cells to form semen.

Semen—A thick whitish fluid containing sperm, produced by the male reproductive organs.

Urethritis—Inflammation of the urethra, which is the duct that carries urine and (in males) semen to the outside of the body.

one of its causes. Although PE is commonly associated with psychological symptoms, especially performance anxiety and guilt, these symptoms are its consequences rather than its causes. Once PE is firmly established, however, the accompanying psychological factors, especially in combination with sexual overstimulation, may form a self-perpetuating cycle that makes the disorder worse.

Premature ejaculation is common in adolescents where it may be made worse by feelings of sinfulness concerning sexual activity, fear of discovery, fear of making the partner pregnant, or fear of contracting a sexually transmitted disease (STD). All of these may be made worse by performance anxiety. Adults may have similar concerns as well as interpersonal factors related to the sexual partner.

Symptoms

In PE, ejaculation occurs earlier than the patient and/or the couple would like, thus preventing full satisfaction from intercourse, especially on the part of the sexual partner, who frequently fails to attain orgasm. PE is almost invariably accompanied by marked emotional upset and interpersonal difficulties that may add frustration to an already tense situation, which makes the loss of sexual fulfillment even worse. It is also important to differentiate male orgasm from ejaculation. Some men are able to distinguish between the two events and enjoy the pleasurable sensations associated with orgasm apart from the emission of semen, which usually ends the moment of orgasm. In these cases, the partner is capable of achieving orgasm and sexual satisfaction.

Diagnosis

The physical examination of a patient who is having problems with PE usually results in normal findings. Abnormal findings are unusual. The best source of information for diagnosing the nature of the problem is the patient's sexual history. On taking the patient's history, the clinician should concentrate on the sexual history, making sure that both partners have adequate and accurate sexual information. Ideally, the sexual partner should participate in the history and is often able to contribute valuable information that the patient himself may be unaware of or unwilling to relate. The female partner should also be examined by a gynecologist in order to ascertain her sexual capabilities and to eliminate the possibility that the size or structure of her genitals is part of the reason for the male's premature ejaculation.

Treatment

Preferably, therapy for PE should be conducted under the supervision of a health professional trained in sexual dysfunction. Both partners must participate responsibly in the therapeutic program. Treatment of PE requires patience, dedication and commitment by both partners, and the therapist must convey this message to both. The first part of therapy requires both partners to avoid intercourse for a period of several weeks. This period of abstinence is helpful in relieving any troublesome performance anxiety on the part of the man that may interfere with therapy.

Behavioral techniques, taught either individually, conjointly, or in groups, are effective in the therapy of PE. A preliminary stage of all treatment is termed "sensate focus" and involves the man's concentration on the process of sexual arousal and orgasm. He should learn each step in the process, most particularly the moment prior to the "point of no return." The sexual partner participates in the process, maintaining an awareness of the patient's sensations and how close he is to ejaculating. At this point, two techniques are commonly used:

- The "stop and start" technique. This approach involves sexual stimulation until the man recognizes that he is about to ejaculate. At this time, the stimulation is discontinued for about thirty seconds and then resumed. This sequence of events is repeated until ejaculation is desired by both partners, with stimulation continuing until ejaculation occurs.

- The "squeeze" technique. This approach involves sexual stimulation, usually by the sexual partner, until the man recognizes that he is about to ejaculate. At this time stimulation ceases. The patient or his partner gently squeezes the end of the penis at the junction of the glans penis (tip of the penis) with the shaft. The squeezing is continued for several seconds. Sexual stimulation is withheld for about 30 seconds and then resumed. This sequence of events is repeated by the patient alone or with the assistance of his partner until ejaculation is desired. At this point stimulation is continued until the man ejaculates.

The patient and his partner should be advised against trying any of the many unproven remedies that are available either over the counter or popularized on the Internet. Certain prescription medications, especially antidepressants that produce delayed ejaculation as a side effect, may be useful as therapeutic adjuncts. Recently, the use of a class of drugs known as selective serotonin receptor inhibitors (SSRIs) has shown promise in the treatment of premature ejaculation. The SSRIs prolong the time it takes the man to ejaculate by as much as 30 minutes. The SSRIs most commonly used to treat PE are **sertraline** (Zoloft) and **fluoxetine** (Prozac), which are currently approved by the Food and Drug Administration (FDA) for use in treating depression and panic attacks. It is important to emphasize that the use of these drugs to treat premature ejaculation is still considered experimental, as the FDA has not approved them for this specific use as of 2002.

Potential complications

Premature ejaculation that takes place before the man's penis enters the woman's vagina will interfere with conception, if the couple is planning a pregnancy. Continued lack of ejaculatory control may lead to sexual dissatisfaction for either or both members of the couple. It may become a source of marital tension, disturbed interpersonal relationships, and eventual separation or divorce.

Failure to respond to treatment for PE and the complications that may result from it should encourage the patient to seek further help from a health provider trained and experienced in treating the problem.

Prognosis

In most cases (some observers claim a 95% success rate), the patient is able to control ejaculation through education and practice of the techniques outlined. In chronic cases that do not respond to treatment, the PE may be related to a serious psychological or psychiatric condition, including depression or anxiety. Patients in this category may benefit from **psychotherapy**.

See also Male orgasmic disorder

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Ralph Myerson, M.D.

Primary hypersomnia *see* **Hypersomnia**

Primary insomnia *see* **Insomnia**

Prolixin *see* **Fluphenazine**

Propranolol

Definition

Propranolol is classified as a beta blocker. It is sold in the United States under the brand name Inderal. When combined with the diuretic, hydrochlorothiazide, it is sold under the brand name Inderide. Propranolol also is produced as a generic product by a number of generic manufacturers.

Purpose

Propranolol is approved by the Food and Drug Administration (FDA) for the treatment of hypertension (high blood pressure), angina, certain types of cardiac arrhythmias, certain types of cardiac output diseases, a sympathetic nervous system disorder known as pheochromocytoma, hyperthyroid conditions, migraine, heart attack, and tremors of a variety of origins. It is also used on occasion for the treatment of **medication-induced movement disorders** caused by antipsychotic drugs and certain anxiety states in people suffering from a specific form of **social phobia**. **Beta blockers**, such as propanolol, are not useful for people with general social phobia who are anxious in most social situations; instead, propanolol may be useful for people who are anxious about specific performance situations, such as presenting a speech before an audience.

KEY TERMS

Beta blocker—Drugs that block beta-adrenergic receptors on neurons in the central nervous system. When these sites are blocked, heart rate, blood pressure, and anxiety levels decrease.

Brachycardia—Slow heartbeat, defined as a rate of less than 60 beats per minute.

Diuretic—An agent that increases the amount of urine; often used to decrease fluid retention in bodily tissues.

Epinephrine (adrenaline)—The principal blood pressure-raising hormone and a bronchial and intestinal smooth muscles relaxant; prescribed to (among other things) stimulate the heart and as a muscle relaxant in bronchial asthma.

Glaucoma—A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

Hypotension—Low blood pressure.

Ischemia—Localized anemia of tissues due to obstructed inflow of blood.

Laryngospasm—Spasms that close the vocal apparatus of the larynx (the organ of voice production).

Norepinephrine (noradrenaline)—A hormone with similar stimulatory effects to epinephrine but, in contrast to epinephrine, has little effect on cardiac (heart) output and in relaxing smooth muscles.

Raynaud's syndrome—A disorder of the circulatory or vascular system characterized by abnormally cold hands and feet because of constricted blood vessels in these areas.

Thrombocytopenia—A condition involving abnormally low numbers of platelets (blood-clotting agents) in the blood; usually associated with hemorrhaging (bleeding).

Description

Propranolol falls into the broad pharmacologic category known as beta blockers. Beta blockers block specific sites in the central nervous system known as beta-adrenergic receptor sites. When these sites are blocked, heart rate and blood pressure are reduced and patients become less anxious. Because of this, propranolol is useful in treating chest pain, high blood pressure, and excessive nervousness. Unfortunately, propranolol often makes breathing disorders, such as asthma, worse because it tends to constrict breathing passages and sometimes causes fluid to build up in the lungs if it excessively depresses the heart.

In the treatment of anxiety, propranolol is usually not administered on a chronic basis but, rather, prior to stressful events such as public speaking or acting. In the treatment of certain types of tremors, especially tremors secondary to a drug, and movement disorders secondary to antipsychotic therapy, propranolol is administered throughout the day in divided doses. Propranolol is available in 10-, 20-, 40-, 60-, and 80-mg tablets; in long-acting capsules; and an injectable form containing 1 mg per mL. It is also combined with the diuretic hydrochlorothiazide in tablets and extended-release capsules.

Recommended dosage

For the treatment of performance anxiety or stage fright, a single dose of 10–40 mg may be administered 20–30 minutes before the event. For the treatment of tremors, especially tremors secondary to lithium, doses range from 20 to 160 mg per day administered in two or three divided doses. For the treatment of movement disorders secondary to antipsychotic drug therapy, doses range from 10 to 30 mg three times daily.

Precautions

Precautions should be taken when administering propranolol in the following situations:

- liver or renal (kidney) failure
- prior to screening tests for glaucoma
- a history of immediate allergic reaction (known as anaphylaxis) to a beta blocker of any kind

Side effects

The following side effects have been observed with propranolol. Most have been mild and transient (temporary) and rarely require the withdrawal of therapy:

- Cardiovascular: bradycardia, congestive heart failure, hypotension, Raynaud's syndrome.
- Central nervous system: light-headedness, mental depression, **insomnia**, vivid dreams, disorientation, memory loss.
- Gastrointestinal: nausea, vomiting, abdominal pain, cramping, diarrhea, constipation, bowel ischemia.
- Allergic: fever, rash, laryngospasm, thrombocytopenia.

- Respiratory: bronchospasm.
- Hematologic: bone marrow suppression, bleeding under the skin.

Interactions

- When drugs that deplete the body of epinephrine and norepinephrine (such as reserpine and guanethidine) are taken with propranolol, interactions have been reported. Some of these interactions include: fainting, hypotension, dizziness, and slow heart rate.
- Drugs known as calcium channel blockers may decrease the pumping ability of the heart and lead to the development of cardiac arrhythmias.
- Nonsteroidal anti-inflammatory agents (i.e., ibuprofen and naproxen) may blunt the blood pressure-lowering effects of propranolol.
- Aluminum hydroxide antacids greatly reduce the rate of intestinal absorption of propranolol.
- Alcohol slows the rate of propranolol absorption.
- Interactions have also been reported with phenytoin, rifampin, phenobarbital, **chlorpromazine**, lidocaine, thyroxin, cimetidine, and theophylline.

See also Alcohol and related disorders; Anxiety and related disorders

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Ralph Myerson, M.D.

Prosom *see* **Estazolam**

Protriptyline

Definition

Protriptyline is an oral tricyclic antidepressant. It is sold in the United States under the brand name Vivactil and is also available under its generic name.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that generally produces effects that are the opposite of those produced by dopamine and norepinephrine. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Benign prostate hypertrophy—Enlargement of the prostate gland.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Purpose

Protriptyline is used primarily to treat depression and to treat the combination of symptoms of anxiety and depression. Like most antidepressants of this chemical and pharmacological class, protriptyline has also been used in limited numbers of patients to treat **panic disorder**, **obsessive-compulsive disorder**, **attention-deficit/hyperactivity disorder**, **enuresis** (bed-wetting), eating disorders such as **bulimia nervosa**, cocaine dependency, and the depressive phase of **bipolar disorder** (manic-depressive) disorder. It has also been used to support smoking cessation programs.

Description

Tricyclic antidepressants act to change the balance of naturally occurring chemicals in the **brain** that regulate the transmission of nerve impulses between cells. Protriptyline acts primarily to increase the concentration

of norepinephrine and serotonin (both chemicals that stimulate nerve cells) and, to a lesser extent, to block the action of another brain chemical, acetylcholine. Protriptyline shares most of the properties of other tricyclic antidepressants, such as **amitriptyline**, **clomipramine**, **desipramine**, **imipramine**, **nortriptyline**, and **trimipramine**. Studies comparing protriptyline with these other drugs have shown that protriptyline is no more or less effective than other antidepressants of its type. Its choice for treatment is as much a function of physician preference as any other factor.

The therapeutic effects of protriptyline, like other antidepressants, appear slowly. Maximum benefit is often not evident for at least two weeks after starting the drug. People taking protriptyline should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Recommended dosage

As with any antidepressant, protriptyline must be carefully adjusted by the physician to produce the desired therapeutic effect. Protriptyline is available as 5-mg and 10-mg tablets. Doses range from 15 to 40 mg per day and can be taken in one daily dose or divided into up to four doses daily. Some people with severe depression may require up to 60 mg per day.

In adolescents and people over age 60, therapy should be initiated at a dose of 5 mg three times a day and increased under supervision of a physician as needed. Patients over age 60 who are taking daily doses of 20 mg or more should be closely monitored for side effects such as rapid heart rate and urinary retention.

Precautions

Like all tricyclic antidepressants, protriptyline should be used cautiously and with close physician supervision in people, especially the elderly, who have benign prostatic hypertrophy (enlarged prostate gland), urinary retention, and glaucoma, especially angle-closure glaucoma (the most severe form). Before starting treatment, people with these conditions should discuss the relative risks and benefits of treatment with their doctors to help determine if protriptyline is the right antidepressant for them.

A common problem with tricyclic antidepressants is sedation (drowsiness, lack of physical and mental alertness). This side effect is especially noticeable early in therapy. In most people, sedation decreases or disappears entirely with time, but, until then, patients taking protriptyline should not perform hazardous activities requiring mental alertness or coordination. The sedative effect is increased when protriptyline is taken with other central nervous system depressants, such as alcoholic beverages, sleeping medications, other sedatives, or antihistamines. It may be dangerous to take protriptyline in combination with these substances. Protriptyline may increase the possibility of having **seizures**. Patients should tell their physician if they have a history of seizures, including seizures brought on by the abuse of drugs or alcohol. These people should use protriptyline only with caution and should be closely monitored by their physician.

Protriptyline may increase heart rate and stress on the heart. It may be dangerous for people with cardiovascular disease, especially those who have recently had a heart attack, to take this drug or other antidepressants in the same pharmacological class. In rare cases in which patients with cardiovascular disease must take protriptyline, they should be monitored closely for cardiac rhythm disturbances and signs of cardiac stress or damage.

Side effects

Protriptyline shares side effects common to all tricyclic antidepressants. The most frequent of these are dry mouth, constipation, urinary retention, increased heart rate, sedation, irritability, dizziness, and decreased coordination. As with most side effects associated with tricyclic antidepressants, the intensity is highest at the beginning of therapy and tends to decrease with continued use.

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Patients may also chew sugarless gum or suck on sugarless candy in order to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement who take protriptyline may be especially likely to have problems with urinary retention. Symptoms include having difficulty starting a urine flow and more difficulty than usual passing urine. In most cases, urinary retention is managed with dose reduction or by switching to another type of antidepressant. In extreme cases, patients may require treatment with bethanechol, a drug that reverses this particular side effect. People who think they may be experiencing any side effects from this or any other medication should tell their physicians.

Interactions

Dangerously high blood pressure has resulted from the combination of tricyclic antidepressants, such as protriptyline, and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors.

Because of this, protriptyline should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting protriptyline or any other tricyclic antidepressant. The same holds true when discontinuing protriptyline and starting an MAO inhibitor.

Protriptyline may decrease the blood pressure–lowering effects of **clonidine**. Patients who take both drugs should be monitored for loss of blood-pressure control and the dose of clonidine increased as needed.

The sedative effects of protriptyline are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or medications used for other mental disorders such as **schizophrenia**. The anticholinergic effects of protriptyline are additive with other anticholinergic drugs such as **benztropine**, **biperiden**, **trihexyphenidyl**, and antihistamines.

See also Neruotransmitters

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Jack Raber, Pharm.D.

Prozac *see* **Fluoxetine**

Pseudocyesis

Definition

Pseudocyesis is the medical term for a false pregnancy. Pseudocyesis can cause many of the signs and symptoms of pregnancy, and often resembles the condition in every way except for the presence of a fetus.

Description

Pseudocyesis has been observed and written about since antiquity. Hippocrates set down the first written account around 300 B.C., and recorded 12 different cases of women with the disorder. One of the most famous historical examples is Mary Tudor (1516-1558), Queen of England, who believed on more than one occasion that she was pregnant when she was not. Some even attribute the violence that gave her the nickname "Bloody Mary" as a reaction to the disappointment of finding out that she was not carrying a child. Other historians believe that the queen's physicians mistook fibroid tumors in her uterus for a pregnancy, as fibroids can enlarge a nonpregnant uterus.

KEY TERMS

Cervix—The neck or narrow lower end of the uterus. Softening of the cervix is one of the signs of pregnancy.

Distension—The condition of being stretched or expanded, as the abdomen of a pregnant woman.

Quickening—A term that refers to the movements or other signs of life of a fetus in the womb.

Pseudocyesis has become increasingly rare in many parts of the world in which accurate pregnancy tests have become widely available. Cultures that place high value on pregnancy, or that make close associations between fertility and a person's worth, still have high rates of the disorder.

Signs and symptoms

The symptoms of pseudocyesis are similar to the symptoms of true pregnancy and are often hard to distinguish from such natural signs of pregnancy as morning sickness, tender breasts, and weight gain. Many health care professionals can be deceived by the symptoms associated with pseudocyesis. Eighteen percent of women with pseudocyesis were at one time diagnosed as pregnant by a medical professional. In some cases, the only difference between pregnancy and pseudocyesis is the presence of a fetus.

The sign of pseudocyesis that is common to all cases is that the affected patient is convinced that she is pregnant. Abdominal distension is the most common physical symptom of pseudocyesis (63– 97% of women are found to experience this). The abdomen expands in the same manner as it does during pregnancy, so that the affected woman looks pregnant. This phenomenon is thought to be caused by buildup of gas, fat, feces, or urine. These symptoms often resolve under general anesthesia and the woman's abdomen returns to its normal size.

The second most common physical sign of pseudocyesis is menstrual irregularity (56–98% of women experience this). Between 48% and 75% of women are also reported to experience the sensation of fetal movements

known as quickening, even though there is no fetus present. Some of the other common signs and symptoms include: gastrointestinal symptoms, breast changes or secretions, labor pains, uterine enlargement, and softening of the cervix. One percent of women eventually experience false labor.

Causes

No single theory about the causes of pseudocyesis is universally accepted by mental health professionals. The first theory attributes the false pregnancy to emotional conflict. It is thought that an intense desire to become pregnant, or an intense fear of becoming pregnant, can create internal conflicts and changes in the endocrine system, which may explain some of the symptoms of pseudocyesis. The second theory concerns wish-fulfillment. It holds that if a women desires pregnancy badly enough she may interpret minor changes in her body as signs of pregnancy. The third leading theory is the depression theory, which maintains that chemical changes in the nervous system associated with some depressive disorders could trigger the symptoms of pseudocyesis.

Demographics

The rate of pseudocyesis in the United States has declined significantly in the past century. In the 1940s there was one occurrence for approximately every 250 pregnancies. This rate has since dropped to between one and six occurrences for every 22,000 births. The average age of the affected woman is 33, though cases have been reported for women as young as 6-1/2 and as old as 79. More than two-thirds of women who experience pseudocyesis are married, and about one-third have been pregnant at least once. Women who have been victims of incest may be at greater risk for developing pseudocyesis. Pseudocyesis is found in some mammals other than humans—most often cats, dogs, and rabbits.

Treatment

Because pseudocyesis is not known to have a direct underlying physical cause, there are no general recommendations regarding treatment with medications. In some cases, however, the patient may be given medications for such symptoms as the cessation of menstruation. Because most patients with pseudocyesis have underlying psychological problems, they should be referred to a psychotherapist for the treatment of these problems. It is important at the same time, however, for the treating professional not to minimize the reality of the patient's physical symptoms.

The treatment that has had the most success is demonstrating to the patient that she is not really pregnant by the use of ultrasound or other imaging techniques.

Alternative therapies

There have been reports of patients being cured of pseudocyesis by hypnosis, purgatives, massage, opioids, or after nine months of symptoms, by experiencing "hysterical childbirth," but there are few data available on the effectiveness of these or similar procedures.

Prognosis

Symptoms of pseudocyesis generally last from a few months to a few years. In most cases, symptoms last for a full nine months. There is a high success rate for treatments involving **psychotherapy**, as it treats the underlying psychological causes of the disorder.

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Tish Davidson, A.M.

Psychiatric assessment *see* **Assessment and diagnosis**

Psychiatrist

Definition

A psychiatrist is a physician who specializes in the **diagnosis** and treatment of mental disorders.

Description

Psychiatrists treat patients privately and in hospital settings through a combination of **psychotherapy** and medication. Their training consists of four years of medical school, followed by one year of internship and at least three years of psychiatric residency. Psychiatrists may receive certification from the American Board of Psychiatry and Neurology (ABPN), which requires two years of clinical experience beyond residency and the successful completion of a written and an oral test. Unlike a medical license, board certification is not legally required in order to practice psychiatry.

Psychiatrists may practice general psychiatry or choose a specialty, such as child psychiatry, geriatric psychiatry, treatment of substance abuse, forensic (legal) psychiatry, emergency psychiatry, **mental retardation**, community psychiatry, or public health. Some focus their research and clinical work primarily on psychoactive medication, in which case they are referred to as psychopharmacologists. Psychiatrists may be called upon to address numerous social issues, including juvenile delinquency, family and marital dysfunction, legal competency in criminal and financial matters, and treatment of mental and emotional problems among prison inmates and in the military.

Psychiatrists treat the biological, psychological, and social components of mental illness simultaneously. They can investigate whether symptoms of mental disorders have physical causes, such as a hormone imbalance or an adverse reaction to medication, or whether psychological symptoms are contributing to physical conditions, such as cardiovascular problems and high blood pressure. Because they are licensed physicians, psychiatrists—unlike psychologists and psychiatric social workers—can prescribe medication; they are also able to admit patients to the hospital. Other mental health professionals who cannot prescribe medication themselves often establish a professional relationship with a psychiatrist.

Psychiatrists may work in private offices, private psychiatric hospitals, community hospitals, state and federal hospitals, or community mental centers. Often, they combine work in several settings. In addition to their clinical work, psychiatrists often engage in related professional activities, including teaching, research, and administration. The American Psychiatric Association, the oldest medical specialty organization in the United States, supports the profession by offering continuing education and research opportunities, keeping members informed about new research and public policy issues, helping to educate the public about mental health issues, and serving as an advocate for people affected by mental illness.

Psychoanalysis

Definition

Psychoanalysis, as a form of therapy, is based on the understanding that human beings are largely unaware of the mental processes that determine their thoughts, feelings, and behavior, and that psychological suffering can be alleviated by making those processes known to the individual.

Sigmund Freud originally developed the theory and technique of psychoanalysis in the 1890s. Freud's ideas are still used in comptemporary practice; however, many have been further developed or refined and some even abandoned. The theory and technique of psychoanalyis contnues to integrate new insights about human development and behavior based on psychoanalytic research and discoveries from related fields. Different schools of psychoanalytic theory have evolved out of the original Freudian one, reflecting a variety of ideas and perspectives. Psychoanalysis is practiced by a trained psychoanalyst, also referred to as an analyst.

Purpose

Primary goals of psychoanalysis include symptom relief, increased self-awareness, and a more objective capacity for self-observation. Other aims might include improved relationships with others and the capacity to live a more deeply satisfying life. Typically, an individual seeks treatment in order to alleviate some difficulty, such as unhappiness in work or love, disturbances in mood or self-esteem, or troubling personality traits. With the exception of those that are physically based, psychoanalysis views such symptoms as related to unconscious mental processes, and because these mental forces are not within the individual's awareness, symptoms cannot be relieved with perseverance or with the help of friends or family. Through a slowly unfolding process, psychoanalysis demonstrates to the individual how unconscious mental processes affect current modes of thinking, feeling and interacting with others. It also demonstrates that these processes can be traced back to early experiences and relationships with caregivers and family members. This kind of insight enables the person to identify the sources of their sometimes troubling thoughts, feelings and behavior and, as a result, gives new meaning to current modes of functioning. This kind of transformation of character takes several years to accomplish due to the intense nature of the process. It requires a sacrifice of time, money, and mental energy. The resulting transformation offers the means for adaptive, enduring changes in

Patients often seek psychoanalysis to help them achieve a greater sense of self-understanding and personal satisfaction. *(Laurent/Meeus/BSIP/Science Source. Photo Researchers, Inc. Reproduced by permission.)*

personality. These are changes that enable the individual to live a more productive, satisfying and pleasurable life.

Precautions

The term "psychoanalyst" can be used by anyone, so it is important to know the credentials of an analyst prior to beginning treatment.

Credentials

In addition to having received advanced degrees in mental health (psychiatry, psychology, social work), trained psychoanalysts have also graduated from psychoanalytic training institutes. Institute training consists of three parts: course work on psychoanalytic theory and technique; supervised analyses (meaning that the candidate conducts analyses while being supervised by a seasoned psychoanalyst); and, third, candidates undergo a personal psychoanalysis. A personal analysis is considered a vital part of the training, as it enables candidates to learn about their own psychological processes. In turn, the knowledge enhances their capacity to treat others. This type of training program takes a minimum of four years to complete. Psychoanalysts also practice psychoanalytic **psychotherapy**, a less intensive form of treatment. It relies on the same theory of human development and a similar technique.

Description

In psychoanalysis, an individual in treatment is seen four to five times per week for 45- to 50-minute sessions. The individual lies comfortably on a couch while the analyst sits in a chair behind the person, out of view. The person is then asked to say whatever comes into his or her mind. Although this structure varies depending on the theory and style of the analyst, this is the most typical and traditional manner in which sessions are conducted. These conditions are maintained consistently, making it possible for thoughts and feelings to emerge that had once been outside of the person's awareness. The process of free associating, or saying whatever comes to mind, is challenging because people are taught at a young age to keep many ideas and feelings to themselves. When the analyst is out of view, it removes the possibility for eye contact, making it easier to speak spontaneously. Free

association is also made easier by the analyst's nonjudgmental attitude—in listening to the individual, in the attention and interest given to seemingly unimportant details, and the objective and caring attitude with which the analyst understands the individual.

As the person speaks, unconscious sources of present-day difficulties gradually appear. Specifically, the analyst begins to notice repetitive aspects of behavior. Some of them may include particular subjects about which the person finds it hard to speak, as well as habitual ways in which the person relates to the psychoanalyst. The analyst begins to reveal these to the person in a gradual and thoughtful manner. Sometimes these revelations are accepted as correct and helpful. At other times they are rejected, corrected, or refined.

During the years of an analysis, the individual will grapple with new insights repeatedly, each time comprehending them in new ways. There will be an enhanced emotional and intellectual understanding, in addition to seeing matters from the perspective of different periods of life. As in all worthwhile learning processes, this one includes times of deep satisfaction and great frustration, periods of growth and regression. Overall, the analyst and individual work together to modify debilitating life patterns, to ameliorate troubling symptoms, and to release emotional and intellectual resources bound up in unconscious psychological processes. A transformation will occur eventually, and be one in which the person's understanding of themselves and of others, along with their productivity in work and capacity to love, changes in profound and enduring ways.

Who can benefit from psychoanalysis?

Anyone interested should seek a consultation with a psychoanalyst in order to determine if this treatment is appropriate. People often begin psychoanalysis also after having participated in psychoanalytic psychotherapy, which is a less intense form of treatment.

Individuals who are the most suited for psychoanalysis are those who have experienced satisfactions in work, with friends, in marriage, but who nonetheless experience a general dissatisfaction with their life—suffering from long-standing depression, anxiety, sexual difficulties, physical symptoms without physical basis, or typically feel isolated or alone. Some people need analysis because their habitual ways of living interfere with experiencing greater pleasure or productivity in life. Individuals need to be psychologically minded with an interest in becoming more self-aware, and a determination to forgo quick symptom relief in favor of a more gradual therapeutic process.

Psychoanalysis is also practiced with children and adolescents, with some variation in technique. Specifically, fantasy play and drawings are used with children in addition to verbal communication. During the treatment of children and young adolescents, parents are consulted on a regular basis so that the analyst can develop a more holistic understanding of the youngster's world. The goal of child and adolescent psychoanalysis is to alleviate symptoms and to remove any obstacles that interfere with normal development.

With other treatments

Psychoanalysis is used at times with other forms of treatment. Medication may be warranted in selected situations—if an individual suffers from a severe mood disturbance which interferes with his or her capacity to participate in treatment, for example. In general, medication is used as a tool that allows the individual to benefit from the psychoanalytic process; it is an adjunct therapy, while psychoanalysis is the primary curative one. There are also occasions in which psychoanalysis is provided concurrently with **couples therapy** or **family therapy** or with **group therapy**. Treatment recommendations, whether for psychoanalysis alone or in combination with couples, family, or group therapy, are based both on the individual's particular needs and the practice of the treating psychoanalyst.

Finally, psychoanalysis is not only a type of therapy. It is also a theory of human development from infancy to old age, a method for understanding thought processes. It offers a way of thinking about aspects of society and culture such as religion, prejudice, and war.

Normal results

Normal results include symptom relief and an enduring, adaptive change in personality.

Abnormal results

Some individuals do not benefit from this in-depth form of treatment. They instead experience increased distress, or do not progress after a sufficient amount of treatment sessions have elapsed. In these cases, people are typically transitioned to a less intensive form of treatment such as psychoanalytic psychotherapy.

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International Psychoanalytical Association. Broomhills, Woodside Lane, London N128UD.<http//www.ipa.org.uk>.

Susan Fine, Psy.D

Psychodynamic psychotherapy

Definition

Psychodynamic **psychotherapy** is a method of verbal communication used to help a person find relief from emotional pain. It is based on the theories and techniques of **psychoanalysis**. Psychodynamic psychotherapy is similar to psychoanalysis in that it attributes emotional problems to the patient's unconscious motives and conflicts. It differs from classical psychoanalysis, however, in that psychodynamic psychotherapists do not necessarily accept Freud's view that these unconscious motives and conflicts are ultimately sexual in nature.

Purpose

The goals of psychodynamic psychotherapy vary depending on the method of treatment, which can be broadly described as either expressive or supportive. Expressive therapy seeks to relieve symptoms through the development of insight, or the slowly developing awareness of feelings and thoughts that were once outside of the person's awareness. Expressive therapy is based on the rationale that difficulties experienced in adult life originate in childhood; that children do not possess the maturity for making effective choices nor the independence to do so; and that methods of adapting that were developed in childhood may no longer be effective for adapting to the world as an adult. Through guidance from a therapist, the adult becomes aware of present ways of coping that are ineffective and how they served a purpose in childhood that is no longer relevant. The person learns that he or she now has a range of new options for solving problems, and for living in general that are now based on his or her maturity and independence.

In contrast to expressive therapy which is exploratory, supportive therapy remains closer to the surface of the patient's issues. Supportive therapy is an approach that is used to relieve immediate distress; to return the person to his or her previous level of functioning; and to strengthen adaptive ways of coping that the individual already possesses in order to prevent further discomfort. Expressive and supportive methods of treatment are not completely separate categories because elements of supportive therapy are used in expressive treatment and vice versa, depending on the therapeutic need. For instance, if a person in exploratory treatment is experiencing distress, a supportive approach may be used for a period of time in order to help the person feel more stable.

While many patients benefit from individual psychotherapy alone, some instances call for such additional therapies as **family therapy**, **couples therapy**, or **group therapy** in combination with individual treatment. A second treatment modality might be recommended when the patient's progress in individual treatment is highly dependent on relationships with significant others or with interpersonal relationships in general. Psychotropic (mood- or behavior-altering) medication may also be prescribed as an adjunct (help) to treatment in order to manage disturbances in anxiety level, mood or thinking. Whether additional treatments are recommended is based on the needs of the individual.

People seek psychodynamic psychotherapy for a variety of reasons that include but are not limited to the following: prolonged sadness, anxiety, sexual difficulties, physical symptoms without physical basis, persistent feelings of isolation and loneliness, and the desire to be more successful in work or love. People seek therapy because they have not been able to develop a stable resolution for their difficulties on their own or with the help of friends and family members.

Description

Sessions of psychodynamic psychotherapy may be scheduled from one to three days per week, with greater frequency allowing for more in-depth treatment. The duration of individual sessions varies, but typically lasts for 45–50 minutes. It is not usually possible at the outset of treatment to estimate the number of sessions that will be necessary in order to achieve the person's goals. It is possible, however, for the person to make arrangements for a specific number of sessions.

Psychodynamic psychotherapy begins with a period of evaluation during which the client discusses with the

therapist the reasons for seeking treatment. This process gives the therapist the opportunity to learn about the person, to develop an understanding of his or her troubles, and to formulate ideas about how treatment should proceed. This phase of interviewing and learning may take place in one session or over a series of sessions; or it may be done in a less structured manner, depending on the therapist's style. During the initial sessions, such factors as the frequency and length of sessions and the policy for payment will also be discussed. At some point within the first few sessions, the therapist and the individual will come to a mutual understanding of the goals for treatment. After this point, the sessions will become less like an interview; the person is asked to say whatever is on his or her mind. It is the therapist's job to listen and to help identify patterns of thinking, feeling and interacting that may be contributing to the patient's current struggles. Consequently, the person becomes more aware of his or her thoughts and feelings; learns how some present ways of coping are no longer adaptive even though they may have been necessary in childhood; and discovers that he or she as an adult has a greatly expanded repertoire of resources and can use far more effective ways of dealing with problems. Deeper awareness and new insights stimulate psychological growth and change.

Psychodynamic psychotherapy places great importance on the therapeutic dyad, which is a medical term for the relationship between the therapist and the patient. It is within the context of the therapeutic dyad that positive changes in the patient's outlook and behaviors are able to unfold. This relationship is unique because the therapist maintains a uniform, neutral and accepting stance. Unlike other well-intentioned people in the person's life, the therapist has been trained to listen objectively and without criticism. This therapeutic attitude makes it easier for the person seeking treatment to speak freely and to therefore provide as much information for the therapist to work with as is possible.

Treatment continues until the troubling symptoms have been reduced or alleviated and the person is consistently making use of more adaptive methods of coping with greater insight. For some people, this positive experience inspires them to proceed with further treatment in order to bring about additional adaptive changes. For others, meeting the initial goals will be sufficient. If so, the focus of sessions turns to issues related to the end of treatment. This final phase of treatment is as important as the beginning and middle stages because it allows the individual to develop insight about his or her therapeutic experience. People need time to clarify how they feel about leaving the therapeutic relationship, and this termination involves identifying and understanding feelings about separation, maturation, loss and change. The length of time allotted to the termination phase varies with the type of treatment and with the needs of the individual.

KEY TERMS

Adjunct—A form of treatment that is not strictly necessary to a therapy regimen but is helpful.

Expressive therapy—An approach to psychotherapy that seeks to relieve the patient's symptoms through exploration of previously unconscious material, leading to greater insight and more adaptive behaviors.

Modality—A term used in medicine for a method of treatment. For example, multimodal treatment plans make use of more than one treatment or therapeutic modality.

Psychotropic—Having an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

Supportive therapy—An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, and to return the patient to previous levels of functioning, as distinct from insight-oriented or educational approaches to treatment.

Therapeutic dyad—A term that refers to the two people involved in a psychotherapeutic relationship, namely the therapist and the person seeking treatment.

Normal results

After a course of psychodynamic psychotherapy has ended, the person should, overall, continue to handle difficulties in a more adaptive manner; experience improved interpersonal relationships and productivity at work; and continue to develop new insights into his or her thoughts, feelings and behavior. In supportive treatment, insight and personality change are not the primary goals of treatment; the therapist and patient work toward a continuation of general stability in the person's life.

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ORGANIZATIONS

American Psychoanalytic Association. 309 East 49th Street, New York, NY 10017. (212) 752-0450. <http://www.apsa.org>.

American Psychological Association. 750 First Street, NE, Washington D.C. 20002. (800) 374-2721. <http://www.apa.org>.

Susan Fine, Psy.D.

Psychologist

Definition

A psychologist is a social scientist who studies behavior and mental processes, generally in a research or clinical setting.

Description

As psychology has grown and changed throughout history, it has been defined in numerous ways. As early as 400 B.C., the ancient Greeks philosophized about the relationship of personality characteristics to physiological traits. Since then, philosophers have proposed theories to explain human behavior. In the late nineteenth century, the emergence of scientific method gave the study of psychology a new focus. In 1879, the first psychological laboratory was opened in Leipzig, Germany by Wilhelm Wundt, and soon afterwards the first experimental studies of memory were published. Wundt was instrumental in establishing psychology as the study of conscious experience, which he viewed as made up of elemental sensations. In addition to the type of psychology practiced by Wundt—which became known as structuralism—other early schools of psychology were functionalism, which led to the development of behaviorism, and Gestalt psychology. The American Psychological Association was founded in 1892 with the goals of encouraging research, enhancing professional competence, and disseminating knowledge about the field.

With the ascendance of the Viennese neurologist Sigmund Freud and his method of **psychoanalysis** early in the twentieth century, emphasis shifted from conscious experience to unconscious processes investigated by means of free association and other techniques. According to Freud, behavior and mental processes were the result of mostly unconscious struggles within each person between the drive to satisfy basic instincts, such as sex or aggression, and the limits imposed by society. At the same time that Freud's views were gaining popularity in Europe, an American psychology professor, John B. Watson, was pioneering the behavioral approach, which focuses on observing and measuring external behaviors rather than the internal workings of the mind. B. F. Skinner, who spent decades studying the effects of reward and punishment on behavior, helped maintain the predominance of behaviorism in the United States through the 1950s and 1960s. Since the 1970s, many psychologists have been influenced by the cognitive approach, which is concerned with the relationship of mental processes to behavior. Cognitive psychology focuses on how people take in, perceive, and store information, and how they process and act on that information.

Additional psychological perspectives include the neurobiological approach, focusing on relating behavior to internal processes within the **brain** and nervous system, and the phenomenological approach, which is most concerned with the individual's subjective experience of the world rather than the application of psychological theory to behavior. While all these approaches differ in their explanations of individual behavior, each contributes an important perspective to the overall psychological understanding of the total human being. Most psychologists apply the principles of various approaches in studying and understanding human nature.

Along with several approaches to psychology there are also numerous, overlapping subfields in which these approaches may be applied. Most subfields can be categorized under one of two major areas of psychology referred to as basic and applied psychology. Basic psychology encompasses the subfields concerned with the advancement of psychological theory and research. Experimental psychology employs laboratory experiments to study basic behavioral processes shared by different species, including sensation, perception, learning, memory, communication, and motivation. Physiological psychology is concerned with the ways in which biology shapes behavior and mental processes, and developmental psychology is concerned with behavioral development over the entire life span. Other subfields include social psychology, quantitative psychology, and the psychology of personality.

Applied psychology is the area of psychology concerned with applying psychological research and theory to problems posed by everyday life. It includes clinical psychology, the largest single field in psychology.

Clinical psychologists—accounting for 40% of all psychologists—are involved in **psychotherapy** and psychological testing. Clinical psychologists are trained in research and often work in university or research settings, studying carious aspects of psychology. Like clinical psychologists, counseling psychologists apply psychological principles to diagnose and treat individual emotional and behavioral problems. Other subfields of applied psychology include school psychology, which involves the evaluation and placement of students; educational psychology, which investigates the psychological aspects of the learning process; and industrial and organizational psychology, which study the relationship between people and their jobs. Community psychologists investigate environmental factors that contribute to mental and emotional disorders; health psychologists deal with the psychological aspects of physical illness, investigating the connections between the mind and a person's physical condition; and consumer psychologists study the preferences and buying habits of consumers as well as their reactions to certain advertising.

In response to society's changing needs, new fields of psychology are constantly emerging. One new type of specialization, called environmental psychology, focuses on the relationship between people and their physical surroundings. Its areas of inquiry include such issues as the effects of overcrowding and noise on urban dwellers and the effects of building design. Another relatively new specialty is forensic psychology, involving the application of psychology to law enforcement and the judicial system. Forensic psychologists may help create personality profiles of criminals, formulate principles for jury selection, or study the problems involved in eyewitness testimony. Yet another emerging area is program evaluation, whose practitioners evaluate the effectiveness and cost efficiency of the programs.

Depending on the nature of their work, psychologists may practice in a variety of settings, including colleges and universities, hospitals and **community mental health** centers, schools, and businesses. A growing number of psychologists work in private practice and may also specialize in multiple subfields. Most psychologists earn a Ph.D. degree in the field, which requires completion of a four- to six-year post-bachelors' degree program offered by a university psychology department. The course of study includes a broad overview of the field, as well as specialization in a particular subfield, and completion of a dissertation and an internship (usually needed only for applied psychology, such as clinical, counseling, and school psychology). Students who intend to practice only applied psychology rather than conduct research have the option of obtaining a Psy.D. degree, which differs in the limited emphasis that is put on research and a dissertation that does not have to be based on an empirical research study.

Psychosis

Definition

Psychosis is a symptom of mental illness characterized by a radical change in personality and a distorted or diminished sense of objective reality.

Description

Psychosis appears as a symptom of a number of mental disorders, including mood and **personality disorders**, **schizophrenia**, **delusional disorder**, and substance abuse. It is also the defining feature of the psychotic disorders (i.e., **brief psychotic disorder**, **shared psychotic disorder**, psychotic disorder due to a general medical condition, and **substance-induced psychotic disorder**).

Patients suffering from psychosis are unable to distinguish the real from the unreal. They experience **hallucinations** and/or **delusions** that they believe are real, and they typically behave in an inappropriate and confused manner.

A mental illness can exhibited through various forms of psychosis, such as:

- *Delusions.* An unshakable and irrational belief in something untrue. Delusions defy normal reasoning, and remain firm even when overwhelming proof is presented to disprove them.
- *Hallucinations.* Psychosis causes false or distorted sensory experience that appear to be real. Psychotic patients often see, hear, smell, taste, or feel things that aren't there.
- *Disorganized speech.* Psychotic patients often speak incoherently, using noises instead of words and "talking" in unintelligible speech patterns.
- *Disorganized or catatonic behavior.* Behavior that is completely inappropriate to the situation or environment. Catatonic patients have either a complete lack of or inappropriate excess of motor activity. They can be completely rigid and unable to move (vegetative), or in constant motion. Disorganized behavior is unpredictable and inappropriate for a situation (such as screaming obscenities in the middle of class).

Paula Ford-Martin, M.A.

Psychosurgery

Definition

Psychosurgery is the treatment of a psychiatric disorder using surgical techniques to destroy **brain** tissue and is now rarely used.

Purpose

It is a last-resort treatment for extreme, debilitating, psychiatric disorders.

Description

Early psychosurgery—historical perspective

Ironically, brain surgery, a medical practice requiring extraordinary levels of skill and care, may be one of the oldest of all medical procedures. This surprising observation is supported by physical evidence dating back 40,000 years ago to Neolithic times. Archeologists have found numerous human skulls showing signs of a procedure called trepanation or trepanning—an operation in which a hole is cut through the bone that covers the brain (skull) in order to access the brain. A key feature of the wounds found in these ancient skulls is the smoothness and shininess around the edges of the holes. This is a clear sign of new bone growth and evidence that the person whose skull was opened not only survived the operation but lived months or even years afterwards while the bone regrew.

Having one's skull opened in a modern surgical setting is not taken lightly, even with the most modern surgical techniques. The prospect of undergoing the operation in the late Stone Age may appear to us to imply certain death. However, the survival rate of the operation was quite high. Close examination of archeological findings suggests that 75% of those who underwent the procedure lived long enough for new bone to grow around the opening. That number is actually higher than the survival rate for brain surgery during the nineteenth century, when Stone Age trepanned skulls were first identified. Brain surgery during the mid-1860s frequently resulted in infections that killed up to 75% of patients.

Trepanned skulls have been found all over the world, including sites in Peru, China, India, and France, and parts of the Middle East and Africa. While trepanning is an effective surgical technique for relieving pressure on the brain caused by bleeding, most archeologists suspect the operation was carried out in the Stone Age to achieve a different goal. Trepanning, they suspect, was performed to release evil spirits or demons, which the shamans or witch doctors of the time believed produced symptoms of what we know as mental disorders and, perhaps, diseases of the brain. The instruments used in trepanning were likely to have been made of obsidian, a very hard, glasslike, volcanic rock that can hold a very sharp cutting edge. There is also evidence that the end of a wooden stick, hardened by fire and turned back-and-forth rapidly while pressed against the skull may have served as a primitive, but effective, surgical drill.

Neuroscientist and author Elliot Valenstein believes that trepanning did not amount to intentional brain surgery. He quotes from the Latin text by the twelfth-century surgeon Roger of Salerno, who wrote: "For mania and melancholy, the skin of the top of the head should be incised in a cruciate fashion and the skull perforated to allow matter to escape."

A curious example of what might be called pseudo-psychosurgery occurred during the Middle Ages. Some unscrupulous individuals wandered across Europe convincing gullible people that mental disorders were caused by a "stone of madness." To fool others, these quacks faked operating on the brains of mentally ill individuals and, using sleight-of-hand, appeared to produce a real stone from the victim's head, thus "proving" their claim and effecting a "cure." No doubt, these frauds quickly moved on to other towns before their patients showed signs of continuing psychiatric symptoms.

The impetus for developing a radical treatment

Unfortunately, effective treatments for mental illnesses remained unavailable until the second half of the twentieth century. Before then, psychiatric "care" consisted mostly of imprisonment, **neglect**, restraint, and/or punishment. During the eighteenth century, more humane conditions of confinement were introduced, but effective treatments remained unavailable. Physicians were desperate for treatments that might make it easier to control violent and deranged patients.

By the end of the nineteenth century, researchers became aware of the role played by the frontal cortex—a part of the brain located behind the forehead—in behavior control. They discovered from the results of animal experiments and observing humans who suffered damage to this part of the brain that the frontal lobes affect emotions and behavior. This bit of knowledge, combined with the development of effective anesthesia, led to the first modern instances of psychosurgery during the 1890s. A Swiss surgeon named Gottlieb Burkhardt deliberately damaged the frontal lobes of six psychiatric patients in hopes of relieving psychiatric symptoms; at least one of his subjects died and the experimental surgery was discontinued amid criticism from other physicians.

Psychosurgery in the twentieth century

PREFRONTAL LEUCOTOMY. In 1900, an Estonian surgeon, Lodivicus Pussepp, picked up where Burkhardt left off. He cut nerve tracks leading from the frontal lobes to other parts of the brain in psychiatric patients, with unimpressive results. A decade later, he injected tissue-destroying chemicals into the frontal lobes of mentally ill patients through holes drilled over the frontal lobes. Although the procedure accomplished little or nothing in the way of therapy, Pussepp remained optimistic about the ability of this procedure to improve the condition of psychiatric patients. Interest in the frontal lobes as a target for treating mental disorders continued on a small scale until the heyday of psychosurgery began in the 1930s.

In 1935, researchers in the United States reported that damaging the frontal lobes and a nearby region of the brain called the prefrontal cortex could pacify a previously aggressive chimpanzee. A Portuguese **psychiatrist**, Antonio Egas Moniz, learned of these results and recruited neurosurgeon Almeida Lima to operate on some humans suffering from severe psychoses. Moniz's aim was to disconnect nerve pathways running from the frontal lobes to a part of the brain called the thalamus, which is located closer to the center of the brain. By cutting these connections, Moniz hypothesized that he could disconnect a neural circuit that ran from the frontal cortex to the thalamus and then to other parts of the brain's surface. He hoped that interrupting this pathway would disrupt the repetitive thoughts that Moniz believed were responsible for psychotic symptoms.

But as Elliot Valenstein writes in his book *Great and Desperate Cures, The Rise and Decline of Psychosurgery and Other Radical Treatments for Mental Illness*, "Although Moniz' rationale for prefrontal leucotomy was so vague as to constitute no theory at all, his explanation was repeated so often that it—like the emperor's new clothes in Hans Christian Andersen's famous story—acquired a veneer of truth and was accepted (or at least repeated) by many other people." Psychiatrists were so desperate for a treatment for severe cases of mental illness that they allowed themselves to support the use of a procedure that was unproven and increasingly subject to abuse.

Moniz and Lima called their procedure leucotomy. It involved trepanning the skull, one hole on each side of the head, inserting a wire knife and cutting the targeted nerve fibers. Results were mixed enough for Moniz to recommend that the procedure be reserved only for the most seriously mentally ill patients for whom no other course of care or treatment worked. Nevertheless, after 1936, use of the technique spread rapidly, with equally unimpressive results overall. With little evidence of effectiveness and facing opposition from many psychiatrists, particularly psychotherapists, the technique would probably have been abandoned were it not for a pair of American physicians who revived the questionable procedure.

KEY TERMS

Frontal lobes—A region of the brain that influences higher mental functions often associated with intelligence, such as the ability to foresee the consequences of actions, planning, comprehension, and mood.

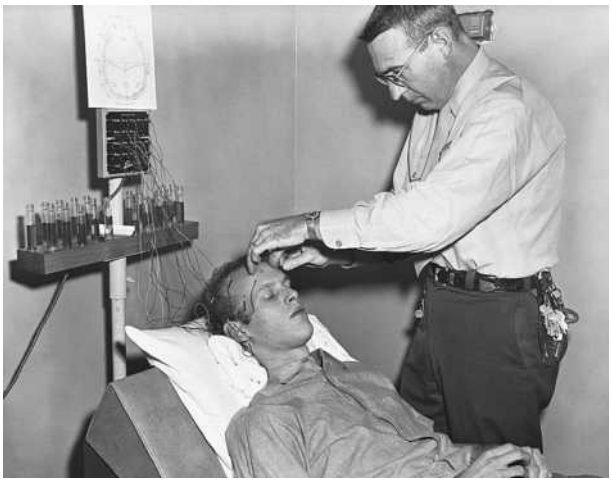
Leucotomy or leukotomy—White matter cutting; severing the white matter of the frontal lobe of the brain.

Lobotomy—A surgical procedure involving the cutting of nerve fiber bundles in the brain.

Trepanation or trepanning—Surgical removal of a piece of the skull to expose the brain.

THE PREFRONTAL LOBOTOMY. American neurologist Walter Freeman and neurosurgeon James Watts began operating on patients in 1936 and soon began aggressively promoting its effectiveness. Eventually, they overcame doubts expressed by their colleagues who somewhat reluctantly accepted the procedure now referred to as prefrontal lobotomy. In 1946, Freeman simplified Moniz's leucotomy procedure, reducing it to a less complicated, less messy, and less time-consuming operation known as the "ice-pick lobotomy." This allowed Freeman to line up patients and, under local anesthesia, tap an ice pick through the thin bone on the roof of their eye sockets. With the ice pick in the brain, Freeman would sweep it back and forth to cut the frontal lobe's connections to the rest of the brain. This in-and-out procedure required no **hospitalization** but many physicians viewed it with alarm. Watts himself refused to cooperate with Freeman after this technique was developed.

Still, in the 1940s, U.S. physicians performed an estimated 18,000 lobotomies. It was equally popular in other countries where more than 50,000 operations were conducted during the same period. Sadly, Moniz's warning was forgotten. The procedure was not reserved for the most hopeless cases but instead applied to "difficult" patients and became a way to control behavior rather than to relieve symptoms of mental disorder. The abuse often bordered on the criminal. Yet, Moniz received the 1949 Nobel Prize for Medicine and Physiology for pioneering the procedure.



Prisoner at Vacaville Penitentiary in California being prepared for a lobotomy in 1961. At the time, many psychiatrists believed that "criminal" behavior was lodged in certain parts of the brain, and lobotomies were frequently practiced on prisoners. *(Ted Streshinsky/ CORBIS. Reproduced by permission.)*

Fortunately, but still too late, critics of the operation began to convince others that there was no scientific proof that lobotomies helped mentally ill patients. It could certainly calm violent patients but it did so at a terrible cost. As one nurse who recently treated an aged patient who had been lobotomized years before said, "You look in her eyes and you see there is no one there." Victims of the procedure lack emotions, ambition, social skills, and the ability to plan. The operation was used to control the mentally ill and others, such as uncontrollable children and political dissidents, whose behavior did not conform to society's standards. Arguments against the procedure were powerful: it permanently and severely damaged the brain and often produced unreactive, lifeless individuals whose personalities were forever destroyed. With the introduction of psychotherapeutic drugs—especially **chlorpromazine** (Thorazine)—in the mid-1950s, lobotomies fell out of fashion.

Psychosurgery today

No one advocates the use of classical lobotomies today as a treatment for mental disorders. However, a small minority of neurologists advocates the use of very precise surgical techniques to produce small lesions in defined areas of the brain to treat rare cases of severe mental illness such as life-threatening depression or incapacitating anxiety or obsessions. However, there is little need for such procedures today. Antipsychotic and antidepressant medications are the treatments of choice for treating mental disorders. Mainstream medicine now classifies psychosurgery as an experimental procedure, and many rules exist to protect patients who might be subjected to it. The majority of mental health professionals believe that psychosurgery is either never justified or should only be considered as a last resort, to be reserved for the most extreme cases of untreatable mental disease when all other therapies have failed.

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Psychotherapy

Definition

The treatment of mental or emotional disorders and adjustment problems through the use of psychological techniques rather than through physical or biological means.

Description

Psychoanalysis, the first modern form of psychotherapy, was called the "talking cure," and the many varieties of therapy practiced today are still characterized by their common dependence on a verbal exchange between the counselor or therapist and the person seeking help. The therapeutic interaction is characterized by mutual trust, with the goal of helping individuals change destructive or unhealthy behaviors, thoughts, and emotions. It is common for experienced therapists to combine several different approaches or techniques.

Psychodynamic approach

Freudian psychoanalysis places emphasis on uncovering unconscious motivations and breaking down

defenses. Therapy sessions may be scheduled once or even twice a week for a year or more. This type of therapy is appropriate when internal conflicts contribute significantly to a person's problems.

Behavioral techniques

In contrast to the psychodynamic approach, behavior-oriented therapy is geared toward helping people see their problems as learned behaviors that can be modified, without looking for unconscious motivations or hidden meanings. According to the theory behind this approach, once behavior is changed, feelings will change as well. Probably the best-known type of behavioral therapy is **behavior modification**, which focuses on eliminating undesirable habits by providing positive **reinforcement** for the more desirable behaviors.

Another behavioral technique is **systematic desensitization**, in which people are deliberately and gradually exposed to a feared object or experience to help them overcome their fears. A person who is afraid of dogs may first be told to visualize a dog, then is given a stuffed toy dog, then exposed to a real dog seen at a distance, and eventually forced to interact with a dog at close range. Relaxation training is another popular form of behavior therapy. Through such techniques as deep breathing, visualization, and progressive muscle relaxation, clients learn to control fear and anxiety.

Cognitive methods

Some behavior-oriented therapy methods are used to alter not only overt behavior, but also the thought patterns that drive it. This type of treatment is known as **cognitive-behavioral therapy** (or just cognitive therapy). Its goal is to help people break out of distorted, harmful patterns of thinking and replace them with healthier ones. Common examples of negative thought patterns include magnifying or minimizing the extent of a problem; "all or nothing" thinking (i.e., a person regards himself as either perfect or worthless); overgeneralization (arriving at broad conclusions based on one incident, for example); and personalization (continually seeing oneself as the cause or focus of events).

In cognitive-behavioral therapy, a therapist may talk to the client, pointing out illogical thought patterns, or use a variety of techniques, such as thought substitution, in which a frightening or otherwise negative thought is driven out by substituting a pleasant thought in its place. Clients may also be taught to use positive self-talk, a repetition of positive affirmations. Cognitive therapy usually takes a longer amount of time as it treats more serious problems.

Couples therapy

Couples therapy focuses on the relationship between two people, typically who have a romantic or sexual connection. The therapy aims to concentrate on the problems of the relationship and make each partner feel that they have an equal role. The therapy can be administered by either a male or female therapist, but many couples feel that having both a male and female therapist in the session is beneficial.

Family and group therapy

Family therapy has proven effective in treating a number of emotional and adjustment problems. While the client's immediate complaint is the initial focus of attention, the ultimate goal of family therapy is to improve the interaction between all family members and enhance communication and coping skills on a long-term basis (although therapy itself need not cover an extended time period). **Group therapy**, which is often combined with individual therapy, offers the support and companionship of other people experiencing the same or similar problems and issues.

Therapy is terminated when the treatment goals have been met or if the client and/or therapist conclude that it is not working. It can be effective to phase out treatment by gradually reducing the frequency of therapy sessions. Even after regular therapy has ended, the client may return for periodic follow-up and reassessment sessions.

Psychotherapy integration

Definition

Psychotherapy integration is defined as an approach to **psychotherapy** that includes a variety of attempts to look beyond the confines of single-school approaches in order to see what can be learned from other perspectives. It is characterized by an openness to various ways of integrating diverse theories and techniques. Psychotherapy integration can be differentiated from an eclectic approach in that an eclectic approach is one in which a therapist chooses interventions because they work (the therapist relies solely on supposed efficacy) without looking for a theoretical basis for using the technique. The rationale of efficacy is reasonable, but it often is based on imprecise memories of past experience without any reference to theory or research data. In contrast, psychotherapy integration attends to the relationship between theory and technique.

Description

The term psychotherapy integration has been used in several different ways. The term has been applied to a Common Factors approach to understanding psychotherapy, to Assimilative Integration, to Technical Integration, and to Theoretical Integration.

Common Factors

Common Factors refers to aspects of psychotherapy that are present in most, if not all, approaches to therapy. These techniques cut across all theoretical lines and are present in all psychotherapeutic activities. Because the techniques are common to all approaches to psychotherapy, the name Common Factors has been given to this variety of psychotherapy integration. There is no standard list of common factors, but if a list were to be constructed, it surely would include:

- a therapeutic alliance established between the patient and the therapist
- exposure of the patient to prior difficulties, either in imagination or in reality
- a new corrective emotional experience that allows the patient to experience past problems in new and more benign ways
- expectations by both the therapist and the patient that positive change will result from the treatment
- therapist qualities, such as attention, empathy, and positive regard, that are facilitative of change in treatment
- the provision by the therapist to the patient of a reason for the problems that are being experienced

No matter what kind of therapy is practiced, each of these common factors is present. It is difficult to imagine a treatment that does not begin with the establishment of a constructive and positive therapeutic alliance. The therapist and the patient agree to work together and they both feel committed to a process of change occurring in the patient. Within every approach to treatment, the second of the common factors, the exposure of the patient to prior difficulties, is present. In some instances the exposure is in vivo (occurs in real life), and the patient will be asked directly to confront the source of the difficulties. In many cases, the exposure is verbal and in imagination. However, in every case, the patient must express those difficulties in some manner and, by doing so, re-experiences those difficulties through this exposure. In successful treatment, the exposure usually is followed by a new corrective emotional experience. The corrective emotional experience refers to a situation in which an old difficulty is re-experienced in a new and more positive way. As the patient re-experiences the problem in a new way, that problem can be mastered and the patient can move on to a more successful adjustment.

Having established a therapeutic alliance, and being exposed to the problem in a new and more positive context, both the therapist and the patient always expect positive change to occur. This faith and hope is a common factor that is an integral part of successful therapy. Without this hope and expectation of change, it is unlikely that the therapist can do anything that will be useful, and if the patient does not expect to change, it is unlikely that he or she will experience any positive benefit from the treatment. The therapist must possess some essential qualities, such as paying attention to the patient, being empathic with the patient, and making his positive regard for the patient clear in the relationship. Finally, the patient must be provided with a credible reason for the problems that he or she is undergoing. This reason is based in the therapist's theory of personality and change. The same patient going to different therapists may be given different reasons for the same problem. It is interesting to speculate as to whether the reason must be an accurate one or whether it is sufficient that it be credible to the patient and not remarkably at variance with reality. As long as the reason is credible and the patient has a way of understanding what previously had been incomprehensible, that may be sufficient for change to occur.

Assimilative Integration

The second major approach to psychotherapy integration is Assimilative Integration. Assimilative Integration is an approach in which the therapist has a commitment to one theoretical approach but also is willing to use techniques from other therapeutic approaches.

As an example, a therapist may try to understand patients in terms of psychodynamic theory, because he or she finds this most helpful in understanding what is going on in the course of the treatment. However, the therapist may also recognize that there are techniques that are not suggested by psychodynamic theory that work very well, and these may then be used in the treatment plan. The psychodynamic therapist can occasionally use cognitive-behavioral techniques such as homework, and may occasionally use humanistic approaches, such as a two-chair technique, but always retains a consistent psychodynamic understanding. The treatment can take place in a way that is beneficial to the patient and is not bound by the restrictions of the therapist's favorite way of intervening. The patient may not be aware that integration is taking place, but he or she does feel that a consistent approach is being maintained. Most patients are not familiar with theory, don't realize that different techniques are generat-

ed by different theoretical understandings, and only are concerned with whether or not the treatment is helpful.

Inherent in psychotherapy integration is the conviction that there is no one approach to therapy that is suitable to every patient. Both in single-school approaches and in psychotherapy integration, the treatment must be suitable for the individual patient. In making the treatment suitable for the individual patient, the therapist must understand the patient, and that establishes a place for theory. Assimilative Integration is particularly useful in that theory helps in the understanding of the needs of the patient, but then several different approaches to technique can help to design a treatment that fits that particular understanding. The treatment plan then must undergo continuous revision as the understanding of the patient gets fuller and deeper over the course of the treatment.

Technical Eclecticism

Technical Eclecticism is a variation of Assimilative Integration and is most common among those practitioners who refer to themselves as eclectic. In Technical Eclecticism, the same diversity of techniques is displayed as in Assimilative Integration, but there is no unifying theoretical understanding that underlies the approach. Rather, the therapist relies on previous experience and on knowledge of the theoretical and research literature to choose interventions that are appropriate for the patient.

The obvious similarity between Assimilative Integration and Technical Eclecticism is that both rely on a wide variety of therapeutic techniques, focusing on the welfare of the patient rather than on allegiance to any particular school of psychotherapy. The major difference between the two is that Assimilative Integration is bound by a unifying theoretical understanding whereas Technical Eclecticism is free of theory and relies on the experience of the therapist to determine the appropriate interventions.

Theoretical Integration

The fourth approach to psychotherapy integration is called Theoretical Integration. This is the most difficult level at which to achieve integration because it requires integrating theoretical concepts from different approaches, and these approaches may differ in their fundamental philosophy about human behavior. Whereas Assimilative Integration begins with a single theory and brings together techniques from different approaches, Theoretical Integration tries to bring together those theoretical approaches themselves and then to develop what in physics is referred to as a "Grand Unified Theory." Neither psychotherapists nor physicists have been successful to date in producing a Grand Unified Theory. It is difficult to imagine a theory that really can combine an approach that has one philosophical understanding with another approach that has a different philosophical understanding. For example, a psychodynamic approach believes that an early difficulty leads to a pattern of behavior that is repetitive, destructive, and nearly impossible to resolve. In contrast, behavior therapy sees problems as much more amenable to change. This difference may represent a basic incompatibility between the two theories. Therefore, theoretical integration would be faced with the task of integrating a theory about the stability of behavior with a theory about the ready changeability of behavior, and unless this obstacle can be overcome, Theoretical Integration will not be achieved.

Conclusions

In any case, the general point in three of these approaches, Common Factors, Assimilative Integration, and Theoretical Integration, is that there is a clear value to the role of theory in psychotherapy integration, whether the theory deals with the way integration works (Theoretical Integration), the framework that governs the choice of interventions (Assimilative Integration), or the organizing principle for understanding the Common Factors that are present in all psychotherapy. The fourth approach, Technical Eclecticism, is not concerned with theory, but does view the benefit of the patient to be of more significance than the adherence to any single theory.

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George Stricker, Ph.D.

Pyromania

Definition

Pyromania is defined as a pattern of deliberate setting of fires for pleasure or satisfaction derived from the relief of tension experienced before the fire-setting. The name of the disorder comes from two Greek words that mean "fire" and "loss of reason" or "madness." The clinician's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders****, also known as the* DSM, classifies pyromania as a disorder of impulse control, meaning that a person diagnosed with pyromania fails to resist the impulsive desire to set fires—as opposed to the organized planning of an arsonist or terrorist.

The position of the **impulse-control disorders** as a group within the *DSM-IV-TR* (*DSM,* fourth edition, text revised) diagnostic framework, however, has been questioned by some psychiatrists. The differential **diagnosis** of pyromania and the other five disorders listed under the heading of impulse-control problems (**intermittent explosive disorder**, **kleptomania**, pathological gambling, **trichotillomania**, and impulse-control disorder not otherwise specified) includes **antisocial personality disorder** (ASPD), mood disorders, conduct disorders (among younger patients), and temporal lobe epilepsy. It is not clear whether the impulse-control disorders derive from the same set of causes as ASPD and mood disorders, or whether "impulse-control disorder" is simply an all-inclusive category for disorders that are otherwise difficult to classify. Some American researchers would prefer to categorize pyromania and the other disorders of impulsivity as a subset of the obsessive-compulsive spectrum.

In addition, the relationship between pyromania in adults and firesetting among children and adolescents is not well defined as of 2002. Although pyromania is considered to be a rare disorder in adults, repeated firesetting at the adolescent level is a growing social and economic problem that poses major risks to the health and safety of other people and the protection of their property. In the United States, fires set by children and adolescents are more likely to result in someone's death than any other type of household disaster. The National Fire Protection Association stated that for 1998, fires set by juveniles caused 6,215 deaths, 30,800 injuries, and $11 billion in property damage. It is significant that some European psychiatrists question the *DSM-IV-TR* definition of pyromania as a disorder of impulse control precisely because of the connection they find between adolescent firesetting and similar behavior in adults. One team of German researchers remarked, "Repeated firesetting, resulting from being fascinated by fire, etc., may be less a disturbance of impulse control but rather the manifestation of a psychoinfantilism, which, supported by alcohol abuse, extends into older age." Pyromania is considered a relatively rare impulse-control disorder in the adult population in North America.

Description

Firesetting in children and adolescents

Although most cases of firesetting in the United States involve children or adolescents rather than adults, the *DSM-IV-TR* criteria for pyromania are difficult to apply to this population. Most younger firesetters are diagnosed as having conduct disorders rather than pyromania as *DSM-IV-TR* defines it; significantly, most of the psychiatric literature dealing with this age group speaks of "firesetting" rather than using the term "pyromania" itself.

Some observers have attempted to classify children and adolescents who set fires as either pathological or nonpathological. Youngsters in the former group are motivated primarily by curiosity and the desire to experiment with fire; some are teenagers playing "scientist." Most are between five and 10 years of age, and do not understand the dangers of playing with fire. Few of them have major psychological problems.

Those who are considered to be pathological firesetters have been further subdivided into five categories, which are not mutually exclusive:

- Firesetting as a cry for help. Youngsters in this category set fires as a way of calling attention to an intrapsychic problem such as depression, or an interpersonal problem, including parental separation and divorce or physical and sexual abuse.
- Delinquent firesetters. Firesetters in this category are most likely to be between the ages of 11 and 15. Their firesetting is part of a larger pattern of aggression, and may include vandalism and hate crimes. They are, however, more likely to damage property with their firesetting than to injure people.
- Severely disturbed firesetters. These youths are often diagnosed as either psychotic or paranoid, and appear to be reinforced by the sensory aspects of fire setting. Some set fires as part of **suicide** attempts.
- Cognitively impaired firesetters. This group includes youngsters whose impulse control is damaged by a neurological or medical condition such as fetal alcohol syndrome.
- Sociocultural firesetters. Youngsters in this group are influenced by antisocial adults in their community, and set fires in order to win their approval.

Pyromania in adults

Pyromania in adults resembles the other disorders of impulse control in having a high rate of comorbidity with other disorders, including substance abuse disorders, **obsessive-compulsive disorder** (OCD), anxiety disorders, and mood disorders. As of 2002, however, few rigorously controlled studies using strict diagnostic criteria have been done on adult patients diagnosed with pyromania or other impulse-control disorders.

Causes and symptoms

Causes

Most studies of causation regarding pyromania have focused on children and adolescents who set fires. Early studies in the field used the categories of Freudian **psychoanalysis** to explain this behavior. Freud had hypothesized that firesetting represented a regression to a primitive desire to demonstrate power over nature. In addition, some researchers have tried to explain the fact that pyromania is predominantly a male disorder with reference to Freud's notion that fire has a special symbolic relationship to the male sexual urge. A study done in 1940 attributed firesetting to fears of castration in young males, and speculated that adolescents who set fires do so to gain power over adults. The 1940 study is important also because it introduced the notion of an "ego triad" of firesetting, **enuresis** (bed-wetting), and cruelty to animals as a predictor of violent behavior in adult life. Subsequent studies have found that a combination of firesetting and cruelty to animals is a significant predictor of violent behavior in adult life, but that the third member of the triad (bed-wetting) is not.

INDIVIDUAL. The causes of firesetting among children and teenagers are complex and not well understood as of 2002. They can, however, be described in outline as either individual or environmental. Individual factors that contribute to firesetting include:

- Antisocial behaviors and attitudes. Adolescent firesetters have often committed other crimes, including forcible rape (11%), nonviolent sexual offenses (18%), and vandalism of property (19%).
- Sensation seeking. Some youths are attracted to firesetting out of boredom and a lack of other forms of recreation.
- Attention seeking. Firesetting becomes a way of provoking reactions from parents and other authorities.
- Lack of social skills. Many youths arrested for firesetting are described by others as "loners" and rarely have significant friendships.
- Lack of fire-safety skills and ignorance of the dangers associated with firesetting.

There are discrepancies between adult researchers' understanding of individual factors in firesetting and reports from adolescents themselves. One study of 17 teenaged firesetters, 14 males and three females, found six different self-reported reasons for firesetting: revenge, crime concealment, peer group pressure, accidental firesetting, **denial** of intention, and fascination with fire. The motivations of revenge and crime concealment would exclude these teenagers from being diagnosed with pyromania according to *DSM-IV-TR* criteria.

ENVIRONMENTAL. Environmental factors in adolescent firesetting include:

- Poor supervision on the part of parents and other significant adults.

KEY TERMS

Arson—The deliberate setting of fires for criminal purposes, usually to collect insurance money or to cover up evidence of another crime. It is distinguished from pyromania by its connection with planning and forethought rather than failure of impulse control.

Comorbidity—Association or presence of two or more mental disorders in the same patient. A disorder that is said to have a high degree of comorbidity is likely to occur in patients diagnosed with other disorders that may share or reinforce some of its symptoms.

Delusion—A false belief that is resistant to reason or contrary to actual fact. Common delusions include delusions of persecution, delusions about one's importance (sometimes called delusions of grandeur), or delusions of being controlled by others. Pyromania is excluded as a diagnosis if the patient is setting fires on the basis of a delusion.

Kleptomania—A disorder of impulse control characterized by repeated stealing or shoplifting of items that the person does not need.

Spontaneous remission—Recovery from a disease or disorder that cannot be attributed to medical or psychiatric treatments.

Trichotillomania—A disorder marked by repeated pulling and tugging of one's hair, usually resulting in noticeable hair loss on the scalp or elsewhere on the body.

- Early learning experiences of watching adults use fire carelessly or inapproriately.
- Parental **neglect** or emotional uninvolvement.
- Parental psychopathology. Firesetters are significantly more likely to have been physically or sexually abused than children of similar economic or geographic backgrounds. They are also more likely to have witnessed their parents abusing drugs or acting violently.
- Peer pressure. Having peers who smoke or play with fire is a risk factor for a child's setting fires himself.
- Stressful life events. Some children and adolescents resort to firesetting as a way of coping with crises in their lives and/or limited family support for dealing with crises.

Symptoms

Firesetting among children and adolescents and pyromania in adults may be either chronic or episodic; some persons may set fires frequently as a way of relieving tension, others apparently do so only during periods of unusual **stress** in their lives.

In addition to the outward behavior of firesetting, pyromania in adults has been associated with symptoms that include depressed mood, thoughts of suicide, repeated conflicts in interpersonal relationships, and poor ability to cope with stress.

Demographics

The true incidence of pyromania in the general American population remains unknown. Of the six impulse-control disorders listed in *DSM-IV-TR,* only trichotillomania and pathological gambling appear to be common in the general population (4% and 3% respectively). Pyromania, like intermittent explosive disorder and pathological gambling, is diagnosed more frequently in men than in women.

Repeated firesetting appears to be more common in children and adolescents than in adult males. In addition, the incidence appears to be rising in these younger age groups: in 1992, males 18 and younger accounted for 40% of arrests for firesetting; in 2001, they accounted for 55%. As of 1999, 89% of juvenile arrests for firesetting involved males; 79% involved Caucasian juveniles. Within the group of male juveniles, 67% were younger than age 15, and 35% younger than age 12.

Less is known about the incidence of pyromania among adults. Some researchers have theorized that children and adolescents attracted to firesetting when they are younger "graduate" in adult life to more serious crimes with a "macho" image, including serial rape and murder. A number of serial killers, including David Berkowitz, the "Son of Sam" killer, and David Carpenter, the so-called Trailside Killer of the San Francisco Bay area, turned out to have been firesetters in their adolescence. David Berkowitz admitted having started more than 2,000 fires in Brooklyn-Queens in the early 1970s.

Another hypothesis regarding pyromania in adults is that it is more likely to emerge in the form of workplace violence. The recent rapid increase in the number of workplace killings and other violent incidents— a 55% rise between 1992 and 1996— is a source of great concern to employers. One of the complications in the situation is that the Americans with Disabilities Act (ADA), passed by Congress in 1990, forbids employers to discriminate against workers with mental or physical disabilities as long as they are qualified to perform their job. Since 1996, the Equal Employment Opportunities Commission (EEOC) reports that the third-largest category of civil rights claims alleging employer discrimination concerns psychiatric disabilities. In 1997, the EEOC issued a set of guidelines on the ADA and psychiatric disabilities. Significantly, the EEOC excluded pyromania (along with kleptomania, compulsive gambling, disorders of sexual behavior, and the use of illegal drugs) from the list of psychiatric conditions for which employers are expected to make "reasonable accommodation." The EEOC's exclusion of pyromania indicates that workers with this disorder are considered a sufficiently "direct threat" to other people and property that employers are allowed to screen them out during the hiring process.

Diagnosis

DSM-IV-TR specifies six criteria that must be met for a patient to be diagnosed with pyromania:

- The patient must have set fires deliberately and purposefully on more than one occasion.
- The patient must have experienced feelings of tension or emotional arousal before setting the fires.
- The patient must indicate that he or she is fascinated with, attracted to, or curious about fire and situations surrounding fire (for example, the equipment associated with fire, the uses of fire, or the aftermath of firesetting).
- The patient must experience relief, pleasure, or satisfaction from setting the fire or from witnessing or participating in the aftermath.
- The patient does not have other motives for setting fires, such as financial motives; ideological convictions (such as terrorist or anarchist political beliefs); anger or revenge; a desire to cover up another crime; **delusions** or **hallucinations**; or impaired judgment resulting

from substance abuse, **dementia**, **mental retardation**, or traumatic **brain** damage.

- The fire setting cannot be better accounted for by antisocial personality disorder, a **conduct disorder**, or a manic episode.

Diagnosis of pyromania is complicated by a number of factors; one important factor is the adequacy of the diagnostic category itself. As was mentioned earlier, some psychiatrists are not convinced that the impulse-control disorders should be identified as a separate group, in that problems with self-control are part of the picture in many psychiatric disorders. **Bulimia nervosa**, **borderline personality disorder**, and antisocial personality disorder are all defined in part by low levels of self-control.

Another complication in diagnosis is the lack of experience on the part of mental health professionals in dealing with firesetting. In many cases they are either unaware that the patient is repeatedly setting fires, or they regard the pattern as part of a cluster of antisocial or dysfunctional behaviors.

Treatments

Children and adolescents

Treatment of children and adolescents involved with repeated firesetting appears to be more effective when it follows a case-management approach rather than a medical model, because many young firesetters come from chaotic households. Treatment should begin with a structured interview with the parents as well as the child, in order to evaluate stresses on the family, patterns of supervision and discipline, and similar factors. The next stage in treatment should be tailored to the individual child and his or her home situation. A variety of treatment approaches, including problem-solving skills, anger management, communication skills, aggression replacement training, and cognitive restructuring may be necessary to address all the emotional and cognitive issues involved in each case.

Adults

Pyromania in adults is considered difficult to treat because of the lack of insight and cooperation on the part of most patients diagnosed with the disorder. Treatment usually consists of a combination of medication— usually one of the selective serotonin reuptake inhibitors—and long-term insight-oriented **psychotherapy**.

Prognosis

The prognosis for recovery from firesetting among children and adolescents depends on the mix of individual and environmental factors involved. Current understanding indicates that children and adolescents who set fires as a cry for help, or who fall into the cognitively impaired or sociocultural categories, benefit the most from therapy and have fairly positive prognoses. The severely disturbed and delinquent types of firesetters have a more guarded outlook.

The prognosis for adults diagnosed wih pyromania is generally poor. There are some cases of spontaneous remission among adults, but the rate of spontaneous recovery is not known.

Prevention

Prevention of pyromania requires a broad-based and flexible approach to treatment of children and adolescents who set fires. In addition to better assessments of young people and their families, fire-safety education is an important preventive strategy that is often overlooked.

In addition to preventive measures directed specifically at firesetting, recent research into self-control as a general character trait offers hope that it can be taught and practiced like many other human skills. If programs could be developed to improve people's capacity for self-control, they could potentially prevent a wide range of psychiatric disorders.

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Q

Quazepam

Definition

Quazepam belongs to a class of drugs called benzodiazepines. These drugs ease anxiety and slow the central nervous system. In the United States quazepam is sold under brand name Doral.

Purpose

Quazepam is approved by the United States Food and Drug Administration for the treatment of **insomnia**.

Description

Quazepam is unique in its drug properties in two ways. Several medications from the same class of drugs have an effect called rebound insomnia. This means that the insomnia becomes worse than the original insomnia when the drug is used for extended periods. Quazepam has a minimal tendency to cause rebound insomnia. Secondly, quazepam is eliminated from the body slowly. This gives quazepam advantage over certain other medications in the benzodiazepine class, such as **alprazolam** or halazepam, in that patients do not experience early-morning insomnia, since there is still enough medication to induce sleep in the very early morning hours.

Quazepam's sedating effect that reduces insomnia lasts only for about four weeks of continuous use. The medication is most effective for an intermediate-term treatment of insomnia (two weeks), rather than a long duration of treatment of over four weeks. Hence, long-term treatment for insomnia with quazepam should be avoided.

Quazepam comes in 7.5-mg and 15-mg tablets.

Recommended dosage

Effective doses of quazepam for the treatment of insomnia range from 7.5 mg to 30 mg at bedtime. Most patients start by taking 15 mg at bedtime. Adjustments from this dosage can be made as determined by individual. In some patients, a dosage as low as 7.5 mg is sufficient to reduce insomnia.

Elderly patients (over age 65) should receive a reduced dosage of 7.5 mg, because it takes a longer time to eliminate the drug from their bodies. Because quazepam is eliminated by the liver, dosage reduction may be necessary in patients with liver problems.

Precautions

Patients who have a condition known as sleep apnea should not use quazepam. This condition involves episodes of breathing difficulty and oxygen deficiency that occur throughout the night. Patients who are pregnant or who had an allergic reaction to quazepam should not take quazepam

People who need to remain mentally alert such as those who are driving or operating dangerous machinery, need to take quazepam with caution as it may cause drowsiness. This effect is intensified when quazepam is taken with alcohol. It is best not to drink alcoholic beverages while taking quazepam. Patients with compromised respiratory function (breathing problems), as well as patients with a history of drug or alcohol abuse, should closely be monitored during the short-term treatment with quazepam.

Side effects

The effects of quazepam taken at bedtime may last, or hang over, into the next day. This is the most common side effect of quazepam. The symptoms of this condition include drowsiness, daytime sleepiness, slurred speech, and mental sluggishness. This effect is dose related, and seems to occur most frequently in patients taking 30-mg doses. These effects are experienced less commonly with the 15-mg dose, but this dose may not be effective in

KEY TERMS

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

eliminating insomnia some patients. Some people experience headache and dizziness when taking quazepam.

A small number of patients experience dry mouth, weight loss, abnormal taste perception, abdominal pain, nausea, vomiting, and either diarrhea or constipation due to quazepam. These effects occur in about 1% to 10% of people taking the drug.

Side effects that occur in less than 1% of patients include skin problems, such as rash or skin inflammation, muscle cramps, rigidity, and blurred vision.

Interactions

Cimetidine (Tagamet) and ketoconazole increase the levels of quazepam in the body, potentially causing toxicity or increased side effects.

Theophylline decreases the effectiveness of quazepam. **Valerian**, **kava kava**, and alcohol cause increased central nervous depression, which may increase sedation, drowsiness, and slowed reflexes if used while taking quazepam.

Resources

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Quetiapine

Definition

Quetiapine is an atypical antipsychotic drug used to treat symptoms of **schizophrenia**. It is available with a prescription under the trade name Seroquel.

Purpose

Quetiapine is classified as an atypical antipsychotic. It is used to treat psychotic disorders such as schizophrenia.

Description

Quetiapine is thought to modify the actions of several chemicals in the **brain**. It is chemically related to another atypical antipsychotic agent, **clozapine**, but differs both chemically and pharmacologically from the earlier phenothiazine antipsychotics.

It is available 25-mg, 100-mg, and 200-mg tablets.

Recommended dosage

Initially, a dosage of 25 mg should be taken twice a day. Each dose should be increased by 25-50 mg increments every three to four days until a target dose of 300-400 mg per day, administered in two or three divided doses, is achieved. It is not known whether doses higher than 800 mg per day are safe.

Precautions

Caution should be used in patients with heart disease because the drug may cause blood pressure to fall too low resulting in dizziness, rapid heartbeat, or fainting.

Quetiapine may cause liver damage. As a result, patients should notify their health care provider if they experience flu-like symptoms, notice yellowing of their skin or eyes, or experience abdominal pain. Liver function should be assessed periodically. The drug should be used cautiously in people with a history of liver disease or alcoholic cirrhosis.

Quetiapine may alter the function of the thyroid gland. Those taking supplements for low thyroid function may require dosage adjustments in their thyroid medication.

Quetiapine may increase cholesterol levels and contribute to the formation of cataracts. Because of this possibility, cholesterol levels should be checked periodically and yearly eye exams should be performed.

Quetiapine should be used carefully in those with a history of seizure disorders because it may increase the tendency to have **seizures**.

Quetiapine may cause extreme drowsiness and should be used carefully by people who need to be mentally alert.

Quetiapine should not be taken while pregnant or breast-feeding.

Side effects

Relatively common side effects that accompany quetiapine include drowsiness, dizziness, rash, dry mouth, **insomnia**, **fatigue**, muscular weakness, anorexia, blurred vision, some loss of muscular control, and amenorrhea (lack of menstruation) in women.

Dystonia (difficulty walking or moving) may occur with quetiapine use. This condition may subside in 24 to 48 hours even when the person continues taking the drug and usually disappears when quetiapine is discontinued.

Quetiapine use may lead to the development of symptoms that resemble Parkinson's disease. These symptoms may include a tight or mask-like expression on the face, drooling, tremors, pill-rolling motions in the hands, cogwheel rigidity (abnormal rigidity in muscles characterized by jerky movements when the muscle is passively stretched), and a shuffling gait. Taking anti-Parkinson drugs **benztropine** mesylate or **trihexyphenidyl** hydrochloride along with the quetiapine usually controls these symptoms.

Quetiapine has the potential to produce a serious side effect called **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements that may appear late in therapy and may not disappear even after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage of quetiapine. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period.

An occasionally reported side effect of quetiapine is neuroleptic malignant syndrome. This is a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia (fast heartbeat), and arrhythmias (irregular heartbeat). People who think they may be experiencing any side effects from this or any other medication should talk to their physician promptly.

Interactions

Quetiapine may be less effective when it is taken with drugs like **carbamazepine** (Tegretol), phenytoin (Dilantin), rifampin (Rifadin), **barbiturates**, **thioridazine** (Mellaril), or corticosteroids such as prednisolone, methlylprednisolone, prednisone, and dexamethasone because these drugs increase the breakdown of quetiapine in the liver causing lower-than-normal levels of the drug.

Antifungal drugs such as fluconazole (Diflucan) or ketoconazole (Nizerol), antibiotics such as erythromycin or clarithromycin (Biaxin), and cimetidine (Tagamet), because these drugs may decrease the breakdown of quetiapine in the liver causing higher-than-normal levels of the drug.

Any drug that causes drowsiness may lead to decreased mental alertness and impaired motor skills when taken with Quetiapine. Some examples include alcohol, antidepressants such as **imipramine** (Tofranil) or **paroxetine** (Paxil), antipsychotics such as thioridazine (Mellaril), and some antihistamines.

KEY TERMS

Neuroleptic malignant syndrome—An unusual but potentially serious complication that develops in some patients who have been treated with antipsychotic medications. NMS is characterized by changes in blood pressure, altered states of consciousness, rigid muscles, and fever. Untreated NMS can result in coma and death.

Parkinsonian—Related to symptoms associated with Parkinson's disease, a nervous system disorder characterized by abnormal muscle movement of the tongue, face, and neck, inability to walk or move quickly, walking in a shuffling manner, restlessness, and/or tremors.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

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R

Rational behavior therapy *see* **Rational emotive therapy**

Rational emotive therapy

Definition

Rational emotive therapy (RET) is a psychotherapeutic approach which proposes that unrealistic and irrational beliefs cause many emotional problems.

Purpose

RET is a form of **cognitive-behavioral therapy** (CBT). The primary focus of this treatment approach is to suggest changes in thinking that will lead to changes in behavior, thereby alleviating or improving symptoms. The therapy emphasizes changing irrational thinking patterns that cause emotional distress into thoughts that are more reasonable and rational. RET can be used to treat people affected from disorders such as anxiety, depression and stess.

Precautions

There are no major precautions, except that persons entering treatment must be willing to change behaviors that promote symptoms.

Description

Rational emotive therapy was developed by Albert Ellis in the mid-1950s. Ellis proposed that people become unhappy and develop self-defeating habits because of unrealistic or faulty beliefs. In research reports from Ellis in 1979 and 1987 he introduced the model that most irrational beliefs originate from three core ideas, each one of which is unrealistic. These three core and unrealistic views include: 1) I must perform well to be approved of by others who are perceived significant; 2) you must treat me fairly—if not, then it is horrible and I cannot bear it; 3) conditions must be my way and if not I cannot stand to live in such a terrible and awful world. These irrational thoughts can lead to **grief** and needless suffering.

As a therapy, RET is active. The RET therapist strives to change irrational beliefs, challenge thinking, and promote rational self-talk, and various strategies are used to achieve these goals. These strategies may include: disputing irrational beliefs (the therapist points out how irrational it would be for a client to believe he or she had to be good at everything to be considered a worthwhile person), reframing (situations are viewed from a more positive angle), problem solving, role-playing, **modeling**, and the use of humor. The client may also be requested to complete certain exercises at home, and **bibliotherapy** (reading about the disorder) may also be used as components of RET.

Preparation

Before a client begins RET, he or she may undergo an assessment with the therapist. This assessment is called a biopsychosocial assessment, consisting of a structured interview. The questions and information-gathering during this assessment typically cover areas such as past medical and psychological history, family and social history, sex and drug history, employment and education history and criminal history. The interview provides information for a **diagnosis** or a tentative diagnosis that requires further testing or consultation.

Aftercare

Aftercare may or may not be indicated. This is usually decided on between the patient and mental health practitioner. Aftercare follow-up may be recommended if the affected person is at risk of relapse behaviors (returning to old behaviors that the client had sought to change).

Risks

There are no real risks associated with RET. There is a possibility that treatment may not benefit the affected person. This possibility becomes more likely for patients who have multiple psychological disorders.

Normal results

The person undergoing RET will begin to understand the repetitive patterns of irrational thoughts and disruption caused by symptoms. The individual in therapy will develop skills to improve his or her specific problems, and usual results include improved self-esteem and the development of a sense that life events change and that outcomes may not always be favorable.

Abnormal results

There are no abnormal results per se, but persons who are unwilling to change and adhere to treatment recommendations may not gain any new beneficial behaviors.

Resources

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ORGANIZATIONS

The Albert Ellis Institute. 45 East 65th Street, New York, NY 10021. Telephone: (800) 323-4738.

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Reactive attachment disorder of infancy or early childhood

Definition

In reactive attachment disorder, the normal bond between infant and parent is not established or is broken. Infants normally "bond" or form an emotional attachment, to a parent or other caregiver by the eighth month of life. From about the second through the eighth month, most infants will respond to attention from a variety of caregivers, if the caregivers are familiar. By the eighth month, however, normal infants have established a strong emotional preference for one or two primary caregivers. They are distressed if separated from these caregivers for even a few hours, even if another familiar person is present. If this bonding process is interfered with, it can have severe emotional and physical consequences for the child.

Reactive attachment disorder is sometimes called a post-traumatic disorder.

Description

In reactive attachment disorder, an infant or young child has not formed an emotional bond with a parent or other caregiver. This affects the child's ability to interact normally with others. The child may have severe emotional and social problems that extend into adulthood. There may be learning problems and physical problems such as slow growth and failure to develop as expected.

Causes and symptoms

Causes

An infant does not know how to form an emotional attachment to another person, any more than it knows how to feed or clean itself. Bonding is a necessary developmental step in a baby's growth. It occurs as the infant is cared for, talked to, played with, and comforted consistently. This helps the infant feel like it knows what will happen every time it sees a certain person. When this process is interfered with, the infant may never learn how to trust or love.

Many things can interfere with the bonding process:

- Loss of parents. The most common cause of reactive attachment disorder is being orphaned or put in foster care at a very early age. The infant may receive care from many people or be moved from place to place often. A bond to a single consistent caregiver cannot be formed.
- **Neglect** or impaired caregiving. If the infant is not cared for consistently, it will not learn to trust. This includes emotional neglect, where the caregivers may keep the baby clean and fed, but do not allow time for play and bonding. Very often this occurs when the parent or caregiver has a problem that prevents him or her from giving adequate, consistent attention to the infant. Such problems include major depression, **psychosis**, drug or alcohol abuse, **mental retardation**, physical illness, and poverty. The parent may also have been a neglected child or may be very young themselves and simply not know how to parent adequately.
- **Abuse** or pain. Even if an infant is getting love and attention some of the time, it may not learn to attach if it comes to expect pain on occasion from the caregiver.

Illness or pain that the caregiver cannot ease can have the same effect.

In disrupted families with more than one child, one child may have reactive attachment disorder while others do not. It is not clear what role personality plays in this problem.

Symptoms

Infants with this problem often resist being held or touched. They may seem sleepy or "slow." They may not seem aware of what's going on around them. They may be slow to gain weight. On the other hand, some appear to be overly aware and nervous.

Young children may seem withdrawn and passive. They may ignore others or respond to others in odd ways. Some may seem overly familiar with strangers and touch or cling to people they've just met. However, they lack empathy for others. Their behavior comes across to others as needy and strange, unlike the normal friendliness of children.

Other symptoms of reactive attachment disorder in children can include the following:

- inability to learn from mistakes (poor cause-and-effect thinking)
- learning problems or delays in learning
- impulsive behavior
- abnormal speech patterns
- destructive or cruel behavior

Demographics

The prevalence of reactive attachment disorder has been estimated at 1% of all children under the age of five. Children orphaned at a young age have a much higher likelihood of this problem.

Diagnosis

The standard manual for mental health professionals in the United States is the ***Diagnostic and Statistical Manual of Mental Disorders.*** This manual lists criteria for diagnosing various mental disorders. The most recent edition, the fourth edition text revised, is also known as the *DSM-IV-TR.* According to the *DSM-IV-TR,* reactive attachment disorder is diagnosed when the following criteria are met:

- Presence of strange and developmentally inappropriate social interactions, beginning before age five years. The child does not respond to or initiate social interactions in a way that would be developmentally appropriate; instead, the child is either inhibited or is disinhibited in his or her interactions. Inhibited reactions may be excessively vigilant, restrained or ambivalent. (The child may respond to caregivers with a mixtures of approach, avoidance, and resistance to comforting, as an example from the manual.) Disinhibited reactions occur in a variety of social interactions and the child does not discriminate among people he or she chooses as attachment figures. This child will treat near strangers with inappropriate familiarity.
- The child's inappropriate social skills are not due exclusively to developmental delay (as in mental retardation) and the child's symptoms do not meet criteria for a **pervasive developmental disorder**.
- The child has received care in which his or her basic needs—either emotional or physical— are often unmet, or in which stable attachments have not been able to form (such as when primary caregivers change often).

An infant is diagnosed as having reactive attachment disorder when he or she fails to show signs of bonding to a parent or caregiver by the age of eight months. Infants normally start to follow the parent or caregiver with their eyes and smile in response to attention by about two months. By about five months, the child should reach out to be picked up and obviously enjoy simple interactive games like "peekaboo."

KEY TERMS

Behavioral therapy—An approach to treatment that focuses on extinguishing undesirable behavior and replacing it with desired behavior.

Cognitive therapy—Psychological treatment aimed at changing a person's way of thinking in order to change his or her behavior and emotional state.

Holding therapy—A controversial treatment for autism, reactive attachment disorder, and other problems of children in which an adult holds a child despite any resistance from the child until the child submits and experiences an emotional release.

Treatments

First, the child's safety and physical health must be attended to. A child that is being abused or has been physically neglected may need to be hospitalized. This is done to separate the child from the harmful situation and

take care of any medical problems resulting from neglect or abuse.

The next step is to either make the child's home environment stable, or place the child in a more stable home. Child protective services may be brought in at this point. The home situation must be evaluated, and the parents or caregivers assessed for emotional fitness to care for the child. The parents or caregivers may be given training in proper childcare and emotional nurturing. **Family therapy** may be needed in some cases to help the parents or caregivers and other children in the family.

With a young infant, the parents or caregivers will be encouraged to have a regular schedule for the infant and to spend time each day simply holding and playing with the infant.

Treatment of children who are past infancy is difficult. It is important to find a therapist experienced in the treatment of children with reactive attachment disorder. Most therapists use a mix of techniques. The therapist may seek to help the child relive and work through **grief** and anger from a prior trauma or loss. Cognitive therapy may be used to help an older child understand and reframe negative thoughts about himself or herself, or about parents or caregivers. If the child is too young to verbalize or think rationally, techniques such as **play therapy** or art therapy may be used to help bring out and work through feelings. Behavioral therapy may be used to help guide development of wanted behaviors.

Prognosis

There has not been much research to date on the course of this problem. It appears that children who are identified and treated early have a better chance of learning how to form appropriate bonds with other people.

Children who are not treated or who are treated later in life have a greater chance of having permanent problems relating to other people.

Prevention

Prevention of reactive attachment disorder begins with good parenting. As far as possible, health care providers and families should be on the lookout for any problem that may prevent parents from giving children the structure and attention they need. If a child loses its primary caregivers, a stable environment with consistent attention from one or two caregivers should be provided as soon as possible.

Early identification of reactive attachment disorder is necessary to get help to the child and family as soon as possible. The earlier this problem is identified and treated, the more likely it is that the child will be able to develop healthy patterns of relating to others.

See also Cognitive-behavioral therapy; Creative therapies; Post-traumatic stress disorder

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ORGANIZATIONS

Association for Treatment and Training in the Attachment of Children (ATTACh). <http://www.attach.org>.

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Reading disorder

Definition

Reading disorder is a learning disorder that involves significant impairment of reading accuracy, speed, or comprehension to the extent that the impairment interferes with academic achievement or activities of daily life. People with reading disorder perform reading tasks well below the level one would expect on the basis of their general intelligence, educational opportunities, and physical health. Reading disorder is most commonly called dyslexia. Dyslexia, however, usually includes deficits in spelling and writing as well as reading.

Description

Reading disorder is a learning disorder characterized by a significant disparity between an individual's general intelligence and his or her reading skills. **Learning disorders**, formerly called academic skills disorders, are disorders that account for difficulty learning and poor academic performance when low performance cannot be attributed to **mental retardation**, low intelligence, lack of learning opportunities, or such specific physical problems as vision or hearing deficits. Common learning disabilities include reading disorder (often called dyslexia), **mathematics disorder**, **disorder of written expression**, and some language processing disorders.

Reading disorder can cause severe problems in reading, and consequently in academic work, even in people

with normal intelligence, educational opportunities, motivation to learn to read, and emotional self-control. Reading disorder is different from slowness in learning or mental retardation. In reading disorder, there is a significant gap between the expected level of performance and actual achievement. Difficulties in reading can occur on many levels, and reading disorder may have several causes that manifest in different ways. Common problems in people with reading disorder include:

- slow reading speed
- poor comprehension when reading material either aloud or silently
- omission of words while reading
- reversal of words or letters while reading
- difficulty decoding syllables or single words and associating them with specific sounds (phonics)
- limited sight word vocabulary

Causes and symptoms

Causes

Reading disorder was first recognized in the late nineteenth century, when it was called pure word blindness, then developmental alexia. Starting in the 1960s, educators commonly referred to reading disorder as dyslexia, from the Greek word *dys*, meaning poor or inadequate, and the word *lexis* meaning words or language. Despite the long history of reading disorder, its cause is not known.

Learning to read is a complex task. It requires coordination of the eye muscles to follow a line of print, spatial orientation to interpret letters and words, visual memory to retain the meaning of letters and sight words, sequencing ability, a grasp of sentence structure and grammar, and the ability to categorize and analyze. In addition, the **brain** must integrate visual cues with memory and associate them with specific sounds. The sounds must then be associated with specific meanings. For comprehension, the meanings must be retained while a sentence or passage is read. Reading disorder occurs when any of these processes are disrupted. For that reason, the roots of reading disorder have proved difficult to isolate, and may be different in different individuals.

Despite the complexity of reading disorder, researchers have found that the condition is at least partially inherited. In 1999, the Centre for Reading Research in Norway studied a large family with reading problems. By evaluating the reading and writing abilities of about 80 family members across four generations, the researchers were able to pinpoint mutations in specific genes that are associated with reading and writing deficits.

KEY TERMS

Digraph—A pair of letters that represents a single speech sound. In English, the *th* in "thumb" and the *ei* in "vein" are examples of digraphs.

Dyslexia—Another term for reading disorder.

Phonics—A method of teaching reading and spelling based on the phonetic interpretation of ordinary spelling.

It appears that reading disorder may also have causes other than genetic inheritance, as about half the people with this learning disability do not come from families with a history of the problem. Many theories suggest that functional problems in specific areas of the brain underlie reading disorder. Given the complicated demands on the human nervous system involved in reading, it is entirely possible that there are several different problems in brain function related to difficulty in learning to read. What is known is that 90% of children diagnosed with reading disorder have other language deficits. Still other research suggests a possible link with a subtle visual problem that affects the speed with which affected people can read.

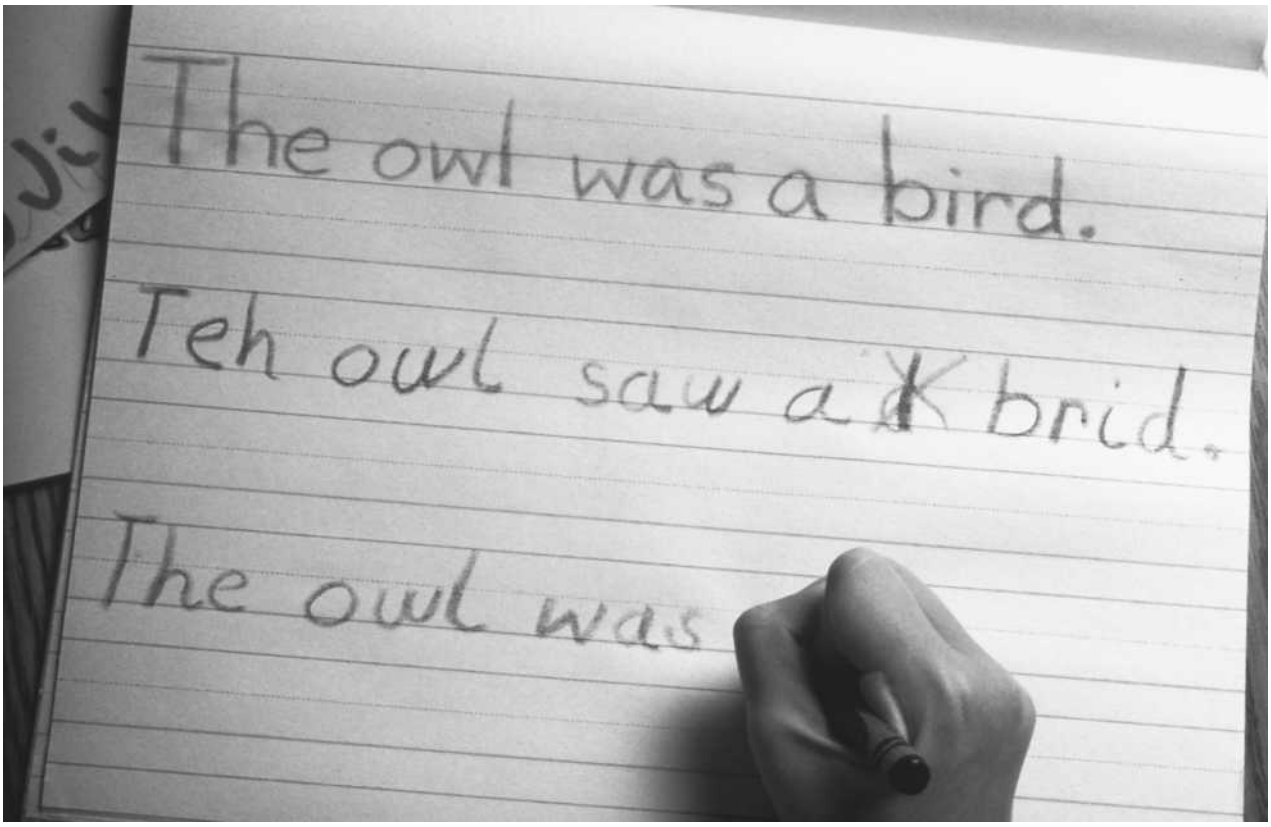
Symptoms

Common characteristics of children with reading disorder include:

- difficulty identifying single words
- problems understanding the sounds in words, sound order, or rhymes
- problems with spelling
- transposing letters in words
- omitting or substituting words
- poor reading comprehension
- slow reading speed (oral or silent)

In addition to these symptoms, children with reading disorder often have other delays or learning problems. These include:

- delays in spoken language
- confusion with directions, or right/left-handedness
- confusion with opposites (up/down, early/late)
- mathematics disorder
- disorder of written expression



Reading disorder is most commonly called dyslexia. Dyslexia, however, usually includes deficits in spelling and writing as well as reading. Symptoms of reading disorder include poor comprehension, reversal of words or letters while reading, and difficulty decoding syllables or single words and associating them with specific sounds (phonics). Here, a child with dyslexia attempts to reproduce a teacher's sentence. *(Will and Deni McIntyre/Science Source, National Audubon Society Collection/ Photo Researchers, Inc. Reproduced with permission.)*

Diagnosis

Evaluation of children's reading ability must be done on an individual basis in order to make a **diagnosis** of reading disorder and distinguish it from slow learning or low intelligence. The examiner must take into account the child's age, intelligence, educational opportunities, and such cultural factors as whether the language spoken at home is different from the language taught and used at school. Reading disorder is diagnosed when a child's reading achievement is substantially below what would be expected after taking these factors into account.

In addition, the reading problems must interfere in significant ways with the person's schoolwork or daily life. If a physical condition is present (for example, mental retardation, poor eyesight, or hearing loss), the reading deficit must be in excess of what one would normally associate with the physical handicap.

Diagnosis is complicated by the fact that 20%–55% of children with reading disorder have **attention-deficit/hyperactivity disorder** (ADHD), a behavioral disorder that aggravates learning difficulties. In addition, about one-quarter of children with reading disorder have **conduct disorder**. **Oppositional defiant disorder** and depression also occur in higher-than-average rates in children with reading disorder. Almost all people with reading disorder have difficulties spelling, and about 80% of them have other language problems.

Anyone who is suspected of having reading disorder or any other learning disability should have a comprehensive evaluation, including hearing, vision, and intelligence testing. The test should include all areas of learning and learning processes, not just reading. In school-age children, this evaluation often involves a team of educators, educational psychologists, and child psychiatrists.

Demographics

Estimates by the National Institutes of Health of the number of people with learning disorders range from 5%–15% of the general population. About 80% of people

with a learning disorder have reading disorder. Other studies suggest that about 4% of school-age children have reading disorder. People with reading disorder are more likely to have a parent or sibling with the disorder.

Between 60% and 80% of children diagnosed with reading disorder are boys. For various reasons often related to behavior, boys tend to be referred more frequently to special education classes, which suggests that girls with reading disorder may be underdiagnosed. Some experts think that this disparity comes about because boys are more often disruptive in class.

Treatments

Reading disorder, like other learning disorders, falls under the federal Individuals with Disabilities Education Act (IDEA). Definitions of learning disabilities vary among the states, and some school districts are more willing than others to recognize specific learning disabilities. Any child, however, who has a diagnosed learning disability, including reading disorder or dyslexia, should be eligible for an Individual Education Program (IEP) that provides customized instruction at school designed to address the disability.

Treatment approaches vary from visual stimulation to special **diets** to enhanced reading instruction. However, it is generally agreed that customized education is the only successful remedy. The American Academy of Ophthalmology, the American Academy of Pediatrics, and the American Association for Pediatric Ophthalmology and Strabismus have issued a policy statement warning against visual treatments and recommending a cross-disciplinary educational approach.

The first researcher to identify and study dyslexia, Samuel Torrey Orton, developed the core principles of such an approach in the 1920s. The work of three of his followers—teachers Bessie Stillman, Anna Gillingham, and Beth Slingerland—underlies many of the programs in use today, including Project READ, the Wilson Reading System, and programs based on the Herman method. There are many successful programs to address individual reading needs. In general, all good programs are:

- Sound/symbol (phonics)-based. They break words down into their smallest visual components: letters and the sounds associated with them.
- Multisensory. Good programs attempt to form and strengthen mental associations among visual, auditory, and kinesthetic channels of stimulation. The student simultaneously sees, feels, and says the sound-symbol association. For example, a student may trace the letter or letter combination with his or her finger while pronouncing a word out loud.
- Highly structured. Remediation begins at the level of the single letter-sound; works up to digraphs (a pair of letters representing a single speech sound); then syllables; then into words and sentences in a systematic fashion. Repetitive drill and practice serve to form necessary associations between sounds and written symbols.

Prognosis

Many famous and successful people have suffered from reading disorders, including at least two Presidents of the United States. How well a person compensates for this disorder depends on the severity of the impairment and the type of educational remediation that he or she receives. Generally, people who are identified as having a reading disorder before grade three and who receive intensive reading education can do well. There is, however, a great deal of variation among people in intelligence, educational opportunities, and the will to overcome a reading disorder, as well as in the type and severity of the problem. All these factors combine to determine the ultimate outcome of this disorder. The prognosis is usually good if the condition is diagnosed early and the person is enrolled in a good remedial program. Strong self-esteem, together with supportive family, friends, and teachers also improve a person's chances of overcoming this disorder.

Prevention

There is no known way to prevent reading disorder. Early **intervention** is the key to preventing the associated symptoms of low self-esteem, lack of interest in school, and poor behavior that often accompany low academic achievement.

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National Center for Learning Disabilities. 381 Park Avenue South, Suite 1401, New York, NY 10016. (212) 545-7510. <http://www.ncld.org>.

OTHER

Dyslexia Resources on the Web. <http://home.clara.net/ghrow/subjects/dyslexia.html>. Extensive links to dyslexia resources; updated frequently.

Tish Davidson, A.M.

Reinforcement

Definition

A reinforcer is a stimulus that follows some behavior and increases the probability that the behavior will occur. For example, when a dog's owner is trying to teach the dog to sit on command, the owner may give the dog a treat every time the dog sits when commanded to do so. The treat reinforces the desired behavior.

Description

In operant conditioning (as developed by B. F. Skinner), positive reinforcers are rewards that strengthen a conditioned response after it has occurred, such as feeding a hungry pigeon after it has pecked a key. Negative reinforcers are stimuli that are removed when the desired response has been obtained. For example, when a rat is receiving an electric shock and presses a bar that stops the shock, the shock is a negative reinforcer— it is an aversive stimulus that reinforces the bar-pressing behavior. The application of negative reinforcement may be divided into two types: escape and avoidance conditioning. In escape conditioning, the subject learns to escape an unpleasant or aversive stimulus (a dog jumps over a barrier to escape electric shock). In avoidance conditioning, the subject is presented with a warning stimulus, such as a buzzer, just before the aversive stimulus occurs and learns to act on it in order to avoid the stimulus altogether.

Punishment can be used to decrease unwanted behaviors. Punishment is the application of an aversive stimulus in reaction to a particular behavior. For children, a punishment could be the removal of television privileges when they disobey their parents or teacher. The removal of the privileges follows the undesired behavior and decreases its likelihood of occurring again.

Reinforcement may be administered according to various schedules. A particular behavior may be reinforced every time it occurs, which is referred to as continuous reinforcement. In many cases, however, behaviors are reinforced only some of the time, which is termed partial or intermittent reinforcement. Reinforcement may also be based on the number of responses or scheduled at particular time intervals. In addition, it may be delivered in regularly or irregularly. These variables combine to produce four basic types of partial reinforcement. In fixed-ratio (FR) schedules, reinforcement is provided following a set number of responses (a factory worker is paid for every garment he assembles). With variable-ratio (VR) schedules, reinforcement is provided after a variable number of responses (a slot machine pays off after varying numbers of attempts). Fixed-interval (FI) schedules provide for reinforcement of the first response made within a given interval since the previous one (contest entrants are not eligible for a prize if they have won one within the past 30 days). Finally, with variable-interval (VI) schedules, first responses are rewarded at varying intervals from the previous one.

See also Behavior modification

Relapse and relapse prevention

Definition

In the course of illness, relapse is a return of symptoms after a period of time when no symptoms are present. Any strategies or treatments applied in advance to prevent future symptoms are known as relapse prevention.

Purpose

When people seek help for mental disorders, they receive treatment that, hopefully, reduces or eliminates symptoms. However, once they leave treatment, they may gradually revert to old habits and ways of living. This results in a return of symptoms known as relapse. Relapse prevention aims to teach people strategies that will maintain the wellness skills they learned while in treatment.

Prevention of relapse in mental disorders is crucial—not only because symptoms are detrimental to quality of life but also because the occurrence of relapse increases chances for future relapses. In addition, with each relapse, symptoms tend to be more severe and have more serious consequences.

Description

Relapse is a concern with any disorder, whether physical or psychological. Cancer is a prime example of a physical condition where relapse is common, either after a short period or many years of remission (being symptom-free). Psychological disorders can follow a similar pattern, and certain psychological disorders tend to have a higher rate of relapse than others. Addictive disorders, such as alcohol and drug abuse, smoking, overeating, and **pathological gambling**, are well known for high levels of relapse. Many addictions involve a lifestyle centered around the addictive behavior. In such cases, individuals must not only discontinue the addictive habit, they must also restructure their entire lives in order for changes to last. Such vast changes are difficult at best, approaching impossible in the worst scenarios. For example, an individual with a drug **addiction** may live in a neighborhood where drugs are prevalent but may lack the resources to move. According to recent statistics, relapse rates are approximately 33% for people who gamble pathologically (within three months of treatment), 90% for people who quit smoking, and 50% for people who abuse alcohol. Within one year of treatment, people struggling with **obesity** typically regain 30% to 50% of the weight they lost.

Affective disorders, such as depression and anxiety, also have high rates of relapse. People with affective disorders are thought to engage in self-defeating, negative thought patterns that occur more or less automatically. These thought patterns affect behavior, resulting in unproductive or negative consequences. Negative consequences are regarded by such individuals as proof that their original self-defeating thoughts must be correct. The thought-behavior pattern becomes a repetitive cycle, with negative thoughts resulting in negative behavioral outcomes, and consequences of negative behavior encouraging more self-defeating thoughts. This cycle is extremely difficult to break because it becomes a habitual way of responding to the world that occurs almost without awareness. Relapse rates for depression are reportedly as high as 80%.

Relapse among people who commit sex offenses is a constant safety concern for those in the community. However, some statistics show that this population has a very low rate of relapse. A recent report by Robin J. Wilson and colleagues indicated rates as low as 3.7% to 6.3%. This same report stated that, among various criminal offenses, those who commit sex offenses relapse at lower rates than those who commit general offenses. Other professionals may not necessarily agree with this study, however. Those who commit sex offenses are considered at a higher risk for relapse if they display little insight into the impact of their crime. Those at high risk of committing a sex offense are not typically released back into the community.

For many types of disorders, initial treatment is often effective at eliminating the unwanted behavior. However, these effects are rarely maintained long-term without

KEY TERMS

Addictive disorder—A disorder involving repetitive participation in a certain activity, in spite of negative consequences and despite attempts to stop the behavior. Alcohol abuse is an example.

Affective disorder—A disorder involving extreme emotional experience that is not congruent with the environmental circumstances (for example, feeling sad when there is no easily identifiable reason, as in depression).

Cognitive restructuring—An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

Guided imagery—Techniques in which individuals actively imagine themselves in a scene (usually a different location, such as a relaxing beach, or a trigger situation where one handles the situation successfully), typically guided by another person describing the scene.

Lapse—A single, isolated occurrence of a symptom or negative behavior.

Positive affirmation statements—Statements repeated to oneself, either aloud or mentally, that reflect attitudes of self-worth.

Progressive muscle relaxation—Relaxation exercises where one slowly tenses and then relaxes each muscle group separately in a systematic order.

Refocusing techniques—Techniques that direct one's attention away from overwhelming, negative thoughts and emotions by focusing on inner peace and managing one issue at a time.

Remission—In the course of an illness or disorder, a period of time when symptoms are absent.

Trigger—Any situation (people, places, times, events, etc.) that causes one to experience a negative emotional reaction, which is often accompanied by a display of symptoms or problematic behavior.

some type of preventive planning. Results of medications are similar; symptoms are alleviated, but once the medication is discontinued, symptoms return unless the individual has had some type of training in coping with his or her disorder and that training has been effective. There are various forms of relapse prevention training. Most follow a similar pattern with and employ the following common elements:

- Identifying high-risk situations: Symptoms are often initiated by particular times, places, people, or events. For example, a person with **agoraphobia** is more likely to experience symptoms of panic in a crowded building. An essential key to preventing relapse is to be aware of the specific situations where one feels vulnerable. These situations are called "triggers," because they trigger the onset of symptoms. While people with the same mental disorder may share similar triggers, triggers can also be highly individual. People tend to react—sometimes unknowingly—to negative experiences in their past. For example, a woman who was sexually abused as a child may have negative emotions when in the presence of men who resemble her abuser. Because some triggers occur without conscious awareness, individuals may not know all their triggers. Many prevention programs encourage individuals to monitor their behavior closely, reflecting on situations where symptoms occurred and determining what was happening immediately before the onset of symptoms. With this kind of analysis, a pattern often emerges that gives clues about the trigger.
- Learning alternate ways to respond to high-risk situations: Once triggers have been identified, one must find new ways of coping with those situations. The easiest coping mechanism for high-risk situations is to avoid them altogether. This may include avoiding certain people who have a negative influence or avoiding locations where the symptom is likely to occur. In some instances, avoidance is a good strategy. For example, individuals who abuse alcohol may successfully reduce their risk by avoiding bars or parties. In other instances, avoidance is not possible or advisable. For example, individuals attempting to lose weight may notice that they are more likely to binge at certain times during the day. One cannot avoid a time of day. Rather, by being aware of this trigger, one can purposely engage in alternate activities during that time. Strategies for coping with unavoidable triggers are generally skills that need to be learned and practiced in order to be effective. Strategies include—but are not limited to—discussion of feelings, whether with a friend, counselor, or via a hotline; distraction, such as music, exercise, or engaging in a hobby; refocusing techniques, such as **meditation**, deep-breathing exercises, progressive muscle relaxation (focusing on each muscle group separately, and routinely tensing then relaxing that muscle), prayer, or journaling; and cognitive restructuring, such as positive affirmation statements (such as, "I am worthwhile"), active problem solving (defining the problem, generating possible solutions, identifying the consequences of those solutions, choosing the best solution), challenging the validity of negative thoughts, or guided imagery (imagining oneself in a different place or handling a situation appropriately).
- Creating a plan for healthy living: Besides being prepared for high-risk situations, relapse prevention also focuses on general principles of mental health that, if followed, greatly reduce the likelihood of symptoms. These include factors such as balanced nutrition, regular exercise, sufficient sleep, health education, reciprocally caring relationships, productive and recreational interests, and spiritual development.
- Developing a support system: Many research studies have demonstrated the importance of social support in maintaining a healthy lifestyle. Individuals who are socially isolated tend to display more symptoms of mental disorders. Conversely, individuals with mental disorders tend to have more difficultly initiating and maintaining relationships due to inappropriate social behavior. For such people, a support system may be nonexistent. Research suggests that support systems are most effective when they are naturally occurring—in other words, when a circle of family and friends who genuinely care about the individual is already in place. However, artificially created support systems are certainly better than none at all. For this reason, relapse prevention programs strive to involve family members and other significant persons in the treatment program. Everyone in the support system should be knowledgeable about the person's goals, what that person is like when he or she is doing well, and warning signs that the person may be on a path toward relapse. The support system agrees on who will take what role in encouraging, confronting, or otherwise caring for that person. **Self-help groups** such as Alcoholics Anonymous or Moderation Management are often examples of artificially created support systems.
- Preparing for possible relapse: Although the ultimate goal of relapse prevention is to avoid relapse altogether, statistics demonstrate that relapse potential is very real. Individuals need to be aware that, even when exerting their best efforts, they may occasionally experience lapses (one occurrence of a symptom or behavior) or relapses (return to a previous, undesirable level of symptoms or behavior). Acknowledging the potential for relapse is important, because many people consider a lapse or relapse as evidence of personal failure

and give up completely. In their widely acclaimed book for professionals, *Motivational Interviewing*, William R. Miller and Stephen Rollnick cite a study by Prochaska and DiClemente that found that smokers typically relapse between three and seven times before quitting for good. From the perspective of Miller and Rollnick, each relapse can be a step closer to full recovery if relapse is used as a learning experience to improve prevention strategies. Although some argue that such a tolerant attitude invites relapse, general consensus is that individuals need to forgive themselves if relapse occurs and then move on. Some prevention programs include designing a crisis plan to be put into effect if a relapse occurs. The crisis plan involves specific actions to be taken by the individual or members of the support system.

These elements are common to all relapse prevention programs, but programs can be further customized to meet the particular characteristics of a disorder. For example, prevention of depression or anxiety may focus on becoming aware of thoughts as passing mental events rather than facts about self or reality. Learning to identify bodily sensations that accompany maladaptive thoughts is also important for preventing depression and anxiety. Addictive disorders concentrate on reactions to social pressure, interpersonal conflicts, and negative emotional states as part of a relapse prevention plan.

Preparation

As with any type of therapeutic treatment, success of relapse prevention programs depend heavily on motivation. If an individual is not interested in making life changes, he or she is not likely to follow a prevention plan. Individuals low in motivation may need to participate in group or individual **psychotherapy** before deciding whether to enter a relapse prevention program.

Aftercare

Aftercare typically consists of participation in **support groups**. For addictions, 12-step groups (such as Alcoholics Anonymous) are most commonly recommended. These types of groups can be attended daily. Support groups exist for other types of mental disorders, and may be run by peers or a professional facilitator. Aftercare groups, usually run in treatment facilities by professional staff, may be used to continue practicing skills and to trouble-shoot problems individuals are experiencing with their prevention plans in everyday life. Aftercare groups usually meet less frequently (once a week or month) and may gradually taper off. Some relapse-prevention programs may use telephone contacts or individual therapy sessions to help individuals continue to use prevention skills effectively.

Normal results

Successful relapse prevention programs will empower individuals to make choices about how they respond in stressful, high-risk situations (triggers) rather than responding in habitual, unhealthy ways. Individuals should be aware of their personal triggers, use positive strategies for coping with **stress**, practice healthy lifestyle choices, involve others in their efforts, and have a realistic attitude regarding relapse. Use of these prevention skills should reduce symptoms and increase the time span between occurrences of lapses or relapses.

Abnormal results

If an individual is unmotivated to make life changes, or a relapse prevention program has been ineffective, that individual will demonstrate few (if any) of the prevention skills learned. The individual will show little improvement in symptomatic or problematic behavior. Periods of remission (symptom-free behavior) will be short and relapses will occur frequently.

See also Alcohol and related disorders; Anxiety-reduction techniques; Cognitive-behavioral therapy; Cognitive problem-solving skills training; Substance abuse and related disorders

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National Institute on Alcohol Abuse and Alcoholism.6000 Executive Boulevard, Willco Building, Bethesda, Maryland 20892-7003. <http://www.niaaa.nih.gov>.

National Institute on Drug Abuse, National Institutes of Health. 6001 Executive Boulevard, Room 5213, Bethesda, Maryland 20892-9561. (301) 443-1124. <http://www.nida.nih.gov>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8194, MSC 9663, Bethesda, Maryland 20892-9663. (301) 443-4513. <http://www.nimh.nih.gov>.

Sandra L. Friedrich, M.A.

Remeron *see* **Mirtazapine**

Respite

Definition

Respite literally means a period of rest or relief. Respite care provides a caregiver temporary relief from the responsibilities of caring for individuals with chronic physical or mental disabilities. Respite care is often referred to as a gift of time.

Purpose

Respite was developed in response to the **deinstitutionalization** movement of the 1960s and 1970s. Maintaining individuals in their natural homes rather than placing them in long-term care facilities was viewed as beneficial to the individual, the involved family, and society (in terms of lowered health care costs). The primary purpose of respite care is to relieve caregiver **stress**, thereby enabling them to continue caring for the individual with a disability.

Respite care is typically provided for individuals with disorders related to aging (**dementia**, frail health), terminal illnesses, chronic health issues, or developmental disabilities. More recently, children with behavior disorders have also been eligible for respite care. Respite care is usually recreational and does not include therapy or treatment for the individual with the disability.

Caregivers frequently experience stress in the forms of physical **fatigue**, psychological distress (resentment, frustration, anxiety, guilt, depression), and disruption in relations with other family members. The emotional aspects of caring for a family member are often more taxing than the physical demands. Increased caregiver stress may result in health problems such as ulcers, high blood pressure, difficulty sleeping, weight loss or gain, or breathing difficulties.

Types of respite

Length of respite care can be anywhere from a few hours to several weeks. Services may be used frequently or infrequently, such as for emergencies, vacations, one day per week or month, weekends, or everyday.

A variety of facilities provide respite care services. The type of service available is often closely related to the characteristics of the facility, including:

- In-home respite services consist of a worker who comes to the family home while the caregiver is away. These services are usually provided by agencies that recruit, screen, and train workers. This type of respite is usually less disruptive to the individual with the disability, provided there is a good match between the worker and the individual. However, issues of reliability and trustworthiness of the worker can be an additional source of stress for the caregiver.
- Respite centers are residential facilities specifically designed for respite care. Adult day care programs and respite camps also fall into this category. This type of respite offers more peace of mind to the caregiver, and may provide a stimulating environment for the individual with the disability. However, centers usually restrict

length of stay and may exclude individuals based on severity of disability.

- Institutional settings sometimes reserve spaces to be used for respite purposes. These include skilled nursing facilities, intermediate care facilities, **group homes**, senior housing, regular day care or after-school programs for children, and hospitals. Some of these facilities provide higher levels of care, but are less home-like. The individual with the disability may oppose staying in an institutional setting or may fear abandonment.
- Licensed foster care providers can also provide respite services in their homes.

Funding

Costs of respite care present a financial burden to many families. **Community mental health** centers often fund respite services if the individual meets certain criteria, including eligibility for Medicaid. Wraparound programs (also accessed through community mental health centers) for children with emotional or behavioral disorders also pay for respite services. Veteran's Administration hospitals provide respite care at little or no charge if the individual receiving the care is a veteran (but not if the caregiver is a veteran). Private insurance companies rarely pay for respite, and many respite providers do not accept this form of payment. Some respite facilities have sliding-scale fees. Other facilities operate as a co-op, where caregivers work at the facility in exchange for respite services.

In addition, respite agencies may have difficulty recruiting and retaining qualified employees, because limited funding prevents agencies from offering desirable salaries. The high turnover and unavailability of employees may result in delays in service delivery or family dissatisfaction with services. Advocacy for policy changes regarding funding is needed.

Barriers to using respite services

Recent research suggests that families who use respite tend to have higher levels of perceived stress, lower levels of support from others, and fewer resources. In many of these families, the individuals in need of care have more severe disabilities, problem behaviors such as aggression or self-injury, and communication difficulties; are school-aged; and are more dependent for basic needs such as eating, toileting, and dressing.

It has been well documented that many families eligible for respite care never utilize these services. Research regarding the use, availability, and effectiveness of respite care is still in the preliminary stages. Various reasons for non-utilization of respite include:

- Unfamiliarity: Some families are unaware that such services exist, or may be uncertain about how to access services. This implies a need for improved referral services.
- Funding: Limited funding may prevent some families from receiving services.
- Caregiver qualities: Some caregivers experience guilt or anxiety over allowing someone else to care for their loved one. Being able to maintain one's family independently may be tied to gender roles or cultural cus-

KEY TERMS

Behavior disorders—Disorders characterized by disruptive behaviors such as conduct disorder, oppositional defiant disorder, and attention-deficit/hyperactivity disorder.

Community mental health centers—Organizations that manage and deliver a comprehensive range of mental health services, education, and outreach to residents of a given community.

Deinstitutionalization—The process of moving people out of mental hospitals into treatment programs or halfway houses in local communities. With this movement, the responsibility for care shifted from large (often governmental) agencies to families and community organizations.

Developmental disabilities—Disabilities that are present from birth and delay or prevent normal development, such as mental retardation or autism.

Intermediate care facility—An inpatient facility that provides periodic nursing care.

Medicaid—A program jointly funded by state and federal governments that reimburses hospitals and physicians for the care of individuals who cannot pay for their own medical expenses. These individuals may be in low-income households or may have chronic disabilities.

Skilled nursing facility—An inpatient facility that provides 24-hour nursing services to individuals in need of extended care.

Veteran's Administration hospitals—Medical facilities operated by the federal government explicitly for veterans of the United States military.

Wraparound—A relatively new form of mental health service delivery that strives to accommodate all family members based on self-defined needs, flexibly incorporating both formal and informal community services

An adolescent swings on the playground at a respite care facility that provides short-term care for families who have children with developmental disabilities. *(AP Photo/ Fort Collins Coloradoan, Sherry Barber. Photo reproduced by permission.)*

toms. Relatives and friends may assist in caregiving, making formal respite unnecessary.

- Care recipient qualities: Occasionally the individual with the disability is opposed to respite care. He or she may not trust strangers or may refuse to leave home. In other instances, the individual may have behaviors, or require physical care, that is too challenging for the respite provider.
- Program qualities: Many researchers believe that respite programs are not adequately meeting the needs of families. In some cases, times that services are offered are inconvenient. Individuals with severe disabilities who pose the most need for services are sometimes excluded.

Many caregivers obtain respite in informal ways not offered by respite services. Some researchers have suggested that respite care should be just one form of service available to caregivers. Other services that may alleviate caregiver stress could include home-delivered meals, transportation assistance, recreational resources, or care skills training.

See also Case management

Resources

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The Arc National Headquarters, P.O. Box 1047, Arlington, Texas 76004. (817) 261-6003; (817) 277-0553 TDD. thearc@metronet.com.<http://www.thearc.org>.

ARCH National Respite Network and Resource Center. Chapel Hill Training-Outreach Project, 800 Eastowne Drive, Suite 105, Chapel Hill, North Carolina 27514. (888) 671-2594; (919) 490-5577. <http://www.chtop.com>.

National Aging Information Center. Administration on Aging, 330 Independence Avenue, SW, Room 4656, Washington, DC 20201. (202) 619-7501. <http://www.aoa.gov/naic>.

National Information Center for Children and Youth with Disabilities. P.O. Box 1492, Washington, DC 20013. (800)-695-0285. <http://www.nichcy.org>.

OTHER

Senior Care Web. <http://www.seniorcareweb.com>.

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Response prevention *see* **Exposure treatment**

Restoril *see* **Temazepam**

Rett's disorder

Definition

Rett's disorder, which is also known as Rett's syndrome or RS, belongs to a group of childhood disorders known as **pervasive developmental disorders** (PDDs) or autistic spectrum disorders. It is classified by the mental health professional's handbook (the ***Diagnostic and Statistical Manual of Mental Disorders*** or the *DSM-IV-TR*) as a developmental disorder of childhood. Rett's disorder is characterized by an early-onset slowing of the infant's head growth and a reduction in **brain** size, as much as 30%.

Description

RS was first described by an Austrian physician, Andreas Rett, in 1966; prior to 1983, however, little was known about the syndrome because its occurrence is quite rare. Although RS was thought at first to result from the destruction or degeneration of brain tissue, genetic research has indicated that it is caused by the failure of the infant's brain to develop normally. This developmental failure is in turn caused by a genetic mutation affecting production of a key protein that regulates brain development.

Rett's disorder has a distinctive onset and course. The child— almost always a girl— develops normally during the first five months of life. After the fifth month, head growth slows down and the child loses whatever purposeful hand movements she had developed during her first five months. After 30 months, the child frequently develops repetitive hand-washing or hand-wringing gestures; 50%–80% of children with the disorder will eventually develop epilepsy. Rett's disorder is also associated with severe or profound **mental retardation**.

Causes and symptoms

Causes

The cause of Rett's disorder is a genetic mutation on the long arm of the X chromosome (Xq28) at a locus known as MECP2. The gene was discovered in 1999, and it produces a protein known as MeCP2, which is essential to life and crucial to the normal development of the human brain. The mutation that causes Rett's disorder allows other genes to become or remain active at inappropriate points in the brain's development. These activated genes interfere with the normal pattern of development and maturation of the brain's functions. Although Rett's disorder was previously thought to result from degeneration or deterioration of brain tissue, the discovery of the Rett's gene provides evidence that the disorder may be due to a failure of normal brain development. The sensory, motor, and emotional functions of the brain are not integrated in Rett's patients as they are in persons without the mutation. Certain regions of the brain in Rett's patients essentially remain at an infantile stage of development.

RS is classified by geneticists as an X-linked dominant disorder with a high rate of new mutations. Most of these mutations (99.5%) occur while the fetus is developing in the mother's womb; only 0.5% of cases of Rett's disorder are recurrences within families. One of the most important aspects of the discovery of the Rett gene is that RS is the first disorder in humans to be traced to defects in a protein (MeCP2) that controls the expression of other genes through its interaction with methylated DNA. The discovery uncovered a new class of genetic disease that might extend far beyond RS in its applications to other disorders related to developmental failures of the nervous system.

Symptoms

The symptoms of Rett's disorder have been described in terms of four stages in the child's development.

KEY TERMS

Hyperventilation—A pattern of rapid, shallow breathing that is frequently found in patients with Rett's disorder.

Mosaicism—A genetic condition in which some cells in an organism have one set of chromosomes and other cells have a different set.

Mutation—A spontaneous change in the sequence of nucleotides in a chromosome or gene. Mutations may affect the number and structure of chromosomes or cause deletions of part of a chromosome. Rett's disorder is caused by a mutation on the long arm of the X chromosome.

Pervasive developmental disorders (PDDs)—A category of childhood disorders that includes Asperger's syndrome and Rett's disorder. The PDDs are sometimes referred to collectively as autistic spectrum disorders.

Scoliosis—An abnormal lateral (sidewise) curvature of the spine. Many patients with RS develop scoliosis after puberty.

STAGE ONE, EARLY-ONSET (SIX–18 MONTHS OF AGE). The early symptoms of RS are not always noticeable in Stage 1. The infant may not make eye contact with family members and may not show much interest in toys. She may be considered a "good baby" because she is so calm and quiet. On the other hand, there may be noticeable hand-wringing and slowing of head growth.

STAGE TWO, RAPID DETERIORATION (ONE–FOUR YEARS). This stage may be either rapid or gradual in onset. The child loses her ability to speak and to make purposeful hand movements. Hand-to-mouth movements may appear, as well as hand-wringing or hand-clapping gestures. These movements may be nearly constant while the child is awake but disappear during sleep. There may be noticeable episodes of breath holding and hyperventilating (rapid shallow breathing). The child may have trouble sleeping, and may become irritable. If she is able to walk, she will start to look unsteady on her feet and may have periods of trembling or shaking. Slowed growth of the head is usually most noticeable during this stage.

STAGE THREE, PLATEAU (TWO–10 YEARS). Motor problems and **seizures** often appear during this stage. The child's behavior, however, often shows some improvement, with less irritability and crying. She may show greater interest in her surroundings, and her attention span and communication skills often improve. Many patients with RS remain in stage 3 for most of their lives.

STAGE FOUR, LATE DETERIORATION OF MOTOR SKILLS (USUALLY AFTER 10 YEARS OF AGE). In stage 4, patients with RS gradually lose their mobility; some stop walking while others have never learned to walk. There is, however, no loss of cognitive or communication skills, and the repetitive hand movements may decrease. The spine begins to develop an abnormal sideways curvature (scoliosis), and the patient may develop muscle rigidity. Puberty begins at the same age as in most girls.

Demographics

RS is less common than the other PDDs. Recent estimates of its prevalence range between 1:10,000 births and 1:15,000 births. As of 2002, little is known about its prevalence across different racial and ethnic groups.

Until 2000, Rett's disorder was thought to occur only in girls, but at least two cases have been reported in boys as well. Since RS is caused by a mutation on the X chromosome that affects the production of a protein essential to life, and the Y chromosome that determines male sex cannot compensate for a damaged X chromosome, a male fetus with a defective X chromosome does not usually survive. The two known cases of RS in boys involve one child who has two X chromosomes as well as a Y, and a child whose X chromosome is faulty in some of the cells in his body but not all. This condition is known as mosaicism.

Diagnosis

The **diagnosis** of Rett's disorder is made on the basis of observation of the child—usually over a period of several hours or days—and interviews with the parents. There are no laboratory or diagnostic imaging tests for RS. The diagnosis can be made by a pediatrician or primary care physician, but should be confirmed by a pediatric neurologist (specialist in disorders of the nervous system in children) or developmental pediatrician. After the examiner has excluded the possibility of other developmental disorders, there are six criteria that must be met for a diagnosis of Rett's disorder, and a secondary group of supportive criteria that are frequently observed in RS patients but are not necessary to make the diagnosis.

Diagnostic criteria

The diagnostic criteria for RS include the following:

- a period of apparently normal development before six–18 months of age
- a normal-sized head at birth followed by slowing of head growth between five months and four years
- severe impairment in the use of language and loss of purposeful hand motion
- repetitive hand movements that include one or more of the following: hand washing, hand wringing, or hand clapping
- shaking of the chest or torso, particularly when the child is agitated or upset
- in children able to walk, an unsteady, stiff-legged, wide-based gait

Supportive criteria

Supportive criteria are criteria that are not essential to the diagnosis of a particular disorder (because some people with the disorder do not have them). Supportive criteria are nonetheless strong evidence that a person who exhibits these criteria does in fact have the disorder. Supportive criteria for Rett's disorder include:

- dysfunctional breathing, which may include hyperventilation, breath holding, and air swallowing
- abnormal electroencephalogram (EEG) patterns
- seizures
- difficulties in chewing and swallowing
- constipation

- muscle rigidity and contracting of the joints that increase with age
- scoliosis (curvature of the spine from side to side)
- teeth grinding
- small feet in relation to overall height
- slow overall growth
- loss of body fat and muscle mass
- abnormal sleeping patterns combined with irritability or agitation
- poor circulation in the feet and legs

These supportive criteria do not always appear in young children with RS but are often observed as the child grows older.

Treatments

There is no single treatment regimen that is applicable to all patients with Rett's disorder. Some patients benefit from medications for muscular rigidity or for specific mood or behavioral problems, such as anxiety or irritability. A child **psychiatrist** should be consulted in regard to medications.

The degree of mental retardation associated with RS means that patients with this disorder will not benefit from **psychotherapy**. Parents of children with RS, however, are often helped by supportive therapy groups for parents of children with PDDs. Another type of program that is helpful for parents is learning skills for coping with the behaviors of RS children. These programs are usually led by a behavioral **psychologist**.

The U. S. National Institute of Mental Health (NIMH) is presently conducting research studies of psychosocial approaches to treatment of Rett's and other PDDs as well as studies of medications given for these disorders. Readers who would like more information about this research may contact NIMH Public Inquiries at 6001 Executive Boulevard, Rm. 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513; Fax (301) 443-4279; TTY (301) 443-8431.

Prognosis

It is important to note that current information about the prognoses of children with Rett's syndrome is derived from treatments given to patients in the 1970s or 1980s. As knowledge of effective treatments continues to accumulate, children with RS are receiving treatment earlier than they did two decades ago. It is likely that future prognoses for the disorder will reflect these improvements.

As of 2002, the prognosis for RS patients is poor. In most cases, there is a steady loss of cognition, movement-related, social, and behavioral skills throughout the patient's lifetime. Some patients, however, make modest developmental gains in adolescence. The average life expectancy of patients with RS has not yet been determined, although some are presently middle-aged.

Prevention

As of 2002, there are no effective strategies for preventing Rett's disorder, since most cases result from new mutations of the MECP2 gene rather than transmission of a defective gene from the parents.

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Institute for Community Inclusion/UAP. 300 Longwood Avenue, Boston, MA 02115. (617) 355-6506. TTY (617) 355-6956. E-mail: ici@a1.tch.harvard.edu.

International Rett Syndrome Association (IRSA). 9121 Piscataway Road, Suite 2-B, Clinton, MD 20735. (301) 856-3334 or (800) 818-RETT. Fax: (301) 856-3336. <www.rettsyndrome.org>.

National Association of Rare Disorders (NORD). P.O. Box 8923, New Fairfield, CT 06812-8923. (800) 999-NORD or (203) 746-6518.

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Revia *see* **Naltrexone**

Risperdal *see* **Risperidone**

Risperidone

Definition

Risperidone is classified as an atypical antipsychotic drug. It is sold in the United States under the brand name of Risperdal.

Purpose

Risperidone is used for the management of symptoms of psychotic disorders such as **schizophrenia**.

Description

Risperidone is an atypical antipsychotic agent for two reasons. First, it is chemically unrelated to the older antipsychotic drugs. Second, unlike older antipsychotic drugs that primarily inhibit the actions of dopamine, a chemical in the **brain**, risperidone may also have some action against another brain chemical, serotonin. The proper level of both dopamine and serotonin are influential in maintaining mental well-being.

An advantage of using risperidone over one of the older antipsychotic drugs is a lower incidence of parkinsonian-like side effects. These side effects may be sufficiently troublesome to cause patients to discontinue treatment for their schizophrenia. For this reason, patients who have had negative experiences with older antipsychotics may benefit from risperidone. Also, some patients who showed little improvement with older antipsychotic drugs respond better to risperidone.

Risperidone is available in 0.25-mg, 0.5-mg, 1-mg, 2-mg, 3-mg, and 4-mg tablets and a solution containing 1 mg of drug in each milliliter of solution.

KEY TERMS

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Parkinsonian—Related to symptoms associated with Parkinson's disease, a nervous system disorder characterized by abnormal muscle movement of the tongue, face, and neck, inability to walk or move quickly, walking in a shuffling manner, restlessness, and/or tremors.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Recommended dosage

For treating psychotic disorders in adults, the usual starting dose of risperidone is 1 mg twice daily. Dosage is increased gradually until a target dose of 3 mg twice daily is reached. Some patients do just as well with a single daily dose (6 mg once a day, for example). There is little clinical evidence to indicate that increasing the daily dose beyond 8 mg offers additional benefit. However, higher doses may contribute to additional side effects. If the dose needs to be adjusted, the changes should made no more often than once per week.

In older patients (over age 60), starting dosage should not exceed 1 mg daily. Most patients should not take more than 3 mg daily. People with low blood pressure and those who have kidney disease should take a similarly reduced dose.

Precautions

Patients with a history of cardiovascular disease or low blood pressure should take risperidone only after discussing the risks and benefits with their physician, and then with close physician monitoring.

Risperidone has occasionally been associated with **seizures**. People with a past history of seizures should discuss with their doctor whether risperidone is the right antipsychotic for them to use.

People taking risperidone should avoid operating a motor vehicle or other dangerous machinery until they see how risperidone affects them.

Some people have trouble regulating their body temperature while taking risperidone. Patients receiving this drug should be aware of this and avoid extremes in outdoor temperatures.

Side effects

The most common and bothersome side effect associated with risperidone is decreased blood pressure while standing up (known as orthostatic hypotension). This can cause dizziness or fainting. A decrease in blood pressure usually occurs early in therapy, while the proper dose is being established. It is more common in older patients than in younger ones. Usually, this side effect disappears entirely with time. If it continues, the physician may decrease the dose. Meanwhile, people taking risperidone should be aware of this side effect and get up slowly if they have been sitting for an extended time.

The most common nervous system side effects of risperidone include **insomnia**, agitation, anxiety, and headache. Early in therapy, patients may experience an inability to think clearly or perform certain tasks that require mental alertness. High doses of risperidone can cause unwanted sleepiness in about 40% of patients.

Antipsychotic drugs, including risperidone, can cause side effects that are similar to the symptoms of Parkinson's disease. The patient does not have Parkinson's disease, but may have shaking in muscles at rest, difficulty with voluntary movements, and poor muscle tone. These symptoms normally disappear if the drug is stopped.

The most common gastrointestinal side effects include nausea, vomiting, constipation, and difficulty digesting food.

Up to 10% of patients taking risperidone experience rhinitis (runny nose).

Interactions

There is very little information about how risperidone interacts with other drugs. However, because some patients receiving risperidone experience lowered blood pressure while standing, it is expected that other drugs that lower blood pressure may increase the incidence and severity of this side effect when taken with risperidone.

Resources

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Ritalin *see* **Methylphenidate**

Rivastigmine

Definition

Rivastigmine is a drug used to treat symptoms of **Alzheimer's disease**. In the United States, rivastigmine is sold as the brand name drug Exelon.

Purpose

Rivastigmine is used to treat symptoms of Alzeheimer's disease in individuals with mild to moderate illness. It has also been used to treat **dementia** caused by other conditions such as Lewy-body disease or following strokes. The drug may produce mild improvements in symptoms of thinking for a short period of time, but rivastigmine does not cure or stop progression of underlying diseases.

Description

The Food and Drug Administration approved rivastigmine in 2000 specifically for treating Alzheimer's disease. In Alzheimer's disease, some cells in specific regions of the **brain** die. Because of this cell death, these brain cells lose their ability to transmit nerve impulses. Brain cells normally transmit nerve impulses another by secreting various chemicals known as **neurotransmitters**.

Brain cells that make and secrete a neurotransmitter called acetylcholine are affected early in the course of Alzheimer's disease. Rivastigmine prevents the breakdown of acetylcholine in the brain, thus temporarily increasing its concentration. In doing so, rivastigmine may improve the thinking process by facilitating nerve impulse transmission within the brain.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Lewy-body disease—A type of dementia that resembles Alzheimer's disease, but progresses more rapidly. Common symptoms include fluctuations in confusion and recurring visual hallucinations. In this disease, abnormal brain cells are distributed throughout the brain.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Placebo—An inactive substance or preparation used as a control in experiments with human subjects to test the effectiveness of a drug or herbal preparation. Some patients may experience a medicinal response or experience side effects to a placebo simply because they have faith in its powers even though it contains no medicine.

Rivastigmine is available as capsules in four different strengths and as an oral solution for use by people who have difficulty swallowing. Unlike some other drugs used to treat Alzheimer's disease, the liver does not break down rivastigmine. As a result, it may be preferred in the treatment of people with Alzeheimer's disease who have liver disease.

Recommended dosage

The initial dosage of rivastigmine is 1.5 mg taken two times per day. If this dose is tolerated without difficulty, the dosage may be increased to 3 mg twice a day after at least two weeks at the lower dosage. Some people are unable to tolerate nausea, vomiting, anorexia, and weight loss that occur with higher dosages. If the drug does not cause significant adverse effects, the dose may be increased to 4.5 mg two times per day, followed by 6 mg two times per day. The dosage should be increased slowly, at two-week intervals. If side effects occur and cannot be tolerated, the drug may be stopped for several doses. When the drug is started again, the same dosage or the next lower dosage may be tried. The maximum daily dosage is 6 mg two times per day.

Precautions

Rivastigmine may slow heart rates, increase acid in the stomach, make urination difficult, cause breathing difficulties, and may possibly contribute to **seizures**. As a result, it should be used with close physician supervision and monitoring in people with certain heart conditions, those who are prone to stomach ulcers, people with bladder obstruction, individuals with asthma or chronic obstructive pulmonary disease, and people with a history of seizures disorders.

Individuals taking rivastigmine should be reassessed periodically to determine whether the drug is providing any benefits. If caregivers feel the drug is no longer beneficial, it may be stopped.

Side effects

The most frequent side effects associated with rivastigmine involve stomach upset. Nausea, vomiting, anorexia, heartburn, and weakness occur in more than 5% of people and at twice the rate of placebo pills. Dizziness and headaches also occur in more than 10% of people taking rivastigmine.

Other, less common, side effects are difficulty sleeping, confusion, depression, anxiety, sleepiness, **hallucinations**, tremors, fainting, aggression, constipation, gas, overwhelming **fatigue**, weight loss, increased sweating, and infections.

Interactions

Drugs such as dicyclomine may inhibit the effects of rivastigmine. Other drugs like bethanechol may possibly increase some of the side effects of rivastigmine. Rivastigmine may interact with some of the drugs used to relax muscles during surgery. The interaction increases the effects of both drugs.

Resources

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KEY TERMS

Projective test—A psychological test in which the test taker responds to or provides ambiguous, abstract, or unstructured stimuli, often in the form of pictures or drawings.

Reliability—The ability of a test to yield consistent, repeatable results.

Standardization—The administration of a test to a sample group of people for the purpose of establishing test norms.

Validity—The ability of a test to measure accurately what it claims to measure.

Rorschach technique

Definition

The Rorschach technique, sometimes known as the Rorschach test or the inkblot test, is a projective personality assessment based on the test taker's reactions to a series of 10 inkblot pictures.

The Rorschach technique is the most widely used projective psychological test. The Rorschach is used to help assess personality structure and identify emotional problems and mental disorders. Like other projective techniques, it is based on the principle that subjects viewing neutral, ambiguous stimuli will project their own personalities onto them, thereby revealing a variety of unconscious conflicts and motivations. Administered to both adolescents and adults, the Rorschach can also be used with children as young as three years old, although the commonly used Exner scoring system (discussed below) is appropriate only for test taker five years or older.

Purpose

The Rorschach technique is used to elicit information about the structure and dynamics of an individual's personality functioning. The test provides information about a person's thought processes, perceptions, motivations, and attitude toward his or her environment, and it can detect internal and external pressures and conflicts as well as illogical or psychotic thought patterns.

The Rorschach technique can also be used for specific diagnostic purposes. Some scoring methods for the Rorschach elicit information on symptoms related to depression, **schizophrenia**, and anxiety disorders. Also, the test can be used to screen for coping deficits related to developmental problems in children and adolescents.

Precautions

The Rorschach is generally used as part of a battery of tests and must be administered by a trained **psychologist**. Also, scoring the Rorschach test requires training in and knowledge of a comprehensive scoring system.

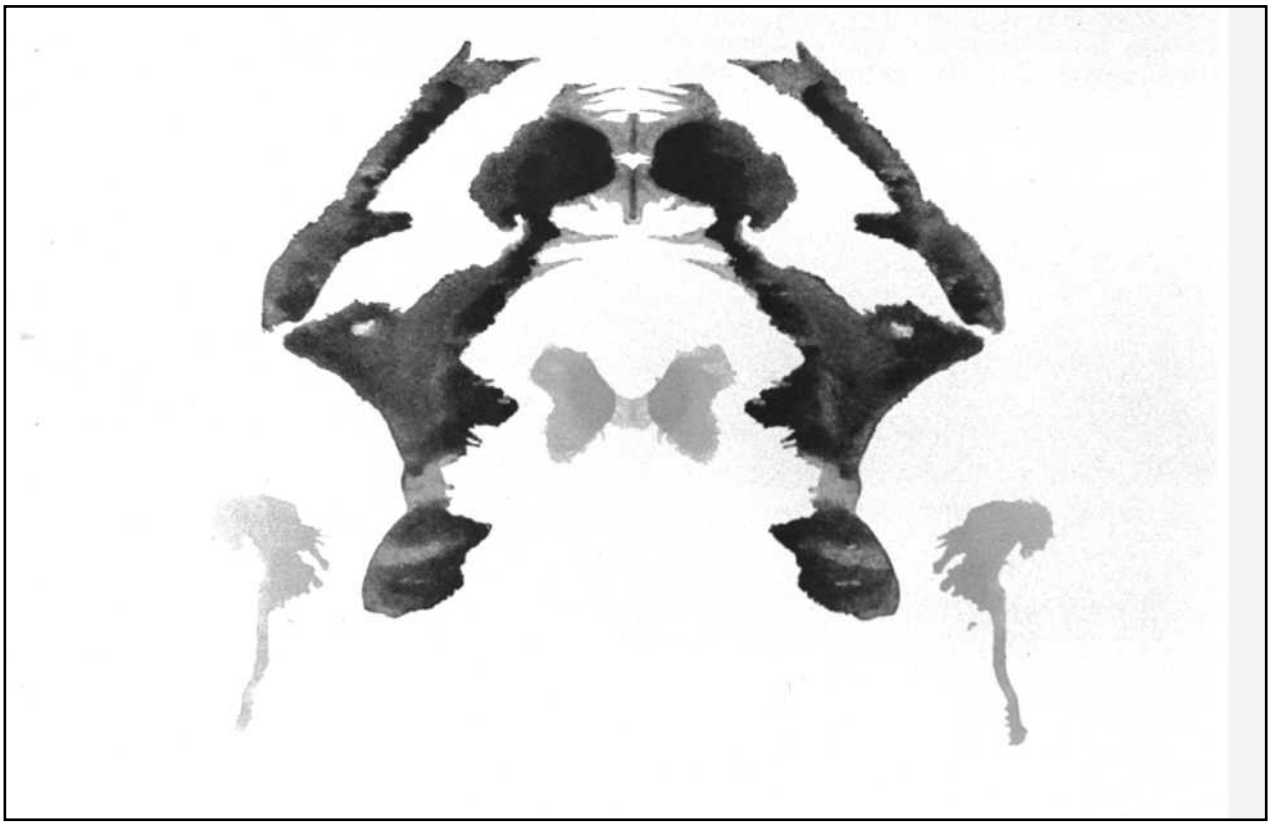
There is some disagreement concerning the reliability, validity, and clinical utility of the test and its scoring systems. Diagnoses for clinical disorders should not generally be based solely on the Rorschach test.

Description

The Rorschach technique is named for its developer, Swiss **psychiatrist** Hermann Rorschach (1884-1922). Rorschach, whose primary interest was in the psychoanalytic work of Carl Jung, began experimenting with inkblots as early as 1911 as a means of assessing introversion and extroversion.

The Rorschach technique is administered using 10 cards, each containing a complicated inkblot pattern, five in black and gray, two in black and red, and three in various pastel colors. Subjects look at the cards one at a time and describe what each inkblot resembles. They are instructed to look at the shape, shading, and color of the inkblots. After the subject has viewed all 10 cards, the examiner usually goes back over the responses for additional information. The subject may be asked to clarify some responses or to describe which features of each inkblot prompted the responses. Actually, there is no one correct response to any inkblot card, although there are certain common responses to some cards.

The test taker is given a lot of flexibility with how to respond to the inkblots. If a test taker asks if he or she is allowed to turn the card upside-down, the test adminis-



Example of a Rorschach inkblot test. *(Stan Goldblatt. Photo Researchers, Inc. Reproduced by permission.)*

trator will be non-directive, indicating it is the test taker's choice. A response like this from the test administrator is consistent with the projective nature of the Rorschach technique in that the test taker is projecting his or her personality onto the test stimuli.

Results

Rorschach, who pioneered the test in 1921, did not provide a comprehensive scoring system. In response to complaints about validity, scoring methods have been devised that aim at providing greater objectivity by clearly specifying certain personality variables and relating them to clinical diagnoses. Originally published in the 1960s, the Exner Comprehensive Rorschach System used today (the 1987 updated version) is a computer-based scoring system that provides score summaries and lists likely personality and adjustment descriptions for each test taker. Specifically, this scoring system considers aspects of a test taker's response such as the content of the response, the reasons for the events present on the card, the location of events on the card, and elaboration on cooperative and aggressive behavior. Exner also recorded certain popular and common responses to the cards and the degree to which test takers chose these responses. It should be noted, however, that many examiners still interpret the scores without benefit of a computer.

Test scores, whether based on Rorschach's original formulation, Exner's comprehensive scoring system, or other scoring systems, are based on several factors. One is location, or what part of the blot a person focuses on: the whole blot, sections of it, or only specific details within a particular section. Another is whether the response is based on factors such as form, color, movement, or shading. These factors are referred to as determinants. For example, people who tend to see movement in Rorschach blots are thought to be intellectual and introspective; those who see mostly stationary objects or patterns are described as practical and action-oriented. Finally, content refers to which objects, persons, or situations the person sees in the blot. Content categories include humans, animals, clothing, and nature.

Most examiners also assess responses based on the frequency of certain responses as given by previous test takers. Many psychologists interpret the test freely according to their subjective impressions, including their impression of the subject's demeanor while taking the test (cooperative, anxious, defensive, etc.). Such interpretations, especially when combined with clinical obser-

vation and knowledge of a client's personal history, can help a therapist arrive at a more expansive, in-depth understanding of the client's personality.

While the Rorschach technique is still widely used, its popularity has decreased somewhat in recent decades. Unlike objective personality inventories, which can be administered to a group, the Rorschach test must be given individually. A skilled examiner is required, and the test can take several hours to complete and interpret. Like other projective tests, it has been criticized for lack of validity and reliability. Interpretation of responses is highly dependent on an examiner's individual judgment: two different testers may interpret the same responses quite differently. In addition, treatment procedures at mental health facilities often require more specific, objective types of personality description than those provided by the Rorschach technique.

There have, however, been studies that support the validity of the Rorschach test. When trained psychologists use a comprehensive scoring system, agreement between administrators on certain variables ranges between 80% and 100%. Also, Exner's comprehensive system is based on a standardization sample of more than 2,000 children, adolescents, and adults. This sample included a large number of schizophrenic and depressed individuals.

See also Figure drawings; House-Tree-Person

Resources

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Ali Fahmy, Ph.D.

KEY TERMS

Antioxidant—Substance that protects the body from damaging reactive oxygen molecules in the body. These reactive oxygen molecules can come from inside the body or from environmental pollution and are thought to play a role in the aging process and the development of degenerative disease. The phenolic compounds in essential oil of rosemary have been shown to be effective antioxidants.

Astringent—A substance or compound that causes contraction or constriction of soft tissue. Rosemary's astringent qualities have made it a popular ingredient in treatments for oily skin.

Essential oil—The product of special ducts or cells in the tissues of aromatic plants (or the sap of certain trees) that gives the plant its characteristic aroma and therapeutic properties. Essential oils are sometimes called volatile oils because they evaporate readily at room temperature.

Flavonoids—Plant pigments that have a variety of effects on human physiology. Some of these pigments have anti-inflammatory, anti-carcinogenic, and antioxidant effects, for example. The diosmin contained in rosemary is a flavonoid.

Middle note—A term used in perfumery and aromatherapy to designate essential oils whose odors emerge later than "top notes" but evaporate more rapidly than "bottom notes." Rosemary is considered a middle note in aromatherapy.

Phenol—A white crystalline water-soluble substance used chiefly as an antiseptic and disinfectant.

Topical—A type of medication or preparation intended for use on the skin or external suface of the body. Rosemary is a common ingredient in astringent cleansers and in hand lotions or similar preparations intended to warm the skin or increase blood circulation.

Rosemary

Definition

Rosemary is an herb derived from an evergreen shrub, *Rosmarinus officinalis*, related to the mint or Lamiaceae family of plants. Rosemary is a native of the Mediterranean regions of Europe and the Near East; Tunisia is a major modern-day source of the plant. Rosemary can grow as tall as 5 ft, producing strongly scented, leathery leaves used in perfumes and seasonings. Its Latin name, *Rosmarinus*, means "ocean dew." Other names for rosemary include compass weed, compass plant, or polar plant. An interesting tradition about rosemary is that it grows best in gardens tended by forceful or strong-willed women; a Spanish folk

saying has it that "where rosemary thrives the mistress is master."

The major chemical compounds found in essential oil of rosemary include eugenol, borneol, camphene, camphor, cineol, lineol, pinene, and terpineol. Compounds found in rosemary that are considered to be highly effective antioxidants include monoterpenoid ketone compounds, such as thujone, camphor, verbenone and carvone, as well as such phenols as methylchavicol, carvacrol, eugenol and thymole. Rosemary extract also contains numerous polyphenolic compounds that possess high antioxidant activity, including rosmanol, rosmaridiphenol, rosmarinic acid, carnosol, carnosic acid, and ursolic acid.

Purpose

Although rosemary is most familiar to contemporary Westerners as a kitchen herb used to add a spicy or slightly medicinal flavor to some foods, it was traditionally used as an antiseptic, astringent, and food preservative before the invention of refrigeration. It was burned in sickrooms to disinfect the air. Rosemary's antioxidant properties are still used to extend the shelf life of prepared foods.

Rosemary is also a well known "middle note" in the making of perfumes and **aromatherapy** products. The aroma of its essential oil lasts about two to three days, and is regarded as having energizing and invigorating qualities. It is thought to improve memory and the ability to concentrate, and has been used to relieve migraine headaches. Its astringent qualities make it appropriate for use in facial cleansers for oily skin. Rosemary is frequently added to compresses to heal bruises and sprains, and in topical salves, lotions, or creams to relieve muscle cramps or improve circulation. It is a favorite ingredient in hand creams for gardeners or for use in cold weather. The herb contains a flavonoid called diosmin, which has been shown to strengthen capillaries in the circulatory system. Some research studies are investigating the usefulness of rosemary in the treatment of varicose veins and hemorrhoids. The German Commission E has approved the use of rosemary for low blood pressure, and for painful joints or muscles. In addition, rosemary is still listed as a medicinal herb in the official *United States Pharmacopoeia.*

Several of the compounds in rosemary have been shown to have anti-inflammatory effects. As a result, some cancer researchers are studying rosemary as a natural non-steroidal anti-inflammatory drug, or NSAID. Since the use of NSAIDs is associated with a lowered risk of certain types of cancer in the general population, these researchers are investigating the possibility that rosemary may act as a cancer preventive.

Description

Essential oil and extract of rosemary are prepared for use in aromatherapy by steam distillation from the leaves and flowers of the plant during its second year of growth. The leaves can also be stripped from the stems of the second-year plant and dried for internal use. Although rosemary is more commonly used to flavor dishes rather than as a separate item in the diet, it can be taken as a tea.

Recommended dosages

Rosemary tea is made by pouring 1 cup of boiling water into a cup containing 1 teaspoon of the dried leaves. Tea made from fresh rosemary leaves require .35 ounces–.52 ounces of the herb. The tea may be taken up to three times daily.

Essential oil of rosemary should not be used full-strength on the skin, as it has been reported to cause skin irritation. When it is diluted, as in a carrier oil for massage or in a salve, hand cream, or facial cleanser, it is safe for use as often as desired. In aromatherapy rosemary oil can be used in burners, potpouri, or in sachets.

Precautions

Rosemary tea should not be taken by pregnant or lactating women, although they may safely use it in cooking to season food. Children under six months of age also should not be given rosemary tea. Rosemary should not be taken by persons with epilepsy, ulcerative colitis, or high blood pressure.

Side effects

When rosemary is harvested appropriately and used within recommended guidelines, side effects are minimal. A few instances of allergic skin reactions to topical preparations containing rosemary have been reported.

Recent European research has shown that rosemary interferes with the absorption of iron in the diet, which indicates that it should not be used internally by persons with iron deficiency anemia.

Interactions

Rosemary is not known to interact with any current Western prescription medications.

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Rebecca J. Frey, Ph.D.

Rumination disorder

KEY TERMS

Merycism—Another name for rumination disorder.

Regurgitation—The return of partly digested food from the stomach to the mouth. Regurgitation may be either an intentional act or an involuntary physical reaction.

Ruminate—To chew or rechew regurgitated food.

Definition

Rumination disorder may be diagnosed when a person deliberately brings food back up into the mouth and either rechews and reswallows it or spits it out.

Description

Rumination disorder is sometimes called merycism. It is a disorder most commonly found in infants, and associated with **mental retardation**. During rumination, previously eaten food is intentionally brought back into the mouth. Sometimes the child spits it out, but in other cases, the food is rechewed and reswallowed. The regurgitation is not caused by a medical condition. In many cases, the child has had an illness associated with vomiting that occurs before the onset of rumination disorder. Rumination has also been observed in severe cases of eating disorders among teenagers as well as adults.

Causes and symptoms

Causes

There is no general agreement on the causes of rumination disorder. In infants, it is thought to be caused by a lack of nurturing or physical contact. The child's rumination may represent an attempt to stimulate or soothe him- or herself. Biological factors are also being explored as possible causes of rumination disorder.

Symptoms

The symptoms of rumination include both the regurgitation of food and, in infants, the effort made to regurgitate that food. In infants, the attempts to bring up food can include putting fingers in the mouth, sucking on the tongue, and arching the back. When food is brought up, the cheeks expand and appear puffed. Sometimes an observer can detect the rechewing; the person often appears to take pleasure in the act. The person's breath may have a foul or sour odor. Some infants, especially those who have just begun ruminating, will expel most or all of the regurgitated food from their mouths. When this expulsion occurs, it is often mistaken for normal infant vomiting. As an infant continues to ruminate, he or she often learns to keep more and more of the regurgitated food in the mouth.

Demographics

Rumination disorder occurs primarily in infants. The onset usually occurs before the infant's first birthday. The disorder is also more common in people with mental retardation. The onset of rumination disorder is typically later in mentally retarded patients, however; it may not appear until puberty or even the early adult years. Rumination disorder is rare and thought to occur more

often in males than in females. People who have **anorexia nervosa** or **bulimia nervosa** may begin to ruminate only in adult life. One report found that up to 20% of people with bulimia may ruminate.

Diagnosis

The ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (*DSM-IV-TR*), which is the standard reference work for mental health professionals, gives only three general criteria for diagnosing rumination disorder. The first is that the person's behavior of deliberately bringing up and rechewing food must have lasted for at least a month. The regurgitation and rechewing must happen after a period of time in which the person did not ruminate. In addition, the rumination cannot result from a medical condition such as esophageal reflux. In addition, the manual specifies that the rumination cannot be associated with anorexia or bulimia.

Rumination disorder may be difficult to diagnose. One reason for this difficulty is that infants or adults who do not expel any of their regurgitated food can often be identified only by a puffing of the cheeks when the food is in the mouth or by an unpleasant breath odor. In addition, because many people and infants who ruminate find the experience a positive and pleasurable one, there are no physical signs of discomfort to bring the disorder to the attention of caretakers or others.

Some experts disagree with the statement of the *Diagnostic and Statistical Manual* that a **diagnosis** of rumination disorder cannot be made if the rumination is associated with anorexia or bulimia. These experts maintain that diagnosing and treating rumination disorder in patients who have other eating disorders is important for the sake of the patient's health.

Treatment

Treatment for rumination disorder depends on the cause of the behavior. Infants who are thought to ruminate because of a lack of affection may be fed by someone other than their mother or father. This person can be a replacement while their parents receive treatment themselves. Other approaches involve therapy and parenting education to create a stronger bond between the parents and the child.

The treatment of adult patients includes giving them chewing gum to use when rumination might normally occur. Other researchers have found that giving mentally retarded adults filling meals may reduce rumination. Often, treating such eating disorders as anorexia or bulimia helps to resolve the rumination that may be associated with those disorders. **Behavior modification** techniques that help a patient to unlearn the ruminating behavior have also been used.

Prognosis

In many cases, rumination that begins in infancy stops on its own. The disorder should be treated, however, because infants with untreated rumination disorder are at risk of malnutrition and death caused by dehydration. Treatments for rumination disorder are generally very effective. Treatment of associated eating disorders in adults is generally regarded as successful.

Prevention

There is no known way to prevent rumination disorder. It is possible, however, that a strong parent-child bond may reduce the possibility of the disorder occurring in infants.

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ORGANIZATIONS

American Academy of Pediatrics. 141 Northwest Point Boulevard, Elk Grove Village, IL 60007-1098. (847) 434-4000. <www.aap.org>.

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S

SAD *see* **Seasonal affective disorder**

Sadism *see* **Sexual sadism**

SAMe

Definition

SAMe (or S-adenosyl-L-methionine) is a naturally occurring chemical that is found throughout the entire body. It is involved in many chemical reactions that are necessary for life. SAMe is available as a natural dietary supplement that can be found at some pharmacies or health food stores, and can be purchased without a prescription.

Purpose

People take supplements of SAMe for many reasons including its possible antidepressant effects. Some evidence suggests that taking SAMe can improve symptoms of depression within two weeks, which is considerably faster than the time it takes for oral antidepressant prescription drugs to work. (Prescription antidepressants often take a minimum of two weeks for patients to begin noticing any effect, and many take four to six weeks.)

Description

SAMe is a specific form of the amino acid methionine, a substance that, when not metabolized properly, allows homocysteine to build up in the blood. SAMe is also an antioxidant, a substance that protects the body from damaging reactive oxygen molecules in the body. These reactive oxygen molecules can come from inside the body or from environmental pollution and are thought to play a role in the aging process and the development of degenerative disease. In general, SAMe is thought to raise the level of functioning of other amino acids in the body.

Although people use SAMe for many reasons including osteoarthritis, depression, heart disease, fibromyalgia, bursitis, tendonitis, chronic low back pain, **dementia**, **Alzheimer's disease**, improving **brain** function, multiple sclerosis, spinal cord injuries, migraine headaches, lead poisoning, liver disease, and to slow aging, the best evidence to date indicates that SAMe may be effective in relieving symptoms of osteoarthritis and for treating depression.

Several studies have indicated that oral SAMe and intravenous SAMe are effective treatments for depression. The studies researching the oral SAMe were small studies, and often were of short duration. However, the studies indicate that SAMe is effective in treating depression, and that it may be almost as effective as tricyclic antidepressants. Larger studies of SAMe are necessary.

Recommended dosage

SAMe can be taken orally or intravenously. Oral administration is more common. When taken by mouth, doses of 400–1,600 mg have been suggested. For osteoarthritis, 200–600 mg daily is a typical dose. For depression, 400–1,600 mg daily is a typical dose.

200 mg of SAMe have been administered intravenously or intramuscularly for 14 days while the patient simultaneously begins therapy with prescription antidepressant medication. If SAMe is used without prescription antidepressants, 200-400 mg per day by intravenous or intramuscular injections has been used. When treating other medical conditions, doses as high as 800 mg daily by injection have been used. Again, however, intravenous administration is rare in the United States.

Precautions

As a natural supplement, SAMe has not been evaluated by the Food and Drug Administration. Claims of safety or effectiveness for treating any medical disorder have not been thoroughly studied by any governmental

KEY TERMS

Antidepressant—A medication used to treat the symptoms of depression.

Bipolar disorder—A mental disorder characterized by dramatic, and sometimes rapid mood swings, resulting in both manic and depressive episodes; formerly called manic-depressive disorder.

Depression—A mental state characterized by excessive sadness. Other symptoms include altered sleep patterns, thoughts of suicide, difficulty concentrating, agitation, lack of energy, and loss of enjoyment in activities that are usually pleasurable.

Homocysteine—A chemical that builds up in the blood when methionine is not properly processed. High blood levels of homocysteine increase the risk of heart disease and stroke.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

agency and there is no regulation of natural supplements. This means that potencies may vary between lots or among different manufacturers. It is also possible that supplements may not contain the ingredients that are listed on product labels.

SAMe should be used carefully by individuals with a history of **bipolar disorder** since it may aggravate symptoms of mania. When used with prescription antidepressant medications, life-threatening symptoms may occur. It should be used with prescription antidepressant drugs only under close medical supervision.

Side effects

When taken by mouth, SAMe may cause stomach upset including gas, vomiting, diarrhea, headache, and nausea. These symptoms are more common at high doses.

Anxiety has also occurred in people with depression, and mania has been reported in those with a history of bipolar disorder.

Interactions

As stated, use of SAMe with antidepressants (especially selective serotonin reuptake inhibitors, or SSRIs) may cause life-threatening symptoms including agitation, tremors, anxiety, rapid heartbeats, difficulty breathing, diarrhea, shivering, muscle stiffness, and excessive sweating. The combination can also cause **insomnia**. If SAMe is used at the same time that prescription antidepressants are taken, close medical supervision is required.

SAMe may offer beneficial drug interactions when used with some medications. More research is necessary to determine whether SAMe does indeed protect the liver, but some scientists think that SAMe may protect the liver from damage caused by some drugs, including acetaminophen, alcohol, estrogens, steroids, and several other prescription drugs.

People interested in taking SAMe for depression should discuss its use with their doctor to weigh the potential benefits and risks.

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Schizoaffective disorder

Definition

One of the most challenging mental disorders to identify accurately and treat appropriately is schizoaffective disorder. This condition involves both psychotic symptoms and conspicuous, long-enduring, severe symptoms of mood disorder. The cluster of symptoms experienced by persons with schizoaffective disorder can resemble—at various times in its course—bipolar disorder, major depressive episode with psychotic features, or **schizophrenia**.

The schizoaffective disorder classification is applied when a mental health client meets diagnostic criteria for

both schizophrenia and an "affective" (mood) disorder—depression or **bipolar disorder**. In schizoaffective disorder, the experiencing of mood and psychotic symptoms occurs predominantly simultaneously and the mood disturbance is long lasting. However, periods of experiencing serious psychotic symptoms without serious mood disturbance are also a definitive feature. In bipolar disorder and depression with psychotic features, psychotic symptoms *only* occur during an active episode of mania or severe clinical depression. Schizoaffective disorder is characterized by periods during which psychotic symptoms are experienced *without* simultaneous severe mood changes. If the patient is encountered for the first time during such a period of psychotic symptoms in the absence of mood changes, it can appear that the individual has schizophrenia. However, in a person who has psychotic symptoms, the presence of long-standing severe mood disturbance suggests possible schizoaffective disorder if there are also periods of psychotic symptoms *without* concurrent mood fluctuations.

Schizoaffective disorder is typically identified by a process of lengthy observation and elimination of another diagnostic alternative over a long course of care. Because of the need for longitudinal observation and collection of a wealth of information before an accurate **diagnosis** is possible, most people with schizoaffective disorder have borne other diagnostic labels prior to the schizoaffective diagnosis (usually, bipolar disorder).

Description

Psychotic symptoms

Both psychotic symptoms and mood disorder symptoms are experienced by the individual with schizoaffective disorder. In schizoaffective disorder, at least two of the major symptoms of **psychosis** are evident in the client. Classic psychotic symptoms can occur during mood disturbances as well as in periods without extreme mood changes. **Hallucinations**, **delusions**, and strange bodily movements or lack of movements (catatonic behavior) are all psychotic symptoms that may be observed. Additionally, minimal or peculiar speech, lack of drive to act on one's own behalf, bizarre or primitive (socially inappropriate or immature) behavior, a wooden quality to one's emotions, or near-absent emotionality are also typical psychotic symptoms that may occur. Of course, not all of the possible psychotic symptoms will occur concurrently in a single person with schizoaffective disorder. Importantly, to meet the criteria for the schizoaffective disorder diagnosis, delusions or hallucinations (the most "prototypical" of the psychotic symptoms) must be observed within a fairly lengthy period of time during which there is no form of mood disturbance.

Mood disturbance

An extremely important and challenging aspect of schizoaffective disorder is that mood problems are prominent. During mood episodes, psychotic features are simultaneously evident. The disruption of mood may be depressive, manic, or take the form of a **mixed episode** (which includes both depressive and manic features). If only depressed mood occurs, the individual is described as having the depressive subtype of schizoaffective disorder. If mixed episodes or manic episodes are noted, the client is identified as having the bipolar form of schizoaffective disorder.

Causes and symptoms

Causes

Because clear identification of schizoaffective disorder has traditionally been challenging, scientists have conducted far less research relating to the disorder than studies relating to schizophrenia or mood disorders. However, there are indications that there is a genetic component to the disorder. Close relatives of persons with schizoaffective disorder have higher rates of both schizophrenia and mood disorder. The disorder most typically strikes in early adulthood; in some cases, there appears to be a major trigger—some form of life **stress** initiating the occurrence of the symptoms. In cases where there is an identifiable stressor involved, the person tends to have a better outcome than when such is not the case. Some evidence suggests that the bipolar form of schizoaffective disorder is more treatable and yields better outcomes than the depressive form.

RELATIONSHIP TO PERSONALITY DISORDER. Persons with **personality disorders** appear to be more susceptible to developing psychotic reactions in response to stress. One aspect of personality disorder is that, when life becomes more demanding and difficult than can be tolerated, the individual with personality disorder may lapse into a brief psychotic episode. For some individuals, personality disorder may be a predecessor to the development of schizoaffective disorder. Apparently, a chronic problem of lacking effective adult mechanisms for coping with life becomes an ongoing schizoaffective disorder in some predisposed persons. Persons with preexisting schizotypal, paranoid, schizoid, and borderline personality disorders may be more vulnerable to develop a schizoaffective disorder than the general population.

Symptoms

The ***Diagnostic and Statistical Manual of Mental Disorders***, *DSM-IV-TR*, produced by the American

KEY TERMS

Catatonic behavior or catatonia—Catatonic behavior or catatonia is a descriptive term that describes both possible extremes related to movement. Catalepsy is the motionless aspect of catatonia—a person with catalepsy may remain fixed in the same position for hours on end. Rapid or persistently repeated movements, frequent grimacing and strange facial expressions, and unusual gestures are the opposite end of the catatonia phenomenon, involving an excess or distorted extreme of movement.

Delusion—A false belief that is resistant to reason or contrary to actual fact.

Hallucinations—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Psychosis or psychotic symptoms—Disruptions in perceiving reality, thinking logically, and speaking or behaving in normal fashion. Hallucinations, delusions, catatonic behavior, and peculiar speech are all symptoms of psychosis. In *DSM-IV-TR*, psychosis is usually one feature of an overarching disorder, not a disorder in itself (with the exception of the diagnosis *psychosis not otherwise specified.*

Psychiatric Association, is used by most mental health professionals in North America and Europe to diagnose mental disorders. The *DSM-IV-TR* provides these major criteria for schizoaffective disorder:

- At least two symptoms of psychosis from among the following, present for at least one month: Delusions; hallucinations; disorganized speech (strange, peculiar, difficult to comprehend); disorganized (bizarre or child-like) behavior; catatonic behavior; minimal speech (approaching mutism); lack of drive to act on one's own behalf; a wooden quality to one's emotions, or near-absent emotionality.
- Delusions or hallucinations have occurred for at least two weeks in the absence of prominent mood symptoms.
- During a "substantial portion" of the period of active illness, the individual meets criteria for one of the following mood disturbances: Major depressive episode, **manic episode**, mixed episode.
- The symptoms are not caused by a biologically active entity such as drugs, alcohol, adverse reaction to a medication, physical injury, or medical illness.

Demographics

Because of the imprecise nature of the diagnosis, the actual rate of brief schizoaffective disorder in adults is unknown. The proportion of schizoaffective disorder identified in persons undergoing treatment for psychiatric disorders has ranged from 2% to almost 30%, depending on the study cited. More females than males (overall) suffer from schizoaffective disorder. However, similar to gender ratios in clinical depression and bipolar disorder, it seems that there is a much higher ratio of women to men *in the depressive subtype* whereas the bipolar subtype has a more even gender distribution. Thus, the higher ratio of women overall is primarily caused by the concentration of women within the depressive subtype of schizoaffective disorder.

Diagnosis

Even using the *DSM-IV-TR* criteria, identification of schizoaffective disorder remains difficult and relatively subjective. An unusual condition in this set of diagnostic criteria is the need to weigh the relative prominence of the mood symptoms and to identify a period of psychotic symptoms that occurred without significant mood disturbance. In the various other psychotic disorders, there is frequently a low level of depression accompanying the symptoms. When depressive symptoms are the sole form of mood disturbance, only subjective clinical judgment determines whether there has been sufficient severity or duration of that disturbance to merit the possibility of schizoaffective disorder. An additional complication is the cultural relativity of "psychotic symptoms." If the psychotic-like behaviors shown are expected and valued in the person's culture or religion, and these behaviors occur in a traditionally affirming context such as religious services or **meditation**, then schizoaffective disorder would not be diagnosed.

As stated, schizoaffective disorder is typically identified by a process of lengthy observation and elimination of another diagnostic alternative over a long course of care. A very thorough history of the client's entire past experiences of psychiatric symptoms, mental health treatments, and response to different kinds of medications that have been taken, helps in determining whether that individual is suffering from schizoaffective disorder. Information about current and past experiences is collected in interviews with the client and possibly in discussion with the client's immediate family. Data also may be gathered from earlier medical records with the

client's consent. In order to examine the sufferer's ability to concentrate, to remember, to understand his or her situation realistically, and to think logically, the clinician may use a semi-structured interview called a mental status examination. The mental status examination is designed to uncover psychotic or demented thought processes. Psychological assessment instruments, such as the MMPI-2, The Rorschach Inkblot Test, various mood disorder questionnaires, or structured diagnostic interviews, are sometimes used as well to aid in diagnosis. The criteria used by the clinician to classify this constellation of symptoms as schizoaffective disorder are presented in the *DSM-IV-TR*.

Treatments

Atypical, novel, or newer-generation antipsychotic medications are very effective in schizoaffective disorder treatment. Examples of atypical or novel antipsychotic medications include **risperidone** (Risperdal), **quetiapine** (Seroquel), and **olanzapine** (Zyprexa). If the patient's psychotic symptoms are acute and accompanied by agitation, a number of different antipsychotics can be used to terminate the flare-up of acute agitated psychosis. Agitation is a state of frantic activity that is often accompanied by anger or marked fearfulness; when in an agitated state, the client is more likely to cause harm to self or others. In agitated psychotic states, the antipsychotic agent **haloperidol** (Haldol) is often given as an injection, accompanied by other medications that decrease anxiety and slow behavior (often **lorazepam**, also known as Ativan). At this time, there are no atypical antipsychotics available in an injectable formulation. If the client is not extremely agitated, usually a novel antipsychotic is used, given orally daily, for a lengthier period of time.

In some cases, the antipsychotic medication is not sufficient to overcome the mood disturbance component of the disorder, even though some antipsychotics have thymoleptic (mood-affecting) qualities. Some of the atypical antipsychotic medications are thought to have antidepressant properties, while olanzapine has an FDA approval for the management of acute manic psychosis.

If there is little response to novel antipsychotic monotherapy (treatment with only one medication) an additional compound may be given to target the mood disorder aspect of the illness. The choice of which drug should be added to the medication regimen to decrease mood disorder problems is determined by the subtype of schizoaffective disorder shown by the client. If the client experiences the bipolar form, a mood stabilizer is added, often **valproic acid** (Depakote), **carbamazepine** (Tegretol), or lithium (Eskalith or Lithabid). In schizoaffective disorder of the bipolar type, if little response occurs to the usual antipsychotic/mood stabilizer combinations, the mental health consumer may be prescribed **clozapine** (Clozaril or other generic formulations) which appears to be both anti-psychotic and mood-stabilizing. However, because clozapine has the potential (in a very minute number of cases) to cause lethal alterations in the composition of blood, and because its use requires regular monitoring with recurrent blood testing, it is reserved as a "last-resort" therapy. In cases of the depressive subtype, psychiatrists may prescribe an antidepressant such as **citalopram** (Celexa), **venlafaxine** (Effexor), **paroxetine** (Paxil), or **fluoxetine** (Prozac) as an adjunct to the antipsychotic. In certain cases of depressive subtypes, where medications have been ineffective in resolving the extreme mood or where psychosis is so severe as to be life-threatening, **electroconvulsive therapy** may be utilized. Electroconvulsive therapy has also been shown to be effective in major depressive episode with psychotic features.

Medication is not the only treatment avenue. Supportive **psychotherapy** and psychoeducation is helpful to decrease the client's fears and to inform the client about the psychiatric illness. **Cognitive-behavioral therapy** aims to modify the thoughts and behaviors that provoke mood disturbance or prevent full involvement and collaboration in therapy for the mental illness. Psychoeducation and cognitive-behavioral therapy are not effective in lieu of biological therapy, but are enhancing, meaningful components of a "whole-person" approach used in concert with medications for the best possible outcomes.

Prognosis

The prognosis for clients with schizoaffective disorder is largely dependent on the form of the disorder and the presence or absence of a trigger. If there is a major life event as a prompting stressor, or an unusual traumatic experience preceded the occurrence of the disorder, chances for improvement are higher. If there is not a particular triggering event, or if the schizoaffective disorder occurred in an individual with a premorbid personality disorder, the outcome is less likely to be positive. The bipolar form of the disorder may respond better to treatment than the depressive form. Generally, the earlier the disorder is identified and treated, and the fewer lapses from medications, the more positive the outcome.

Prevention

Given that this disorder appears to have a strong genetic or biologic aspect, society-wide prevention approaches are not likely to be fruitful. However, a promising strategy is to educate physicians, psychologists, and

social workers, as well as persons at higher risk for the disorder, about the characteristics and treatability of schizoaffective disorder. Such education of care providers and high-risk individuals would foster early identification and treatment. In schizoaffective disorder, similar to schizophrenia and bipolar disorder, better response is predicted the earlier treatment begins. Because theoretically, severe stressors can be a trigger for this disorder (in some cases), strong social support and immediate post-crisis counseling for severe stress could possibly prevent the development of the disorder in some susceptible persons.

See also Compliance

Resources

BOOKS

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National Alliance for the Mentally Ill. Colonial Place Three, 2107 Wilson Blvd., Suite 300, Arlington, VA 22201. Telephone: (703) 524-7600. NAMI HelpLine: (800) 950-NAMI (6264). Web site: <www.nami.org>.

National Association for Research on Schizophrenia and Affective Disorders (NARSAD). 60 Cutter Mill Road, Suite 404, Great Neck, NY 11021. Main Line: (516) 829-0091. Infoline: (800) 829-8289. Web site: <www.narsad.org>.

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Schizoid personality disorder

Definition

Schizoid personality disorder is characterized by a persistent withdrawal from social relationships and lack of emotional responsiveness in most situations. It is sometimes referred to as a "pleasure deficiency" because of the seeming inability of the person affected to experience joyful or pleasurable responses to life situations.

Description

A person with schizoid personality disorder has little or no interest in developing close interpersonal relationships. They appear aloof, introverted and prefer being alone. Those who know them often label them as shy or a "loner." They turn inward in an effort to shut out social relationships. It is common for a person with schizoid personality disorder to avoid groups of people or appear disinterested in social situations even when they involve family. They are often perceived by others as socially inept.

A closely related trait is the absence of emotional expression. This apparent void of emotion is routinely interpreted by others as disinterested, lacking concern and insensitive to the needs of others. The person with schizoid personality disorder has particular difficulty expressing anger or hostility. In the absence of any recognizable emotion, the person portrays a dull demeanor and is easily overlooked by others. The typical person with schizoid personality disorder prefers to be viewed as "invisible" since it aids their quest to avoid social contact with others.

The person with schizoid personality disorder may be able to hold a job and meet the expectations of an employer if the responsibilities do not require more than minimal interpersonal involvement. People with this disorder may be married, but do not develop close intimate relationships with their spouse and typically show no interest in sexual relations. Their speech is typically slow and monotonous with a lethargic demeanor. Because their tendency is to turn inward, they can easily become preoccupied with their own thoughts to the exclusion of what is happening in their environment. Attempts to communicate may drift into tangents or confusing associations. They are also prone to being absent-minded.

Causes and symptoms

Causes

The schizoid personality disorder has its roots in the family of the affected person. These families are typically emotionally reserved, have a high degree of formality, and have a communication style that is aloof and impersonal. Parents usually express inadequate amounts of affection to the child and provide insufficient amounts of emotional stimulus. This lack of stimulus during the first year of life is thought to be largely responsible for the person's disinterest in forming close, meaningful relationships later in life.

People with schizoid personality disorder have learned to imitate the style of interpersonal relationships modeled in their families. In this environment, affected people fail to learn basic communication skills that would enable them to develop relationships and interact effectively with others. Their communication is often vague and fragmented, which others find confusing. Many individuals with schizoid personality disorder feel misunderstood by others.

Symptoms

DSM-IV-TR specifies seven diagnostic criteria for schizoid personality disorder:

- Avoids close relationships: People with this disorder show no interest or enjoyment in developing interpersonal relationships; this may also include family members. They perceive themselves as social misfits and believe they can function best when not dependent on anyone except themselves. They rarely date, often do not marry, and have few, if any, friends.
- Prefers solitude: They prefer and choose activities that they can do by themselves without dependence upon or involvement by others. Examples of activities they might choose include mechanical or abstract tasks such as computer or mathematical games.
- Avoids sex: There is typically little or no interest in having a sexual experience with another person. This would include a spouse if the affected person is married.
- Lacks pleasure: There is an absence of pleasure in most activities. A person with schizoid personality disorder seems unable to experience the full range of emotion accessible to most people.
- Lacks close friends: People affected with this disorder typically do not have the social skills necessary to develop meaningful interpersonal relationships. This results in few ongoing social relationships outside of immediate family members.

KEY TERMS

Asperger's disorder—A condition in which young children experience impaired social interactions and develop limited repetitive patterns of behavior.

Autistic disorder—A developmental disability that appears early in life, in which normal brain development is disrupted and social and communication skills are retarded, sometimes severely.

Millon Clinical Multiaxial Inventory (MCMI-II)—A self-report instrument designed to help the clinician assess DSM-IV-related personality disorders and clinical syndromes. It provides insight into 14 personality disorders and 10 clinical syndromes.

Minnesota Multiphasic Personality Inventory (MMPI-2)—A comprehensive assessment tool widely used to diagnose personality disorders.

Rorschach Psychodiagnostic Test—This series of 10 "ink blot" images allows the patient to project their interpretations which can be used to diagnose particular disorders.

Thematic Apperception Test (TAT)—A projective test using stories and descriptions of pictures to reveal some of the dominant drives, emotions, sentiments, conflicts, and complexes of a personality.

- Indifferent to praise or criticism: Neither positive nor negative comments made by others elicit an emotionally expressive reaction. They don't appear concerned about what others might think of them. Despite their tendency to turn inward to escape social contact, they practice little introspection.
- Emotional detachment: Their emotional style is aloof and perceived by others as distant or "cold." They seem unable or uninterested in expressing empathy and concern for others. Emotions are significantly restricted and most social contacts would describe their personality as very bland, dull or humorless. The person with schizoid personality disorder rarely picks up on or reciprocates normal communicational cues such as facial expressions, head nods, or smiles.

Demographics

Of all **personality disorders**, schizoid personality disorder is the least commonly diagnosed personality disorder in the general population. The prevalence is approximately one percent. It is diagnosed slightly more often in males.

Diagnosis

The symptoms of schizoid personality disorder may begin in childhood or adolescence showing as poor peer relationships, a tendency toward self-isolation, and underachievement in school. Children with these tendencies appear socially out-of-step with peers and often become the object of malicious teasing by their peers, which increases the feelings of isolation and social ineptness they feel.

For a **diagnosis** of schizoid personality disorder to be accurately made, there must be an ongoing avoidance of social relationships and a restricted range of emotion in interpersonal relationships that begin by early adulthood. There must also be the presence of at least four of the above-mentioned symptoms.

A common difficulty in diagnosing schizoid personality disorder is distinguishing it from Autistic Disorder and **Asperger's Disorder**, which are characterized by more severe deficits in social skills. Other individuals who would display social habits that might be viewed as "isolating" should not be given the diagnosis of schizoid personality disorder unless the personality traits are inflexible and cause significant obstacles to adequate functioning.

The diagnosis is based on a clinical interview to assess symptomatic behavior. Other assessment tools helpful in diagnosing schizoid personality disorder include:

- Minnesota Multiphasic Personality Inventory (MMPI-2)
- Millon Clinical Multiaxial Inventory (MCMI-II)
- Rorschach Psychodiagnostic Test
- **Thematic Apperception Test** (TAT)

Treatments

A major goal of treating a patient diagnosed with schizoid personality disorder is to combat the tendencies toward social withdrawal. Strategies should focus on enhancing self-awareness and sensitivity to their relational contacts and environment.

Psychodynamically oriented therapies

A psychodynamic approach would typically not be the first choice of treatment due to the patient's poor ability to explore his or her thoughts, emotions, and behavior. When this treatment is used, it usually centers around building a therapeutic relationship with the patient that can act as a model for use in other relationships.

Cognitive-behavioral therapy

Attempting to cognitively restructure the patient's thoughts can enhance self-insight. Constructive ways of accomplishing this would include concrete assignments such as keeping daily records of problematic behaviors or thoughts. Another helpful method can be teaching social skills through role-playing. This might enable individuals to become more conscious of communication cues given by others and sensitize them to others' needs.

Group therapy

Group therapy may provide the patient with a socializing experience that exposes them to feedback from others in a safe, controlled environment. It can also provide a means of learning and practicing social skills in which they are deficient. Since the patient usually avoids social contact, timing of group therapy is of particular importance. It is best to develop first a therapeutic relationship between therapist and patient before starting a group therapy treatment.

Family and marital therapy

It is unlikely that a person with schizoid personality disorder will seek **family therapy** or marital therapy. If pursued, it is usually on the initiative of the spouse or other family member. Many people with this disorder do not marry and end up living with and are dependent upon first-degree family members. In this case, therapy may be recommended for family members to educate them on aspects of change or ways to facilitate communication. Marital therapy (also called **couples therapy**) may focus on helping the couple to become more involved in each other's lives or improve communication patterns.

Medications

Some patients with this disorder show signs of anxiety and depression which may prompt the use of medication to counteract these symptoms. In general, there is to date no definitive medication that is used to treat schizoid symptoms.

Prognosis

Since a person with schizoid personality disorder seeks to be isolated from others, which includes those who might provide treatment, there is only a slight chance that most patients will seek help on their own initiative. Those who do may stop treatment prematurely because of their difficulty maintaining a relationship with the professional or their lack of motivation for change.

If the degree of social impairment is mild, treatment might succeed if its focus is on maintenance of relationships related to the patient's employment. The patient's need to support him- or herself financially can act as a higher incentive for pursuit of treatment outcomes.

Once treatment ends, it is highly likely the patient will relapse into a lifestyle of social isolation similar to that before treatment.

Prevention

Since schizoid personality disorder originates in the patient's family of origin, the only known preventative measure is a nurturing, emotionally stimulating and expressive caretaking environment.

See also Cognitive-behavioral therapy; Rorschach technique

Resources

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ORGANIZATIONS

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Schizophrenia

Definition

Schizophrenia is the most chronic and disabling of the severe mental disorders, associated with abnormalities of **brain** structure and function, disorganized speech and behavior, **delusions**, and **hallucinations**. It is sometimes called a psychotic disorder or a **psychosis**.

Description

People diagnosed with schizophrenia do not always have the same set of symptoms; in addition, a given patient's symptoms may change over time. Since the nineteenth century, doctors have recognized different subtypes of the disorder, but no single classification system has gained universal acceptance. Some psychiatrists prefer to speak of schizophrenia as a group or family of disorders ("the schizophrenias") rather than as a single entity. A standard professional reference, **The Diagnostic and Statistical Manual of Mental Disorders** (also known as the *DSM-IV-TR)* acknowledges that its present classification of subtypes is not fully satisfactory for either clinical or research purposes, and states that "alternative subtyping schemes are being actively investigated."

The symptoms of schizophrenia can appear at any time after age six or seven, although onset during adolescence and early adult life is the most common pattern. There are a few case studies in the medical literature of schizophrenia in children younger than five, but they are extremely rare. Schizophrenia that appears after age 45 is considered late-onset schizophrenia. About 1%–2% of cases are diagnosed in patients over 80.

The onset of symptoms in schizophrenia may be either abrupt (sudden) or insidious (gradual). Often, however, it goes undetected for about two to three years after the onset of diagnosable symptoms, because the symptoms occur in the context of a previous history of cognitive and behavioral problems. The patient may have had panic attacks, **social phobia**, or substance abuse problems, any of which can complicate the process of **diagnosis**. In most cases, however, the patient's first psychotic episode is preceded by a prodromal (warning) phase, with a variety of behaviors that may include angry outbursts, withdrawal from social activities, loss of attention to personal hygiene and grooming, anhedonia (loss of one's capacity for enjoyment), and other unusual behaviors. The psychotic episode itself is typically characterized by delusions, which are false but strongly held beliefs that result from the patient's inability to separate real from unreal events; and hallucinations, which are disturbances of sense perception. Hallucinations can affect any of the senses, although the most common form of hallucination in schizophrenia is auditory ("hearing voices"). Autobiographical accounts by people who have recovered from schizophrenia indicate that these hallucinations are experienced as frightening and confusing. Patients often find it difficult to concentrate on work, studies, or formerly pleasurable activities because of the constant "static" or "buzz" of hallucinated voices.

There is no "typical" pattern or course of the disorder following the first acute episode. The patient may never have a second psychotic episode; others have occasional episodes over the course of their lives but can lead fairly normal lives otherwise. About 70% of patients diagnosed with schizophrenia have a second psychotic breakdown within five to seven years after the first one.

KEY TERMS

Affect—The expression of emotion displayed to others through facial expressions, hand gestures, tone of voice, etc. Types of affect include: flat (inanimate, no expression), blunted (minimally responsive), inappropriate (incongruous expressions of emotion relative to the content of a conversation), and labile (sudden and abrupt changes in type and intensity of emotion).

Agranulocytosis—A blood disorder characterized by a reduction in the number of circulating white blood cells (granulocytes). White blood cells defend the body against infections. Agranulocytosis is a potential side effect of some of the newer antipsychotic medications used to treat schizophrenia.

Akathisia—Agitated or restless movement, usually affecting the legs. Movement is accompanied by a sense of discomfort and an inability to sit, stand still, or remain inactive for periods of time. Akathisia is a common side effect of some neuroleptic (antipsychotic) medications.

Anhedonia—Loss of the capacity to experience pleasure. Anhedonia is one of the so-called negative symptoms of schizophrenia, and is also a symptom of major depression.

Anosognosia—Lack of awareness of the nature of one's illness. The term is usually applied to stroke patients, but is sometimes used to refer to lack of insight on the part of patients with schizophrenia. Anosognosia appears to be caused by the illness itself; it does not appear to be a form of denial or inappropriate coping mechanism. It is, however, a factor in nonadherence to treatment regimens and the increased risk of relapse.

Atypical antipsychotics—A group of newer medications for the treatment of psychotic symptoms that were introduced in the 1990s. The atypical antipsychotics include clozapine, risperidone, quetiapine, ziprasidone, and olanzapine. They are sometimes called serotonin dopamine antagonists, or SDAs.

Blunted affect—A term that refers to the loss of emotional expressiveness sometimes found in patients with schizophrenia. It is sometimes called flattened affect.

Catatonia—Disturbance of motor behavior with either extreme stupor or random, purposeless activity.

Delusion—A false belief that is resistant to reason or contrary to actual fact. Common delusions in schizophrenia include delusions of persecution, delusions about one's importance (sometimes called delusions of grandeur), or delusions of being controlled by others.

Dementia praecox—A late nineteenth-century term for schizophrenia.

Dopamine—A neurotransmitter that acts within certain brain cells to help regulate emotions and movement. Some of the symptoms of schizophrenia are related to excessive levels of dopamine activity in a part of the brain called the striatum.

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

First-rank symptoms—A list of symptoms that have been considered to be diagnostic of schizophrenia. They include delusions; somatic hallucinations; hearing voices commenting on one's behavior; and thought insertion or withdrawal. First-rank symptoms are sometimes called Schneiderian symptoms, after the name of Kurt Schneider, the German psychiatrist who listed them in 1959.

Gingko—A shade tree native to China with fan-shaped leaves and fleshy seeds with edible kernels. Gingko extract is being studied as a possible

Some patients remain chronically ill; of these, some remain at a fairly stable level while others grow steadily worse and become severely disabled.

About 20% of patients with schizophrenia recover the full level of functioning that they had before the onset of the disorder, according to NIMH statistics; but the remaining 80% have problems reintegrating into mainstream society. These patients are often underachievers in school and in the workplace, and they usually have difficulty forming healthy relationships with others. The majority (60%–70%) of patients with schizophrenia do not marry or have children, and most have very few friends or social contacts. The impact of these social difficulties as well as the **stress** caused by the symptoms themselves is reflected in the high **suicide** rate among patients with schizophrenia. About 10% commit suicide

KEY TERMS CONTINUED

complementary or adjunctive treatment for schizophrenia.

Hallucination—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Hebephrenic schizophrenia—An older term for what is now known as the disorganized subtype of schizophrenia.

Insidious—Proceeding gradually and inconspicuously but with serious effect. Schizophrenia sometimes has an insidious rather than an acute onset.

Morbidity—The unhealthiness or disease characteristics associated with a mental disorder.

Negative symptoms—Symptoms of schizophrenia that represent a loss or reduction of normal functioning.

Neuroleptic—Another name for the older antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

Polygenic—A trait or disorder that is determined by a group of genes acting together. Most human characteristics, including height, weight, and general body build, are polygenic. Schizophrenia and late-onset Alzheimer's disease are considered polygenic disorders.

Positive symptoms—Symptoms of schizophrenia that represent excesses or distortions of normal mental functions.

Prodromal—Premonitory; having the character of a warning. The first psychotic episode in schizophrenia is often preceded by a prodromal phase.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Reality testing—A phrase that refers to a person's ability to distinguish between subjective feelings and objective reality. A person who knows that their body is real even though they may be experiencing it as unreal, for example, is said to have intact reality testing.

Referential—A type of delusion in which the person misinterprets items, minor occurrences, or other people's behavior as referring to them. Misinterpretations of this sort that are not as resistant to reality as a delusion are sometimes called ideas of reference.

Schneiderian symptoms—Another name for first-rank symptoms of schizophrenia.

Striatum—A part of the basal ganglia, a deep structure in the cerebral hemisphere of the brain. Abnormally high levels of dopamine in the striatum are thought to be related to the delusions and hallucinations of schizophrenia.

Supportive—An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or educational approaches to treatment.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

Thought insertion/withdrawal—The notion that an outside force (space aliens, evil people, etc.) can put thoughts or ideas into one's mind or remove them. It is considered one of the first-rank symptoms of schizophrenia.

within the first 10 years after their diagnosis— a rate 20 times higher than that of the general population.

Subtypes of schizophrenia

DSM-IV-TR specifies five subtypes of schizophrenia:

- Paranoid type. The central feature of this subtype is the presence of auditory hallucinations or delusions alongside relatively unaffected mood and cognitive functions. The patient's delusions usually involve persecution, grandiosity, or both. About a third of patients diagnosed with schizophrenia in the United States belong to this subtype.
- Disorganized type. The core features of this subtype include disorganized speech, disorganized behavior, and flat or inappropriate affect. The person may lose

the ability to perform most activities of daily living, and may also make faces or display other oddities of behavior. This type of schizophrenia was formerly called hebephrenic (derived from the Greek word for puberty), because some of the patients' behaviors resemble adolescent silliness.

- Catatonic type. **Catatonia** refers to disturbances of movement, whether remaining motionless for long periods of time or excessive and purposeless movement. The absence of movement may take the form of catalepsy, which is a condition in which the patient's body has a kind of waxy flexibility and can be repositioned by others; or negativism, a form of postural rigidity in which the patient resists being moved by others. A catatonic patient may assume bizarre postures or imitate the movements of other people.
- Undifferentiated type. Patients in this subtype have some of the characteristic symptoms of schizophrenia but do not meet the full criteria for the paranoid, disorganized, or catatonic subtypes.
- Residual type. Patients in this category have had at least one psychotic episode, continue to have some **negative symptoms** of schizophrenia, but do not have current psychotic symptoms.

Cultural variables

There appear to be some differences across cultures in the symptoms associated with schizophrenia. The catatonic subtype appears to be more common in non-Western countries than in Europe or North America. Other studies indicate that persons diagnosed with schizophrenia in developing countries have a more acute onset of the disorder but better outcomes than patients in the developed countries.

Causes and symptoms

Causes

As of 2002, schizophrenia is considered the end result of a combination of genetic, biochemical, developmental, and environmental factors, some of which are still not completely understood. There is no known single cause of the disorder.

GENETIC. Researchers have known for many years that first-degree biological relatives of patients with schizophrenia have a 10% risk of developing the disorder, as compared with 1% in the general population. The monozygotic (identical) twin of a person with schizophrenia has a 40%–50% risk. The fact that this risk is not higher, however, indicates that environmental as well as genetic factors are implicated in the development of schizophrenia.

Some specific regions on certain human chromosomes have been linked to schizophrenia. In late 2001, a multidisciplinary team of researchers reported positive associations for schizophrenia on chromosomes 15 and 13. Chromosome 15 is linked to schizophrenia in European-American families as well as some Taiwanese and Portuguese families. A recent study of the biological pedigrees found among the inhabitants of Palau (an isolated territory in Micronesia) points to chromosomes 2 and 13. Still another team of researchers has suggested that a disorder known as 22q deletion syndrome may actually represent a subtype of schizophrenia, insofar as people with this syndrome have a 25% risk of developing schizophrenia. At present scientists are inclined to think that the genetic factors underlying schizophrenia vary across different ethnic groups, so that it is highly unlikely that susceptibility to the disorder is determined by only one gene. As of 2002, schizophrenia is considered a polygenic disorder.

There appears to be a connection between aging and genetic mutations that increases susceptibility to schizophrenia. A recent Israeli study found that the age of a person's father is a risk factor for schizophrenia; the older the father, the higher the rate of mutations in sperm cells. The child of a father older than 50 is three times more likely to develop schizophrenia than children born to younger men. The researchers suggest that mutations in the sperm cells of older men help to explain why schizophrenia has persisted in the human population even though few schizophrenics marry and have children.

DEVELOPMENTAL. As of 2002, there is some evidence that schizophrenia may be a type of developmental disorder related to the formation of faulty connections between nerve cells during fetal development. The changes in the brain that normally occur during puberty then interact with these connections to trigger the symptoms of the disorder. Other researchers have suggested that a difficult childbirth may result in developmental vulnerabilities that eventually lead to schizophrenia.

NEUROBIOLOGICAL. In early 2002, researchers at the NIMH demonstrated the existence of a connection between two abnormalities of brain functioning in patients with schizophrenia. The researchers used radioactive tracers and **positron emission tomography** (PET) to show that reduced activity in a part of the brain called the prefrontal cortex was associated in the patients, but not in the control subjects, with abnormally elevated levels of dopamine in the striatum. High levels of dopamine are related to the delusions and hallucinations of psychotic episodes in schizophrenia. The findings suggest that treatment directed at the prefrontal cortex might be more effective than present antipsychotic medications,

which essentially target dopamine levels without regard to specific areas of the brain.

Another area of investigation concerns abnormalities in brain structure that are found in some patients with schizophrenia. One of these abnormalities is the increased size of the ventricles, which are cavities in the interior of the brain filled with cerebrospinal fluid. Another is a decrease in size of some areas of the brain. A California study of MRI scans of teenagers with early-onset schizophrenia found that they lost over 10% of the gray matter of the brain over the course of five years. The frontal eye fields showed the most rapid rate of tissue loss—about 5% per year. A major difficulty in interpreting these findings is that these abnormalities are not found in the brains of all patients with schizophrenia. In addition, they sometimes occur in the brains of people who do not have the disorder.

ENVIRONMENTAL. Certain environmental factors during pregnancy are associated with an increased risk of schizophrenia in the offspring. These include the mother's exposure to starvation or famine; influenza during the second trimester of pregnancy; and Rh incompatibility in a second or third pregnancy.

Some researchers are investigating a possible connection between schizophrenia and viral infections of the hippocampus, a structure in the brain that is associated with memory formation and the human stress response. It is thought that damage to the hippocampus might account for the sensory disturbances found in schizophrenia. Another line of research related to viral causes of schizophrenia concerns a protein deficiency in the brain. Researchers at the University of Kiel in Germany think that the deficiency is the result of viral infections.

Environmental stressors related to home and family life (parental death or divorce, family dysfunction) or to separation from the family of origin in late adolescence (going away to college or military training; marriage) may trigger the onset of schizophrenia in individuals with genetic or psychological vulnerabilities.

Symptoms

The symptoms of schizophrenia are divided into two major categories: **positive symptoms**, which are defined by *DSM-IV-TR* as excesses or distortions of normal mental functions; and **negative symptoms**, which represent a loss or reduction of normal functioning. Of the two types, the negative symptoms are more difficult to evaluate because they may be influenced by a concurrent depression or a dull and unstimulating environment, but they account for much of the morbidity (unhealthiness) associated with schizophrenia.

POSITIVE SYMPTOMS. The positive symptoms of schizophrenia include four so-called "first-rank" or Schneiderian symptoms, named for a German **psychiatrist** who identified them in 1959:

- Delusions. A delusion is a false belief that is resistant to reason or to confrontation with actual facts. The most common form of delusion in patients with schizophrenia is persecutory; the person believes that others—family members, clinical staff, terrorists, etc.—are "out to get" them. Another common delusion is referential, which means that the person interprets objects or occurrences in the environment (a picture on the wall, a song played on the radio, laughter in the corridor, etc.) as being directed at or referring to them.
- Somatic hallucinations. Somatic hallucinations refer to sensations or perceptions about one's body organs that have no known medical cause, such as feeling that snakes are crawling around in one's intestines or that one's eyes are emitting radioactive rays.
- Hearing voices commenting on one's behavior or talking to each other. Auditory hallucinations are the most common form of hallucination in schizophrenia, although visual, tactile, olfactory, and gustatory hallucinations may also occur. Personal accounts of recovery from schizophrenia often mention "the voices" as one of the most frightening aspects of the disorder.
- Thought insertion or withdrawal. These terms refer to the notion that other beings or forces (God, aliens from outer space, the CIA, etc.) can put thoughts or ideas into one's mind or remove them.

Other positive symptoms of schizophrenia include:

- Disorganized speech and thinking. A person with schizophrenia may ramble from one topic to another (derailment or loose associations); may give unrelated answers to questions (tangentiality); or may say things that cannot be understood because there is no grammatical structure to the language ("word salad" or incoherence).
- Disorganized behavior. This symptom includes such behaviors as agitation; age-inappropriate silliness; inability to maintain personal hygiene; dressing inappropriately for the weather; sexual self-stimulation in public; shouting at people, etc. In one case study, the patient played his flute for hours on end while standing on top of the family car.
- Catatonic behavior. Catatonic behaviors have been described with regard to the catatonic subtype of schizophrenia. This particular symptom is sometimes found in other mental disorders.

NEGATIVE SYMPTOMS. The negative symptoms of schizophrenia include:

- Blunted or flattened affect. This term refers to loss of emotional expressiveness. The person's face may be unresponsive or expressionless, and speech may lack vitality or warmth.
- Alogia. Alogia is sometimes called poverty of speech. The person has little to say and is not able to expand on their statements. A doctor examining the patient must be able to distinguish between alogia and unwillingness to speak.
- Avolition. The person is unable to begin or stay with goal-directed activities. They may sit in one location for long periods of time or show little interest in joining group activities.
- Anhedonia. Anhedonia refers to the loss of one's capacity for enjoyment or pleasure.

OTHER SYMPTOMS AND CHARACTERISTICS. Although the following symptoms and features are not diagnostic criteria of schizophrenia, most patients with the disorder have one or more:

- Dissociative symptoms, particularly **depersonalization** and derealization.
- Anosognosia. This term originally referred to the inability of **stroke** patients to recognize their physical disabilities, but is sometimes used to refer to lack of insight in patients with schizophrenia. Anosognosia is associated with higher rates of noncompliance with treatment, a higher risk of repeated psychotic episodes, and a poorer prognosis for recovery.
- High rates of substance abuse disorders. About 50% of patients diagnosed with schizophrenia meet criteria for substance abuse or dependence. While substance abuse does not cause schizophrenia, it can worsen the symptoms of the disorder. Patients may have particularly bad reactions to **amphetamines**, cocaine, PCP ("angel dust") or marijuana. It is thought that patients with schizophrenia are attracted to drugs of abuse as self-medication for some of their symptoms. The most common substance abused by patients with schizophrenia is tobacco; 90% of patients are heavy cigarette smokers, compared to 25%–30% in the general adult population. Smoking is a serious problem for people with schizophrenia because it interferes with the effectiveness of their antipsychotic medications as well as increasing their risk of lung cancer and other respiratory diseases.
- High risk of **suicide**. About 40% of patients with schizophrenia attempt suicide at least once, and 10% eventually complete the act.
- High rates of **obsessive-compulsive disorder** and panic disorder.
- Downward drift. Downward drift is a sociological term that refers to having lower levels of educational achievement and/or employment than one's parents.

VIOLENT BEHAVIOR. The connection between schizophrenia and personal assault or violence deserves mention because it is a major factor in the reactions of family members and the general public to the diagnosis. Researchers in both the United Kingdom and the United States have found that schizophrenia carries a heavier **stigma** than most other mental disorders, largely because of the mass media's fascination with bizarre murders, dismemberment of animals, or other gruesome acts committed by people with schizophrenia. Many patients report that the popular image of a schizophrenic as "a time bomb waiting to explode" is a source of considerable emotional stress.

Risk factors for violence in a patient diagnosed with schizophrenia include male sex, age below 30, prediagnosis history of violence, paranoid subtype, nonadherence to medication regimen, and heavy substance abuse. On the other hand, it should be noted that most crimes of violence are committed by people without a diagnosis of schizophrenia. In addition, a study of patients with schizophrenia living in the community found that "... individuals in this sample were at least 14 times more likely to be victims of a violent crime than to be arrested for one."

Demographics

In the United States, Canada, and Western Europe, the sex ratio in schizophrenia is 1.2:1, with males being affected slightly more often than females. There is a significant gender difference in average age at onset, however; the average for males is between ages 18 and 25, whereas for women there are two peaks, one between ages 25 and 35, and a second rise in incidence after age 45. About 15% of all women who develop schizophrenia are diagnosed after age 35. In some women, the first symptoms of the disorder appear postpartum (after giving birth). Many women with schizophrenia are initially misdiagnosed as having depression or **bipolar disorder**, because women with schizophrenia are likely to have more difficulties with emotional regulation than men with the disorder. In general, however, females have higher levels of functioning prior to symptom onset than males.

The incidence of schizophrenia in the United States appears to be uniform across racial and ethnic groups, with the exception of minority groups in urban neighborhoods in which they are a small proportion of the total population. A recent study done in the United Kingdom replicated American findings: there are significantly higher rates of schizophrenia among racial minorities living in large cities. The rates of schizophrenia are highest in areas

in which these minority groups form the smallest proportion of the local population. The British study included Africans, Caribbeans of African descent, and Asians.

The incidence of schizophrenia in most developed countries appears to be higher among people born in cities than among those born in rural areas. In addition, there appears to be a small historical/generational factor, with the incidence of schizophrenia gradually declining in later-born groups.

Schizophrenia is a leading cause of disability, not only in the United States, but in other developed countries around the world. In 1997, the World Health Organization (WHO) listed schizophrenia as the world's ninth leading cause of disability. According to the National Institute of Mental Health (NIMH), 2.2 million American adults, or 1.1% of the population over age 18, suffer from schizophrenia. Other estimates run as high as 1.5% of the population.

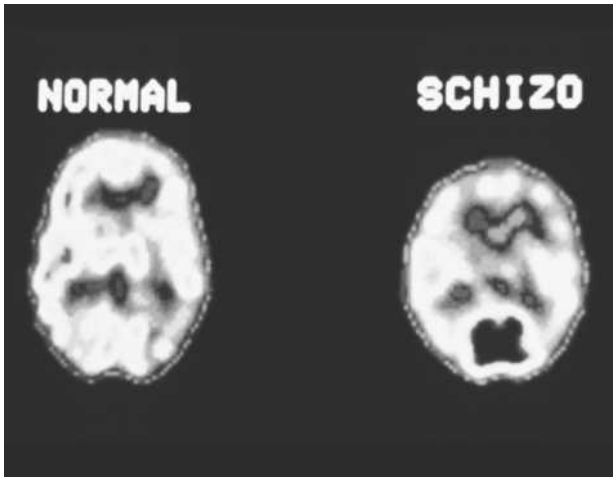
Schizophrenia is disproportionately costly to society for reasons that go beyond the sheer number of people affected by the disorder. Although patients with schizophrenia are little more than 1% of the population, they account for 2.5% of all health care costs—$40 billion per year in the United States, $2.35 billion in Canada (in Canadian dollars), and 2.6 billion pounds sterling (about $7.28 billion in US dollars) in Great Britain. In the United States, patients with schizophrenia fill 25% of all hospital beds and account for about 20% of all Social Security disability days.

In addition, the onset of the disorder typically occurs during a young person's last years of high school or their first years in college or the workforce; thus it often destroys their long-term plans for their future. According to the federal Agency for Healthcare Research and Quality, 70%–80% of people diagnosed with schizophrenia are either unemployed or underemployed (working in jobs well below their actual capabilities). Ten percent of Americans with permanent disabilities have schizophrenia, as well as 20%–30% of the homeless population.

Diagnosis

There are no symptoms that are unique to schizophrenia and no single symptom that is a diagnostic hallmark of the disorder. In addition, as of 2002 there are no laboratory tests or **imaging studies** that can establish or confirm a diagnosis of schizophrenia. The diagnosis is based on a constellation or group of related symptoms that are, according to *DSM-IV-TR,* "associated with impaired occupational or social functioning."

As part of the process of diagnosis, the doctor will take a careful medical history and order laboratory tests of the patient's blood or urine in order to rule out general medical conditions or substance abuse disorders that may be accompanied by disturbed behavior. X rays or other imaging studies of the head may also be ordered. Medical conditions to be ruled out include epilepsy, head trauma, brain tumor, Cushing's syndrome, Wilson's disease, Huntington's disease, and encephalitis. Drugs of abuse that may cause symptoms resembling schizophrenia include amphetamines ("speed"), cocaine, and phencyclidine (PCP). In older patients, **dementia** and **delirium** must be ruled out. If the patient has held jobs involving exposure to mercury, polychlorinated biphenyls (PCBs), or other toxic substances, environmental poisoning must also be considered in the differential diagnosis.



Colored positron emission tomography (PET) scans comparing normal brain (left) with the brain of a person with schizophrenia (right). *(Photo Researchers, Inc. Reproduced by permission.) See color insert for color version of photo.*

The doctor must then rule out other mental disorders that may be accompanied by psychotic symptoms, such as mood disorders; brief psychotic disorders; dissociative disorder not otherwise specified or **dissociative identity disorder**; **delusional disorder**; schizotypal, schizoid, or paranoid **personality disorders**; and **pervasive developmental disorders**. In children, childhood-onset schizophrenia must be distinguished from communication disorders with disorganized speech and from **attention-deficit/hyperactivity disorder**.

After the doctor has ruled out other organic and mental disorders, he or she must then determine whether the patient meets the following criteria, as specified by *DSM-IV-TR*:

- Presence of positive and negative symptoms. The patient must have two (or more) of the following symptoms during a one-month period: delusions; hallucinations; disorganized speech; disorganized or catatonic behavior; negative symptoms.

- Decline in social, interpersonal, or occupational functioning, including personal hygiene or self-care.
- Duration. The symptomatic behavior must last for at least six months.

Treatments

Current treatment of schizophrenia focuses on symptom reduction and relapse prevention, since the causes of the disorder have not yet been clearly identified. Unfortunately, not all patients with schizophrenia receive adequate treatment. In 2000, the NIMH released the results of a large-scale community study, which indicated that fewer than half of patients with schizophrenia receive correct dosages of their medications or adequate psychosocial treatment.

Medications

Medications are the mainstay of treatment for schizophrenia. Drug therapy for the disorder, however, is complicated by several factors: the unpredictability of a given patient's response to specific medications, the number of potentially troublesome side effects, the high rate of substance abuse among patients with schizophrenia, and the possibility of drug interactions between antipsychotic medications and antidepressants or other medications that may be prescribed for the patient.

NEUROLEPTICS. The first antipsychotic medications for schizophrenia were introduced in the 1950s, and known as dopamine antagonists, or DAs. They are sometimes called neuroleptics, and include **haloperidol** (Haldol), **chlorpromazine** (Thorazine), **perphenazine** (Trilafon), and **fluphenazine** (Prolixin). About 40% of patients, however, fail to respond to treatment with these medications. Neuroleptics can control most of the positive symptoms of schizophrenia as well as reduce the frequency and severity of relapses but they have little effect on negative symptoms. In addition, these medications have problematic side effects, ranging from dry mouth, blurry vision, and restlessness (akathisia) to such long-term side effects as **tardive dyskinesia** (TD). TD is a disorder characterized by involuntary movements of the mouth, lips, arms, or legs; it affects about 15%–20% of patients who have been receiving neuroleptic medications over a period of years. Discomfort related to these side effects is one reason why 40% of patients treated with the older antipsychotics do not adhere to their medication regimens.

ATYPICAL ANTIPSYCHOTICS. The atypical antipsychotics are newer medications introduced in the 1990s. They are sometimes called serotonin dopamine antagonists, or SDAs. These medications include **clozapine** (Clozaril), **risperidone** (Risperdal), **quetiapine** (Seroquel), **ziprasidone** (Geodon), and **olanzapine** (Zyprexa). These newer drugs are more effective in treating the negative symptoms of schizophrenia and have fewer side effects than the older antipsychotics. Clozapine has been reported to be effective in patients who do not respond to neuroleptics, and to reduce the risk of suicide attempts. The atypical antipsychotics, however, do have weight gain as a side effect; and patients taking clozapine must have their blood monitored periodically for signs of agranulocytosis, or a drop in the number of white blood cells. These drugs are now considered first-line treatments for patients having their first psychotic episode.

OTHER PRESCRIPTION MEDICATIONS. Patients with schizophrenia have a lifetime prevalence of 80% for major depression; others suffer from phobias or other anxiety disorders. The doctor may prescribe antidepressants or a short course of benzodiazepines along with antipsychotic medications.

Inpatient treatment

Patients with schizophrenia are usually hospitalized during acute psychotic episodes, to prevent harm to themselves or to others, and to begin treatment with antipsychotic medications. A patient having a first psychotic episode is usually given a **computed tomography** (CT) or **magnetic resonance imaging** (MRI) scan to rule out structural brain disease.

Outpatient treatment

In recent years, patients with schizophrenia who have been stabilized on antipsychotic medications have been given psychosocial therapies of various types to assist them with motivation, self-care, and forming relationships with others. In addition, because many patients have had their education or vocational training interrupted by the onset of the disorder, they may be helped by therapies directed toward improving their social functioning and work skills.

Specific outpatient treatments that have been used with patients with schizophrenia include:

- Rehabilitation programs. These programs may offer vocational counseling, job training, problem-solving and money management skills, use of public transportation, and **social skills training**.
- **Cognitive-behavioral therapy** and supportive **psychotherapy**.
- **Family psychoeducation**. This approach is intended to help family members understand the patient's illness, cope with the problems it creates for other family members, and minimize stresses that may increase the patient's risk of relapse.

- **Self-help groups**. These groups provide mutual support for family members as well as patients. They can also serve as advocacy groups for better research and treatment, and to protest social stigma and employment discrimination.

Alternative and complementary therapies

Alternative and complementary therapies that are being investigated for the treatment of schizophrenia include **gingko biloba**, an Asian shrub, and vitamin therapy. One Chinese study reported that a group of patients who had not responded to conventional antipsychotic medications benefited from a thirteen-week trial of gingko extract, with significantly fewer side effects. Vitamin therapy is recommended by naturopathic practitioners on the grounds that many hospitalized patients with schizophrenia suffer from nutritional deficiencies. The supplements recommended include folic acid, niacin, vitamin B_6, and vitamin C.

Prognosis

The prognosis for patients diagnosed with schizophrenia varies. About 20% recover their previous level of functioning, while another 10% achieve significant and lasting improvement. About 30%–35% show some improvement with intermittent relapses and some disabilities, while the remainder are severely and permanently incapacitated. Factors associated with a good prognosis include relatively good functioning prior to the first psychotic episode; a late or sudden onset of illness; female sex; treatment with antipsychotic medications shortly after onset; good **compliance** with treatment; a family history of mood disorders rather than schizophrenia; minimal cognitive impairment; and a diagnosis of paranoid or nondeficit subtype. Factors associated with a poor prognosis include early age of onset; a low level of prior functioning; delayed treatment; heavy substance abuse; noncompliance with treatment; a family history of schizophrenia; and a diagnosis of disorganized or deficit subtype with many negative symptoms.

Prevention

The multifactorial and polygenic etiology (origins or causes) of schizophrenia complicates the search for preventive measures against the disorder. It is possible that the complete mapping of the human genome will identify a finite number of genes that contribute to susceptibility to schizophrenia. The NIMH has presently compiled the world's largest registry of families affected by schizophrenia in order to pinpoint specific genes for further study. The NIMH also sponsors a Prevention Research Initiative to identify points in the development of schizophrenia at which patients could benefit from the application of preventive efforts.

See also Medication-induced movement disorders

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American Academy of Child and Adolescent Psychiatry. 3615 Wisconsin Avenue, NW, Washington, DC 20016-3007. (202) 966-7300. Fax: (202) 966-2891. <www.aacap.org>.

The National Alliance for the Mentally Ill (NAMI). 200 North Glebe Road, Suite 1015, Arlington, VA 22203-3754. (800) 950-NAMI or (703) 524-7600. <www.nami.org>.

National Alliance for Research on Schizophrenia and Depression (NARSAD). 60 Cutter Mill Road, Suite 404, Great Neck, NY 11021. (516) 829-0091. <www.mhsource.com>.

National Institute of Mental Health. 6001 Executive Boulevard, Room 8184, MSC 9663, Bethesda, MD 20892-9663. (301) 443-4513. <www.nimh.nih.gov>.

National Mental Health Association (NMHA). 1021 Prince Street, Alexandria, VA 22314-2971. (800) 969-6942 or (703) 684-7722. <www.nmha.org>.

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Schizophreniform disorder

Definition

Schizophreniform disorder (SFD) is a time-limited illness wherein the sufferer has experienced at least two of the major symptoms of **psychosis** for longer than one month but fewer than six months. **Hallucinations**, **delusions**, and strange bodily movements or lack of movements (catatonic behavior) are all symptoms that may be observed. Additionally, minimal or peculiar speech, lack of drive to act on one's own behalf, bizarre behavior, a wooden quality to one's emotions or near-absent emotionality are all typical psychotic symptoms that may occur in SFD.

Part of defining SFD involves examining possible biological influences on the development of the individual's psychotic symptoms. When the psychotic features result from a physical disease, a reaction to medication, or intoxication with drugs or alcohol, then these symptoms are not considered SFD. Also, if hallucinations,

delusions or other psychotic symptoms are experienced solely during episodes of clinical depression or mania, then SFD is not diagnosed. Instead, a mood disorder **diagnosis** is given.

The ***Diagnostic and Statistical Manual of Mental Disorders*** Fourth Edition Text Revision, or *DSM-IV-TR*, produced by the American Psychiatric Association in 2000, outlines the diagnostic criteria for SFD.

Description

The person experiencing SFD shows at least two psychotic symptoms, which may be either "positive" or "negative" psychotic symptoms. The terms "positive" and "negative" are not used in their usual meanings of positive being good and negative being bad. In discussing psychosis, positive and negative are used with a more formal medical connotation. Medically, "positive" refers to a factor being present that does not normally occur, or to an excess of some factor or behavior. **Positive symptoms** of psychosis include hallucinations, delusions, strange bodily movements or frozen movement (catatonic behavior), peculiar speech and bizarre or primitive (socially inappropriate) behavior. Negative, when used in medical fashion, refers to an absence or deficiency of a factor that is usually at a reasonable level during normal functioning. Various deficiencies in behavior, emotionality or speech constitute the **negative symptoms** of psychosis which are observed in some cases of SFD. Negative symptoms of psychosis include *avolition*, *affective flattening* and *alogia*.

Avolition is a lack of effort to act on one's own behalf or to engage in behaviors directed at accomplishing a purpose. Affective flattening or blunted **affect** refers to a decrease or low level of emotion, shown as a wooden quality to one's emotions or a near absence of emotionality. Alogia derives from the Greek root term for speech or thought, and the "a" that begins the word indicates an absence. Thus, alogia refers to a disruption in thought process reflected in the person's speech. One form of alogia is *poverty of speech*. Impoverished speech is brief, limited, terse and generally emerges only in response to questions or prompts rather than flowing spontaneously. An impairment termed *poverty of content* occurs when the information or concepts that the individual is attempting to convey cannot be understood because of limitations in the method of communicating. The meaning behind the phrases is obscured or missing. Typically, in poverty of content, the person's speech, while comprehensible in terms of its orderliness of grammar and vocabulary, does not convey substantial meaning because the phrasing is overly concrete and literal or overly abstract and fanciful.

KEY TERMS

Erotomanic delusions—Erotomanic delusions involve the mistaken conviction that someone is in love with the delusional person. Often, the love object is a public figure of some prominence, such as an actress, rock star, or political figure. David Letterman and Jodie Foster are celebrities who have both been victimized by persons with erotomanic delusions.

Grandiose delusions—Grandiose delusions magnify the person's importance; the delusional person may believe himself or herself to be a famous person, to have magical superpowers, or to be someone in a position of enormous power (such as being the King or President).

Hallucinations—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Somatic—Somatic comes from *soma*, the Greek word for body; thus, somatic hallucinations are bodily experienced hallucinations. Somatic delusions are strongly held but erroneous ideas about the characteristics or functioning of one's body. An example is a mental health client who refuses to eat because of a belief that there is a hole in the stomach that will spill anything consumed into the body cavity, when such is not actually the case.

Among the various positive symptoms of psychosis that can be a part of SFD, delusions are a fairly common psychotic feature. Delusions are strongly held irrational and unrealistic beliefs that are highly resistant to alteration. Even when the person encounters evidence that would invalidate the delusion, the unjustified and improbable belief remains a conviction. Often, delusions are paranoid or persecutory in tone. In these types of delusions, the person is excessively suspicious and continually feels at the mercy of conspirators believed to be determined to cause harm to the sufferer. However, delusions can also take on other overtones. Some delusions are grandiose, or involve elaborate

love fantasies (erotomanic delusions). Delusions may involve somatic content, or may revolve around extreme and irrational jealousy.

Peculiar or disorganized speech, catatonic behavior and bizarre or primitive behavior are all additional positive psychotic symptoms that may occur in SFD. Disorganized speech is seen in some cases of SFD. Speech disorganization can involve words blended together into incomprehensible statements, also known as "word salad." In some persons disorganized speech takes the form of echolalia, which is the repetition of another person's exact spoken words, restated either immediately after the initial speaker or after a delay of minutes to hours. Catatonic behavior or **catatonia** involves the presence of one of the possible extremes related to movement. Catalepsy is the motionless end of the catatonic spectrum; in catalepsy, a person may remain unmoving in one fixed position for long periods. The opposite end of the catatonia phenomenon is demonstrated in rapid or persistently repeated movements, recurrent grimacing and odd facial expressions, and contorted or strange gestures. Bizarre or primitive behavior in SFD ranges from child-like behaviors in unsuitable circumstances to unusual practices such as hoarding refuse items perceived by the sufferer to be valuable, caching food all over the home, or wandering purposelessly through the streets.

Only rarely would all these various psychotic symptoms be observed simultaneously in one person with SFD. Instead, each individual with SFD has a constellation of symptoms, practices and thought processes that is unique to that person. However, frequent occurrence of at least two of these psychotic symptoms persisting for one month to six months is considered to be SFD. A different diagnosis, which includes the presence of psychotic symptoms, is given if the symptoms have been present for longer than six months. Also, if there is some other psychiatric syndrome that better explains the behaviors, or if there are biological causes (such as a physical illness, like a **brain** tumor) that caused the symptoms to appear, another diagnosis is utilized.

Unlike any other diagnoses offered in *DSM-IV-TR*, the SFD diagnosis always includes an indication of the patient's *prognosis*. Prognosis refers to the potential outcome for an individual with a particular illness, based on the features already observed and the usual course of the illness. If an individual with SFD has several positive prognostic factors, then there is a much higher likelihood of complete recovery without relapse into psychosis. Positive prognostic factors in SFD include: prominent confusion during the illness, rapid (rather than gradual) development of symptoms during a four-week period, good previous interpersonal and goal-oriented functioning, and lack of negative symptoms of psychosis.

Causes and symptoms

Causes

Several views regarding the causes of the disorder have been put forth by researchers and clinicians.

AN EARLY PHASE OF ANOTHER PSYCHIATRIC DISORDER. A number of follow-up studies have examined the relationship between SFD and other disorders such as **schizophrenia**, **schizoaffective disorder** and **bipolar disorder**. The majority of these studies have found that between 50% and 75% of persons with SFD eventually develop schizophrenia. Of those persons with a history of SFD who do not subsequently receive a schizophrenia diagnosis, only a small portion have no further psychiatric disturbance. The other diagnoses that may be observed in persons formerly diagnosed with SFD are schizoaffective disorder or bipolar disorder (the Type I form). The most common subsequent diagnosis is schizophrenia, with the next most common being schizoaffective disorder. Because of the high rate of later schizophrenia in SFD sufferers, many clinicians have come to think of SFD as being an initial phase of schizophrenia. It is impossible to identify, during an episode of SFD, whether any one particular case will improve without any relapse into psychotic symptoms, or if the mental health client is actually in the early phase of schizophrenia or schizoaffective disorder. Follow-up studies indicate that being frequently confused during a period of SFD is often associated with gradual complete recovery.

LENGTHY POSTPARTUM PSYCHOSIS. Intense hormonal changes occurring in childbirth and immediately afterward can result in a short-term psychotic disorder often referred to as *postpartum psychosis*. When the psychotic symptoms in this condition persist for longer than one month but fewer than six months, the SFD diagnosis may be given.

DIATHESIS X STRESS. *Diathesis* is a medical term meaning that some element of one's physiology makes one particularly prone to develop an illness if exposed to the right conditions. Diathesis is another way of saying there is a personal predisposition to develop a disorder; the predisposition is biologically based and is genetically acquired (inherited in the person's genes). Temporary psychotic reactions may occur in persons who have the diathesis for psychosis, when the individual is placed under marked **stress**. The stress may result from typical life transition experiences such as moving away from home the first time, being widowed or getting divorced. In some cases, the stressor is more intense or unusual, such as surviving a natural disaster, wartime service, being taken hostage or surviving a terrorist attack. When the psychotic responses last less than a month, then this reaction is labeled "**brief psychotic disorder**." Highly

susceptible persons may show psychotic symptoms for greater than one month and might be given the SFD diagnosis. If the psychotic symptoms are purely reactive, when the stressor ceases or more support is available, the individual is likely to return to a non-psychotic mode of functioning. In persons with a strong diathesis or predisposition, the initial psychotic reaction may "tip over" from the category of a brief reaction into a longer-term, persistent psychiatric disorder. The diathesis x stress model is applied not only to SFD, but also to schizophrenia, schizoaffective disorder and the most severe forms of mood disorders.

CULTURALLY DEFINED DISORDERS. Many cultures have forms of mental disorder, unique to that culture, that would meet criteria for SFD. In culturally defined disorders, a consistent set of features and presumed causes of the syndrome are localized to that community. Such disorders are termed "culture-bound." Examples of culture-bound syndromes that might meet SFD criteria are *amok* (Malaysia), or *locura* (Latino Americans). Amok is a syndrome characterized by brooding, persecutory delusions and aggressive actions. Locura involves incoherence, agitation, social dysfunction, erratic behavior, and hallucinations.

Symptoms

DSM-IV-TR provides three major criteria for SFD:

AT LEAST TWO PERSISTENT POSITIVE OR NEGATIVE SYMPTOMS OF PSYCHOSIS.

- delusions
- disorganized speech which is strange, peculiar, difficult to comprehend
- disorganized (bizarre or child-like) behavior
- catatonic behavior
- hallucinations
- negative symptoms (affective flattening, alogia, avolition)

LIMITED DURATION.

- The psychotic symptoms have occurred for at least one month but less than six months.

CAUSE. The symptoms cannot:

- occur as part of a mood disorder
- occur as part of schizoaffective disorder or schizophrenia
- be due to intoxication with drugs or alcohol
- be an adverse reaction to a medication
- be caused by a physical injury or medical illness

Demographics

The actual rate of SFD is unknown, mainly because SFD is difficult to measure except in retrospect. In the first few weeks of symptoms, SFD cannot be differentiated from brief psychotic disorder. Once the symptoms persist past one month and are identified as SFD, six months or more must pass before one can determine if a mental health consumer had "classic" SFD or was in the early phase of a more chronic mental disorder. Given that a majority of SFD sufferers go on to be diagnosed with schizophrenia, the best inferences about demographics and gender differences in SFD would be drawn from similar information available regarding schizophrenia.

Diagnosis

Despite the clarity of the *DSM-IV-TR* criteria, identification of SFD is less than clear-cut. The emphasis on the length of time that symptoms have been evident and the presence or absence of good prognostic factors make SFD one of the most unusually defined of the *DSM-IV-TR* disorders. While duration of symptoms is the major distinction among brief psychotic disorder, SFD and schizophrenia, it can be difficult to clearly determine the length of time symptoms have existed. An additional complication is that the cultural context in which the "psychotic symptoms" are experienced determines whether the behaviors are viewed as pathological or acceptable. When psychotic-like behaviors are expected to occur normally as part of the person's culture or religion, and when the behaviors occur in a culturally positive context such as a religious service, SFD would not be diagnosed.

Information about current and past experiences is collected in an interview with the client, and possibly in discussion with the client's family. Psychological assessment instruments, such as the **Rorschach technique**, the **Minnesota Multiphasic Personality Inventory**, and mood disorder questionnaires or structured diagnostic interviews may also be used to aid in the diagnosis.

Treatments

The main line of treatment for SFD is antipsychotic medication. These medications are often very effective in treating SFD. Mood-stabilizing drugs similar to those used in bipolar disorder may be used if there is little response to other interventions. Postpartum psychosis is also treated with antipsychotics and possibly, hormones. Supportive therapy and education about mental illness is often valuable. The most useful interventions in culture-bound syndromes are those that are societally prescribed; for example, a sacred ceremony to ease the rest-

less spirits of deceased ancestors might be a usual method of ending the psychotic-like state, in that particular culture.

Prognosis

Given the large number of mental health consumers with SFD who go on to be diagnosed with a more chronic form of mental illness, the prognosis is fairly poor. As noted earlier, prominent confusion during the illness, rapid (rather than gradual) development of symptoms during a four-week period, good previous interpersonal and goal-oriented functioning and lack of negative symptoms of psychosis suggest a better outcome.

Prevention

If the SFD is a persistent postpartum psychosis, a prevention option is to avoid having additional children. The physician may anticipate the postpartum problem and prescribe an antipsychotic medication regimen to begin immediately after delivery as a preventive measure. Although prevention of psychotic disorders is difficult to accomplish, the earlier treatment begins, the better the outcome. Therefore, efforts are more generally focused on early identification of SFD and other psychotic-spectrum disorders.

See also Delusional disorder; Dementia; Schizotypal personality disorder

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Schizotypal personality disorder

Definition

Schizotypal personality disorder is characterized by an ongoing pattern in which the affected person distances him- or herself from social and interpersonal relationships. Affected people typically have an acute discomfort when put in circumstances where they must relate to others. These individuals are also prone to cognitive and perceptual distortions and a display a variety of eccentric behaviors that others often find confusing.

Description

People with schizotypal personality disorder are more comfortable turning inward, away from others, than learning to have meaningful interpersonal relationships. This preferred isolation contributes to distorted perceptions about how interpersonal relationships are supposed to happen. These individuals remain on the periphery of life and often drift from one aimless activity to another with few, if any, meaningful relationships.

A person with schizotypal personality disorder has odd behaviors and thoughts that would typically be viewed by others as eccentric, erratic, and bizarre. They are known on occasion to have brief periods of psychotic episodes. Their speech, while coherent, is marked by a focus on trivial detail. Thought processes of schizotypals include magical thinking, suspiciousness, and illusions. These thought patterns are believed to be the schizotypal's unconscious way of coping with social anxiety. To some extent, these behaviors stem from being socially isolated and having a distorted view of appropriate interpersonal relations.

Causes and symptoms

Causes

Schizotypal personality disorder is believed to stem from the affected person's original family, or family of origin. Usually the parents of the affected person were emotionally distant, formal, and displayed confusing parental communication. This **modeling** of remote, unaffectionate relationships is then reenacted in the social relationships encountered in the developing years. The social development of people with schizotypal personality disorder shows that many were also regularly humiliated by their parents, siblings, and peers resulting in significant relational mistrust. Many display low self-esteem, self-criticism and self-deprecating behavior. This further contributes to a sense that they are socially incapable of having meaningful interpersonal relationships.

Symptoms

The *Diagnostic and Statistical Manual of Mental Disorders*, a professional manual, specifies nine diagnostic criteria for schizotypal personality disorder:

- Incorrect interpretations of events. Individuals with schizotypal personality disorder often have difficulty seeing the correct cause and effect of situations and how they affect others. For instance, the schizotypal may misread a simple non-verbal communication cue, such as a frown, as someone being displeased with them, when in reality it may have nothing to do with them. Their perceptions are often distortions of what is really happening externally, but they tend to believe their perceptions more than what others might say or do.
- Odd beliefs or magical thinking. These individuals may be superstitious or preoccupied with the paranormal. They often engage in these behaviors as a desperate means to find some emotional connection with the world they live in. This behavior is seen as a coping mechanism to add meaning in a world devoid of much meaning because of the social isolation these individuals experience.
- Unusual perceptual experiences. These might include having illusions, or attributing a particular event to some mysterious force or person who is not present. Affected people may also feel they have special powers to influence events or predict an event before it happens.
- Odd thinking and speech. People with schizotypal personality disorder may have speech patterns that appear strange in their structure and phrasing. Their ideas are often loosely associated, prone to tangents, or vague in description. Some may verbalize responses by being overly concrete or abstract and insert words that serve to confuse rather than clarify a particular situation, yet make sense to them. They are typically unable to have ongoing conversation and tend to talk only about matters that need immediate attention.
- Suspicious or paranoid thoughts. Individuals with schizotypal personality disorder are often suspicious of others and display paranoid tendencies.
- Emotionally inexpressive. Their general social demeanor is to appear aloof and isolated, behaving in a way that communicates they derive little joy from life. Most have an intense fear of being humiliated or rejected, yet repress most of these feelings for protective reasons.
- Eccentric behavior. People with schizotypal personality disorder are often viewed as odd or eccentric due to their unusual mannerisms or unconventional clothing choices. Their personal appearance may look unkempt—clothing choices that do not "fit together," clothes may be too small or large, or clothes may be noticeably unclean.

KEY TERMS

Millon Clinical Multiaxial Inventory (MCMI-II)—A self-report instrument designed to help the clinician assess DSM-IV-related personality disorders and clinical syndromes. It provides insight into 14 personality disorders and 10 clinical syndromes.

Minnesota Multiphasic Personality Inventory (MMPI-2)—A comprehensive assessment tool widely used to diagnose personality disorders.

Rorschach Psychodiagnostic Test—This series of 10 "ink blot" images allows the patient to project their interpretations which can be used to diagnose particular disorders.

Thematic Apperception Test (TAT)—A projective test using stories and descriptions of pictures to reveal some of the dominant drives, emotions, sentiments, conflicts, and complexes of a personality.

- Lack of close friends. Because they lack the skills and confidence to develop meaningful interpersonal relationships, they prefer privacy and isolation. As they withdraw from relationships, they increasingly turn inward to avoid possible social rejection or ridicule. If they do have any ongoing social contact, it is usually restricted to immediate family members.
- Socially anxious. Schizotypals are noticeably anxious in social situations, especially with those they are not familiar with. They can interact with people when necessary, but prefer to avoid as much interaction as possible because their self-perception is that they do not "fit in." Even when exposed to the same group of people over time, their social anxiety does not seem to lessen. In fact, it may progress into distorted perceptions of **paranoia** involving the people with whom they are in social contact.

Demographics

Schizotypal personality disorder appears to occur more frequently in individuals who have an immediate family member with **schizophrenia**. The prevalence of schizotypal personality disorder is approximately 3% of the general population and is believed to occur slightly more often in males.

Symptoms that characterize a typical **diagnosis** of schizotypal personality disorder should be evaluated in the context of the individual's cultural situation, particularly those regarding superstitious or religious beliefs and practices. (Some behaviors that Western cultures may

view as psychotic are viewed within the range of normal behavior in other cultures.)

Diagnosis

The symptoms of schizotypal personality disorder may begin in childhood or adolescence showing as a tendency toward solitary pursuit of activities, poor peer relationships, pronounced social anxiety, and underachievement in school. Other symptoms that may be present during the developmental years are hypersensitivity to criticism or correction, unusual use of language, odd thoughts, or bizarre fantasies. Children with these tendencies appear socially out-of-step with peers and often become the object of malicious teasing by their peers, which increases the feelings of isolation and social ineptness they feel. For a diagnosis of schizotypal personality disorder to be accurately made, there must also be the presence of at least four of the above-mentioned symptoms.

The symptoms of schizotypal personality disorder can sometimes be confused with the symptoms seen in schizophrenia. The bizarre thinking associated with schizotypal personality disorder can be perceived as a psychotic episode and misdiagnosed. While brief psychotic episodes can occur in the patient with schizotypal personality disorder, the **psychosis** is not as pronounced, frequent, or as intense as in schizophrenia. For an accurate diagnosis of schizotypal personality disorder, the symptoms for schizotypal cannot occur exclusively during the course of schizophrenia or other mood disorder that has psychotic features.

Another common difficulty in diagnosing schizotypal personality disorder is distinguishing it from other the schizoid, avoidant, and paranoid **personality disorders**. Some researchers believe that schizotypal personality disorder is essentially the same disorder as schizoid, but many feel there are distinguishing characteristics. Schizoids are deficient in their ability to experience emotion, while schizotypals are more pronounced in their inability to understand human motivation and communication. While **avoidant personality disorder** has many of the same symptoms as schizotypal personality disorder, the distinguishing symptom in schizotypal is the presence of behavior that is noticeably eccentric. The schizotypal differs from the paranoid by tangential thinking and eccentric behavior.

The diagnosis of schizotypal personality disorder is based on a clinical interview to assess symptomatic behavior. Other assessment tools helpful in confirming the diagnosis of schizotypal personality disorder include:

- **Minnesota Multiphasic Personality Inventory** (MMPI-2)
- Millon Clinical Multiaxial Inventory (MCMI-II)
- Rorschach Psychodiagnostic Test
- **Thematic Apperception Test** (TAT)

Treatments

The patient with schizotypal personality disorder finds it difficult to engage and remain in treatment. For those higher-functioning individuals who seek treatment, the goal will be to help them function more effectively in relationships rather than restructuring their personality.

Psychodynamically oriented therapies

A psychodynamic approach would typically seek to build a therapeutically trusting relationship that attempts to counter the mistrust most people with this disorder intrinsically hold. The hope is that some degree of attachment in a therapeutic relationship could be generalized to other relationships. Offering interpretations about the patient's behavior will not typically be helpful. More highly functioning schizotypals who have some capacity for empathy and emotional warmth tend to have better outcomes in psychodynamic approaches to treatment.

Cognitive-behavioral therapy

Cognitive approaches will most likely focus on attempting to identify and alter the content of the schizotypal's thoughts. Distortions that occur in both perception and thought processes would be addressed. An important foundation for this work would be the establishment of a trusting therapeutic relationship. This would relax some of the social anxiety felt in most interpersonal relationships and allow for some exploration of the thought processes. Constructive ways of accomplishing this might include communication skills training, the use of videotape feedback to help the affected person perceive his or her behavior and appearance objectively, and practical suggestions about personal hygiene, employment, among others.

Interpersonal therapy

Treatment using an interpersonal approach would allow the individual with schizotypal personality disorder to remain relationally distant while he or she "warms up" to the therapist. Gradually the therapist would hope to engage the patient after becoming "safe" through lack of coercion. The goal would be to develop trust in order to help the patient gain insight into the distorted and magical thinking that dominates. New self-talk can be introduced

to help orient the individual to reality-based experience. The therapist can mirror this objectivity to the patient.

Group therapy

Group therapy may provide the patient with a socializing experience that exposes them to feedback from others in a safe, controlled environment. It is typically recommended only for schizotypals who do not display severe eccentric or paranoid behavior. Most group members would be uncomfortable with these behavioral displays and it would likely prove destructive to the group dynamic.

Family and marital therapy

It is unlikely that a person with **schizoid personality disorder** will seek family or marital therapy. Many schizoid types do not marry and end up living with and being dependent upon first-degree family members. If they do marry they often have problems centered on insensitivity to their partner's feelings or behavior. Marital therapy (**couples therapy**) may focus on helping the couple to become more involved in each other's lives or improve communication patterns.

Medications

There is considerable research on the use of medications for the treatment of schizotypal personality disorder due to its close symptomatic relationship with schizophrenia. Among the most helpful medications are the antipsychotics that have been shown to control symptoms such as illusions and phobic anxiety, among others. **Amoxapine** (trade name Asendin), is a tricyclic antidepressant with antipsychotic properties, and has been effective in improving schizophrenic-like and depressive symptoms in schizotypal patients. Other antidepressants such as **fluoxetine** (Prozac) have also been used successfully to reduce symptoms of anxiety, paranoid thinking, and depression.

Prognosis

The prognosis for the individual with schizotypal personality disorder is poor due to the ingrained nature of the coping mechanisms already in place. Schizotypals who depend heavily on family members or others are likely to regress into a state of **apathy** and further isolation. While some measurable gains can be made with mildly affected individuals, most are not able to alter their ingrained ways of perceiving or interpreting reality. When combined with poor social support structure, most will not enter any type of treatment.

Prevention

Since schizotypal personality disorder originates in the patient's family of origin, the only known preventative measure is a nurturing, emotionally stimulating and expressive caretaking environment.

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Seasonal affective disorder

Definition

Seasonal affective disorder, often abbreviated as SAD, is a type of mood disorder that follows an annual pattern consistent with the seasons. The most common course for SAD includes an onset of depressive symptoms late in the fall, continuation of symptoms throughout winter, and remission of symptoms in the spring.

Description

According to the handbook used by mental health professionals to diagnose mental disorders, the ***Diagnostic and Statistical Manual of Mental Disorders*** fourth edition text revised, or *DSM-IV-TR*, SAD is not considered a disorder or syndrome on its

own. Instead, SAD is considered a pattern specifier, or subtype, of another mood disorder **diagnosis**. For example, an individual may be diagnosed as having a major depressive episode with a seasonal pattern.

The most common type of seasonal pattern is one in which an individual first experiences symptoms in the late fall, has continued and heightened symptoms in winter, and then experiences a remission of symptoms in the spring. However, other patterns are possible. For example, a person may become depressed in the summer and then become less depressed when the weather becomes colder.

Causes and symptoms

Causes

Lack of sunlight, normally associated with winter, is considered to be the primary cause of SAD. Although winter temperature may also have an impact, especially in colder areas, the lack of light is most important. This is supported by the effectiveness of therapy in which individuals are exposed to high-intensity light (**light therapy**). The causes of rarer types of seasonal symptoms, such as those experienced by individuals who become depressed in summer, are more difficult to determine.

Symptoms

The symptoms experienced by people with SAD are similar to some of those experienced by depressed people in general: change in appetite, weight gain or loss, **fatigue**, reduced energy, irritability, and avoidance of social situations. To meet the diagnostic criteria for the disorder as indicated in the *DSM-IV-TR,* these symptoms must be present during the season the individual is depressed and must lessen or abate when that season is over.

Demographics

Some studies have shown that up to 6% of people experience some depressive symptoms in winter. SAD is a more common phenomenon in women than men. According to the *DSM-IV-TR*, women make up 60–90% of people with the seasonal pattern of depression. SAD primarily affects adults, although it is possible for children and adolescents to suffer from it. Research indicates that SAD is much more common in countries and regions where there are distinct seasonal changes. In countries near the equator, where changes in climate and light are mild, SAD generally does not occur.

Diagnosis

SAD is diagnosed through a clinical interview with the patient and careful history-taking by the physician. For the seasonal pattern specifier to be applied to a *DSM-IV-TR* mood disorder diagnosis, the following criteria must be met: there is a relationship between the onset of the depressive episode and a particular time of year; the depressive symptoms are in remission at a particular time of year; the onset and remissions have occurred at these times for the past two years; and seasonal depressive episodes outnumber non-seasonal depressive episodes over the person's lifetime. Also, the seasonal pattern specifier must not be given when depressive symptoms are due to seasonally linked stressors, such as the beginning of school or an employment schedule.

An individual with seasonal depression must be distinguished from one with who has depressive symptoms all year long.

Treatments

Light therapy, in which the person experiencing SAD is exposed to high-intensity light, is often used—usually for one to two hours per day. Sometimes, briefer periods of exposure to higher-intensity light can be used. The exposure to light may be facilitated through the use of a box which emits the prescribed light or through the use of a light visor the patient wears on his or her head. Tanning beds should not be used for light therapy. Light therapy has been found to be the most effective treatment for people correctly diagnosed with seasonal symptoms in the winter. It does not appear to have serious side effects.

Prognosis

Light therapy is considered to be a safe and effective treatment. However, it is time consuming and people do not always stay on the prescribed course of treatment. Also, SAD can be a persistent problem; even if light therapy is effective one year, symptoms may return the following year.

See also Bipolar disorders; Depressive disorders

Resources

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ORGANIZATIONS

Depression and Related Affective Disorders Association. 600 N. Wolfe St., Baltimore, MD, 21287. <http://www.drada.org>.

Seasonal Affective Disorder Association. PO Box 989, Steyning BN44 3HG, England. <http://www.sada.org.uk>.

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KEY TERMS

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Tolerance—Progressive decrease in the effectiveness of a drug with long-term use.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

Sedatives and related disorders

Definition

Sedatives are compounds that cause physiological and mental slowing of the body. They have many legitimate medical uses. However, people who use them improperly may develop symptoms of abuse, dependence, and withdrawal. Several other classes of compounds, including sleep-promoting drugs (hypnotics) and some anti-anxiety (anxiolytic) drugs produce effects and disorders similar to those of sedatives. Sedatives are often referred to as tranquilizers, and the similar classes of sedatives and hypnotics are sometimes thought of as one group: the sedative-hypnotics.

Description

Sedatives and similar drugs are available by prescription and have many medical uses. They are used in conjunction with surgery and are prescribed to treat pain, anxiety, panic attacks, **insomnia**, and in some cases, convulsions. Most people who take prescription sedatives take them responsibly and benefit from their use. Some people misuse these drugs. They may do so unintentionally by increasing their prescribed dose without medical advice. Intentional abusers buy these drugs off the street for recreational use or get them from friends or family members who have prescriptions. Sedatives are not popular street drugs, and when they are used recreationally, it is usually in conjunction with other illicit drugs or alcohol. When taken exactly as prescribed, sedatives rarely create major health risks.

A chemically diverse group of drugs are discussed together in this entry because they all appear to work in the body the same way and produce similar problems of abuse, dependence, intoxication, and withdrawal. These drugs work in the **brain** by increasing the amount of the neurotransmitter gamma-aminobutyric acid (GABA). **Neurotransmitters** help to regulate the speed at which nerve impulses travel. When the amount of GABA increases, the speed of nerve transmissions decreases. Thus these drugs depress the nervous system and cause reduced pain, sleepiness, reduced anxiety, and muscle relaxation.

The most widely prescribed and best-studied sedatives belong to a group called benzodiazepines. Prescription benzodiazepines and their relatives include **alprazolam** (Xanax), **chlordiazepoxide** (Librium), **clonazepam** (Klonopin), **clorazepate** (Tranxene), **diazepam** (Valium), **estazolam** (ProSom), **flurazepam** (Dalmane), halazepam (Paxipam), **lorazepam**, (Ativan), **oxazepam** (Serax), prazepam (Centrax), **quazepam** (Doral), **temazepam** (Restoril), **triazolam** (Halcion). Other drugs that act in a similar manner include the **barbiturates** amobarbital (Amytal), aprobarbital (Alurate), butabarbital (Butisol), phenobarbitol, (Nebutal), and secobarbital, (Seconal). In addition, **chloral hydrate** (Notec), ethchlorvynol (Placidyl), glutehimide (Doriden), meprobamate (Miltown, Equanil, Equagesic, Deprol) and **zolpidem** (Ambien) have similar actions.

Causes and symptoms

Sedatives and other drugs in this class are physically and sometimes psychologically addicting. People taking sedatives rapidly develop tolerance for the drugs. Tolerance occurs when a larger and larger dose must be taken to produce the same effect. Because sedatives are physically addicting, people with sedative dependence experience physical withdrawal symptoms when these drugs are discontinued.

Sedative abuse occurs when people misuse these drugs but are not addicted to them. Many people who abuse sedatives also use other illicit drugs. They may use sedatives to come down off a cocaine high or to enhance the effect of **methadone**, a heroin substitute.

Sedative dependence occurs when there is a physical **addiction**, when a person actively seeks sedatives

(for example, by going to several doctors and getting multiple prescriptions) and when a person continues to use these drugs despite the fact that they cause interpersonal problems and difficulties meeting the responsibilities of daily life.

Sedative intoxication

Sedative intoxication occurs when a person has recently used one of these drugs and shows certain psychosocial symptoms such as hostility or aggression, swings in mood, poor judgment, inability to function in social settings or at work, or inappropriate sexual behavior. Because sedatives depress the central nervous system, physical symptoms include slurred speech, lack of coordination, inattention, impaired memory or "blackouts" and extreme sluggishness, stupor, or coma. Sedative intoxication can appear very similar to alcohol intoxication in its symptoms. Overdoses can be fatal.

Sedative withdrawal

Physical addiction is the main problem with sedative dependence. Sedative withdrawal is similar to alcohol withdrawal. Symptoms of sedative withdrawal are almost the reverse of the symptoms of sedative intoxication. They include:

- increased heart rate
- faster breathing
- elevated blood pressure
- increased body temperature
- sweating
- shaky hands
- inability to sleep
- anxiety
- nausea
- restlessness

About one-quarter of people undergoing sedative withdrawal have **seizures**. If withdrawal is severe, they may also have visual or auditory **hallucinations** (sedative withdrawal **delirium**). Often people who experience these more severe symptoms are using other drugs and not just sedatives.

The timeframe for withdrawal symptoms to appear varies depending on the chemical structure of the drug being taken. Withdrawal symptoms can occur hours or days after stopping use. For example, people withdrawing from Valium may not develop withdrawal symptoms for a week, and may not have peak symptoms until the second week. Low-level symptoms may linger even longer. Generally the longer a person takes a drug and the higher the dose, the more severe the withdrawal symptoms. It is possible to have withdrawal symptoms when a therapeutically prescribed dose is taken for a long time.

Sedative dependence is thought to be able to induce other mental health disorders, although there is some disagreement in the mental health community about how these disorders are defined and classified. Other disorders that may result from sedative dependence and withdrawal include:

- sedative-induced persisting dementia
- sedative-induced persisting amnestic disorder
- sedative-induced psychotic disorder (with or without hallucinations)
- sedative-induced mood disorder
- sedative-induced anxiety disorder
- sedative-induced sexually dysfunction
- sedative-induced sleep disorder

Demographics

Many people, including about 90% of those who are hospitalized, are given some type of prescription sedative. Of the people who use sedatives, only a few become dependent. People who become dependent usually fall into three categories. Some are drug addicts who use sedatives along with other street drugs. These are usually young people between the ages of 15 and 25. Others are alcoholics who use sedatives to treat chronic anxiety or sleep problems associated with their alcohol dependence. Still others use sedatives under the direction of a doctor to treat long-term pain, anxiety, or sleeplessness. These people may become dependent by increasing the amount of sedative they take as tolerance develops without telling their doctor.

Sedative abuse is not a major addiction problem with street drug users. Many people who are dependent on sedatives are middle-age and middle-class people who start taking the drug for a legitimate medical reason. Women may be more at risk than men for developing sedative dependence. Sedative dependence is the most common type of drug addiction among the elderly. Older people do not clear the drug from their bodies as efficiently as younger people, and thus may become dependent on lower, therapeutic doses.

Diagnosis

Diagnosis of sedative intoxication is made based on recent use of the drug, presence of the symptoms listed above, and presence of the drug in a blood or urine sample. Without a blood or urine test, sedative intoxication

can be difficult to distinguish from alcohol intoxication except for the absence of the odor of alcohol. People experiencing sedative intoxication usually remain grounded in reality. However, if they lose touch with reality they may be diagnosed as having sedative intoxication delirium.

Diagnosis of sedative withdrawal is based on the symptoms listed above. It can be difficult to distinguish from alcohol withdrawal. Withdrawal may occur with or without hallucinations and delirium. Diagnosis depends on whether a person remains grounded in reality during withdrawal.

Diagnosis of other mental disorders induced by sedative dependence requires that the symptoms be in excess of those usually found with sedative intoxication or withdrawal. They cannot be accounted for by other substance abuse or another mental or physical disorder.

Treatments

Treatment depends on how large a dose of sedative the patient is taking, the length of time it has been used, and the patient's individual psychological and physical state.

Physiological treatment

Successful treatment of sedative dependence is based on the idea of gradually decreasing the amount of drug the patient uses in order to keep withdrawal symptoms to a manageable level. This is called a drug taper. The rate of taper depends on the dependency dose of the drug, the length of time the drug has been taken, a person's individual mental and physical response to drug withdrawal, and any complicating factors such as other substance abuse or other physical or mental illness.

For people dependent on a low dose of sedatives, the current level of use is determined, then the amount of drug is then reduced by 10 to 25 percent. If withdrawal symptoms are manageable, reduction is continued on a weekly basis. If withdrawal symptoms are too severe, the patient is stabilized at the lowest dose with manageable symptoms until tapering can be re-started. This gradual reduction of use may take weeks, and the rate must be adjusted to the response of each patient individually.

People dependent on high doses of sedatives are usually hospitalized because of the possibility of life-threatening withdrawal symptoms. A blood or urine test is used to determine the current level of usage. The patient is often switched to an equivalent dose of a different sedative or phenobarbitol (a barbiturate) to aid in withdrawal while controlling withdrawal symptoms. The tapering process begins, but more gradually than with low-dose dependency. Often other drugs are given to combat some of the withdrawal symptoms.

Psychological treatment

Cognitive-behavioral therapy may be used in conjunction with drug tapering. This type of **talk therapy** aims at two things: to educate patients to recognize and cope with the symptoms of anxiety associated with withdrawal, and to help patients change their behavior in ways that promote coping with **stress**. Patients are taught to mentally talk their way through their anxiety and stress. Some people find **support groups** and journal keeping to be helpful in their recovery. Recovering from dependency is a slow process, best achieved when a person has a good social support system, patience, and persistence.

Prognosis

The people who have the best chance of becoming sedative-free are those who became dependent through taking long-term therapeutic doses. Although stopping any addiction takes time and work, with a properly managed course of treatment, chances of success are good.

People who abuse multiple street drugs must receive treatment for their multiple drug dependencies. Sedative abuse is low on their list of problems, and the chances of their becoming drug-free are low. Alcoholics also have a difficult time withdrawing from sedatives.

Prevention

The best way to prevent sedative-related disorders is to take these drugs only for the exact length of time and in the exact amount prescribed by a doctor.

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ORGANIZATIONS

National Clearinghouse for Alcohol and Drug Information. P. O. Box 2345, Rockville, MD 20852. (800) 729-6686. <http://www.health.org>.

National Institute on Drug Abuse. 5600 Fishers Lane, Room 10 A-39, Rockville, MD 20857. 1-888-644-6432<http://niad.nih.gov>.

OTHER

Benzodiazepine Recovery. <www.benzodiazepine.org>. This site offers chat and support groups for people recovering from sedative dependence and has links to many sources of information on these drugs.

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Seizures

Definition

A seizure is a sudden change in behavior characterized by changes in sensory perception (sense of feeling) or motor activity (movement) due to an abnormal firing of nerve cells in the **brain**. Epilepsy is a condition characterized by recurrent seizures that may include repetitive muscle jerking called convulsions.

Description

Seizure disorders and their classification date back to the earliest medical literature accounts in history. In 1964, the Commission on Classification and Terminology of the International League Against Epilepsy (ILAE) devised the first official classification of seizures, which was revised again in 1981. This classification is accepted worldwide and is based on electroencephalographic (EEG) studies. Based on this system, seizures can be classified as either focal or generalized. Each of these categories can also be further subdivided.

Focal seizures

A focal (partial) seizure develops when a limited, confined population of nerve cells fire their impulses abnormally on one hemisphere of the brain. (The brain has two portions or cerebral hemispheres—the right and left hemispheres.) Focal seizures are divided into simple or complex based on the level of consciousness (wakefulness) during an attack. Simple partial seizures occur in patients who are conscious, whereas complex partial seizures demonstrate impaired levels of consciousness.

Generalized seizures

A generalized seizure results from initial abnormal firing of brain nerve cells throughout both left and right hemispheres. Generalized seizures can be classified as follows:

- Tonic-clonic seizures: This is the most common type among all age groups and is categorized into several phases beginning with vague symptoms hours or days before an attack. These seizures are sometimes called grand mal seizures.
- Tonic seizures: These are typically characterized by a sustained nonvibratory contraction of muscles in the legs and arms. Consciousness is also impaired during these episodes.
- Atonic seizures (also called “drop attacks”): These are characterized by sudden, limp posture and a brief period of unconsciousness and last for one to two seconds.
- Clonic seizures: These are characterized by a rapid loss of consciousness with loss of muscle tone, tonic spasm, and jerks. The muscles become rigid for about 30 seconds during the tonic phase of the seizure and alternately contract and relax during the clonic phase, which lasts 30–60 seconds.
- Absence seizures: These are subdivided into typical and atypical forms based on duration of attack and level of consciousness. Absence (petit mal) seizures generally begin at about the age of four and stop by the time the child becomes an adolescent. They usually begin with a brief loss of consciousness and last between one and 10 seconds. People having a petit mal seizure become very quiet and may blink, stare blankly, roll their eyes, or move their lips. A petit mal seizure lasts 15–20 seconds. When it ends, the individual resumes whatever he or she was doing before the seizure began, will not remember the seizure, and may not realize that anything unusual happened. Untreated, petit mal seizures can recur as many as 100 times a day and may progress to grand mal seizures.
- Myoclonic seizures: These are characterized by rapid muscular contractions accompanied with jerks in facial and pelvic muscles.

Subcategories are commonly diagnosed based on EEG results. Terminology for classification in infants and newborns is still controversial.

Causes and symptoms

Causes

Simple partial seizures can be caused by congenital abnormalities (abnormalities present at birth), tumor growths, head trauma, **stroke**, and infections in the brain or nearby structures. Generalized tonic-clonic seizures are associated with drug and alcohol abuse, and low levels of blood glucose (blood sugar) and sodium. Certain psychi-

atric medications, antihistamines, and even antibiotics can precipitate tonic-clonic seizures. Absence seizures are implicated with an abnormal imbalance of certain chemicals in the brain that modulate nerve cell activity (one of these **neurotransmitters** is called GABA, which functions as an inhibitor). Myoclonic seizures are commonly diagnosed in newborns and children.

Symptoms

Symptoms for the different types of seizures are specific.

Partial seizures

SIMPLE PARTIAL SEIZURES. Multiple signs and symptoms may be present during a single simple partial seizure. These symptoms include specific muscles tensing and then alternately contracting and relaxing, speech arrest, vocalizations, and involuntary turning of the eyes or head. There could be changes in vision, hearing, balance, taste, and smell. Additionally, patients with simple partial seizures may have a sensation in the abdomen, sweating, paleness, flushing, hair follicles standing up (piloerection), and dilated pupils (the dark center in the eye enlarges). Seizures with psychological symptoms include thinking disturbances and **hallucinations**, or illusions of memory, sound, sight, time, and self-image.

COMPLEX PARTIAL SEIZURES. Complex partial seizures often begin with a motionless stare or arrest of activity; this is followed by a series of involuntary movements, speech disturbances, and eye movements.

Generalized seizures

Generalized seizures have a more complex set of signs and symptoms.

TONIC-CLONIC SEIZURES. Tonic-clonic seizures usually have vague prodromal (pre-attack) symptoms that can start hours or days before a seizure. These symptoms include anxiety, mood changes, irritability, weakness, dizziness, lightheadedness, and changes in appetite. The tonic phases may be preceded with brief (lasting only a few seconds in duration) muscle contractions on both sides of affected muscle groups. The tonic phase typically begins with a brief flexing of trunk muscles, upward movement of the eyes, and pupil dilation. Patients usually emit a characteristic vocalization. This sound is caused by contraction of trunk muscles that forces air from the lungs across spasmodic (abnormally tensed) throat muscles. This is followed by a very short period (10–15 seconds) of general muscle relaxation. The clonic phase consists of muscular contractions with alternating periods of no movements (muscle atonia) of gradually increasing duration until abnormal muscular contractions stop. Tonic-clonic seizures end in a final generalized spasm. The affected person can lose consciousness during tonic and clonic phases of seizure.

Tonic-clonic seizures can also produce chemical changes in the body. Patients commonly experience lowered carbon dioxide (hypocarbia) due to breathing alterations, increased blood glucose (blood sugar), and elevated level of a hormone called prolactin. Once the affected person regains consciousness, he or she is usually weak, and has headache and muscle pain. Tonic-clonic seizures can cause serious medical problems such as trauma to the head and mouth, fractures in the spinal column, pulmonary edema (water in the lungs), aspiration pneumonia (a pneumonia caused by a foreign body being lodged in the lungs), and sudden death. Attacks are generally one minute in duration.

TONIC SEIZURES. Tonic and atonic seizures have distinct differences but are often present in the same patient. Tonic seizures are characterized by nonvibratory muscle contractions, usually involving flexing of arms and relaxing or flexing of legs. The seizure usually lasts less than 10 seconds but may be as long as one minute. Tonic seizures are usually abrupt and patients lose consciousness. Tonic seizures commonly occur during nonrapid eye movement (nonREM) sleep and drowsiness. Tonic seizures that occur during wakeful states commonly produce physical injuries due to abrupt, unexpected falls.

ATONIC SEIZURES. Atonic seizures, also called "drop attacks," are abrupt, with loss of muscle tone lasting one to two seconds, but with rapid recovery. Consciousness is usually impaired. The rapid loss of muscular tone could

KEY TERMS

Electroencephalograph—(EEG) An instrument that measures the electrical activity of the brain. The EEG traces the electrical activity in the form of wave pattens onto recording paper. Wave patterns that have sudden spikes or sharp waves strongly suggest seizures. An EEG with a seizure-type wave pattern is called an epileptiform EEG.

Hallucination—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Illusion—A misperception or misinterpretation in the presence of a real external stimulus.

be limited to head and neck muscles, resulting in head drop, or it may be more extensive involving muscles for balance, causing unexpected falls with physical injury.

CLONIC SEIZURES. Generalized clonic seizures are rare and seen typically in children with elevated fever. These seizures are characterized by a rapid loss of consciousness, decreased muscle tone, and generalized spasm that is followed by jerky movements.

ABSENCE SEIZURES. Absence seizures are classified as either typical or atypical. The typical absence seizure is characterized by unresponsiveness and behavioral arrest, abnormal muscular movements of the face and eyelids, and lasts less than 10 seconds. In atypical absence seizures, the affected person is generally more conscious, the seizures begin and end more gradually, and do not exceed 10 seconds in duration.

MYOCLONIC SEIZURES. Myoclonic seizures commonly exhibit rapid muscular contractions. Myoclonic seizures are seen in newborns and children who have either symptomatic or idiopathic (cause is unknown) epilepsy.

Demographics

Approximately 1.5 million persons in the United States suffer from a type of seizure disorder. The annual incidence (number of new cases) for all types of seizures is 1.2 per 1,000 and, for recurrent seizures, is 0.54 per 1,000. Isolated seizures may occur in up to 10% of the general population. Approximately 10–20% of all patients have intractable epilepsy (epilepsy that is difficult to manage or treat). It is estimated that 45 million people in the world are affected by seizures. Seizures affect males and females equally and can occur among all age groups. There seems to be a strong genetic correlation, since seizures are three times more prevalent among close relatives than they are in the general population.

Children delivered in the breech position have increased prevalence (3.8%) of seizures when compared to infants delivered in the normal delivery position (2.2%). Seizures caused by fever have a recurrence rate of 51% if the attack occurred in the first year of life, whereas recurrence rate is decreased to 25% if the seizure took place during the second year. Approximately 88% of children who experience seizures caused by fever in the first two years experience recurrence.

Approximately 45 million people worldwide are affected by epilepsy. The incidence is highest among young children and the elderly. High-risk groups include persons with a previous history of brain injury or lesions.

Diagnosis

Patients seeking help for seizures should first undergo an EEG that records brain-wave patterns emitted between nerve cells. Electrodes are placed on the head, sometimes for 24 hours, to monitor brain-wave activity and detect both normal and abnormal impulses. **Imaging studies** such as **magnetic resonance imaging** (MRI) and computed axial tomography (CAT)—that take still "pictures"—are useful in detecting abnormalities in the temporal lobes (parts of the brain associated with hearing) or for helping diagnose tonic-clonic seizures. A complete blood count (CBC) can be helpful in determining whether a seizure is caused by a neurological infection, which is typically accompanied by high fever. If drugs or toxins in the blood are suspected to be the cause of the seizure(s), blood and urine screening tests for these compounds may be necessary.

Antiseizure medication can be altered by many commonly used medications such as sulfa drugs, erythromycin, warfarin, and cimetidine. Pregnancy may also decrease serum concentration of antiseizure medications; therefore, frequent monitoring and dose adjustments are vital to maintain appropriate blood concentrations of the antiseizure medication—known as the therapeutic blood concentration. **Diagnosis** requires a detailed and accurate history, and a physical examination is important since this may help identify neurological or systemic causes. In cases in which a central nervous system (CNS) infection (i.e., meningitis or encephalitis) is suspected, a lumbar puncture (or spinal tap) can help detect an increase in immune cells (white blood cells) that develop to fight the specific infection. (A lumbar puncture is removing from the spinal chord, by syringe, of a small amount of cerebrospinal fluid—the fluid that bathes and nourishes the brain and spinal cord.)

Treatments

Treatment is targeted primarily to:

- assist the patient in adjusting psychologically to the diagnosis and in maintaining as normal a lifestyle as possible
- reduce or eliminate seizure occurrence
- avoid side effects of long-term drug treatment

Simple and complex partial seizures respond to drugs such as **carbamazepine**, **valproic acid** (valproate), phenytoin, **gabapentin**, tiagabine, **lamotrigine**, and topiramate. Tonic-clonic seizures tend to respond to valproate, carbamazepine, phenytoin, and lamotrigine. Absence seizures seem to be sensitive to ethosuximide, valproate, and lamotrigine. Myoclonic seizures can be

treated with valproate and **clonazepam**. Tonic seizures seem to respond favorably to valproate, felbamate, and clonazepam.

People treated with a class of medications called **barbiturates** (Mysoline, Mebral, phenobarbital) have adverse cognitive (thinking) effects. These cognitive effects can include decreased general intelligence, attention, memory, problem solving, motor speed, and visual motor functions. The drug phenytoin (Dilantin) can adversely affect speed of response, memory, and attention. Other medications used for treatment of seizures do not have substantial cognitive impairment.

Surgical treatment may be considered when medications fail. Advances in medical sciences and techniques have improved methods of identifying the parts of the brain that generate abnormal discharge of nerve impulses. Surgical treatment now accounts for about 5,000 procedures annually. The most common type of surgery is the focal cortical resection. In this procedure, a small part of the brain responsible for causing the seizures is removed. Surgical **intervention** may be considered a feasible treatment option if:

- the site of seizures is identifiable and localized
- surgery can remove the seizure-generating (epileptogenic) area
- surgical procedure will not cause damage to nearby areas

Prognosis

About 30% of patients with severe seizures (starting in early childhood), continue to have attacks and usually never achieve a remission state. In the United States, the prevalence of treatment-resistant seizures is about one to two per 1,000 persons. About 60–70% of persons achieve a five-year remission within 10 years of initial diagnosis. Approximately half of these patients become seizure-free. Usually the prognosis is better if seizures can be controlled by one medication, the frequency of seizures decreases, and there is a normal EEG and neurological examination prior to medication cessation.

People affected by seizure have increased death rates compared with the general population. Patients who have seizures of unknown cause have an increased chance of dying due to accidents (primarily drowning). Other causes of seizure-associated death include abnormal heart rhythms, water in the lungs, or heart attack.

Prevention

There are no gold standard recommendations for prevention, since seizures can be caused by genetic factors, blood abnormalities, many medications, illicit drugs, infection, neurologic conditions, and other systemic diseases. If a person has had a previous attack or has a genetic propensity, care is advised when receiving medical treatment or if diagnosed with an illness correlated with possible seizure development.

See also Computed tomography (CAT); Electroencephalography (EEG); Magnetic resonance imaging (MRI); Substance abuse and related disorders

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Selective mutism

Definition

Selective mutism is a childhood disorder in which a child does not speak in some social situations although he or she is able to talk normally at other times.

Description

Selective mutism was first described in the 1870s, at which time it was called "aphasia voluntaria." This name shows that the absence of speech was considered to be under the control of the child's will. In 1934 the disorder began to be called selective mutism, a name that still implied purposefulness on the part of the silent child. In the 1994 edition of the ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV*) the disorder was renamed selective mutism. This name is considered preferable because it suggests that the child is mute only

KEY TERMS

Stimulus fading—A form of behavior modification used to treat children with selective mutism, in which goals of gradually increasing difficulty are set for the child.

in certain situations, without the implication that the child remains silent on purpose.

Selective mutism is characterized by a child's inability to speak in one or more types of social situation, although the child is developmentally advanced to the point that speech is possible. The child speaks proficiently in at least one setting, most often at home with one or both parents, and sometimes with siblings or extended family members. Some children also speak to certain friends or to adults that are not related to them, but this variant of selective mutism is somewhat less common.

The most common place for children to exhibit mute behavior is in the classroom, so that the disorder is often first noticed by teachers. Because of this characteristic, selective mutism is most frequently diagnosed in children of preschool age through second grade. As the expectation of speech becomes more evident, selective mutism can have more pronounced negative effects on academic performance. Children who do not talk in classroom settings or other social situations because the language of instruction is not their first tongue are not considered to have the disorder of selective mutism.

Causes and symptoms

The symptoms of selective mutism are fairly obvious. The child does not talk in one or more social situations in which speech is commonly expected and would facilitate understanding. Some children with selective mutism do not communicate in any way in certain settings, and act generally shy and withdrawn. The disorder is also often associated with crying, clinging to the parent, and other signs of social anxiety. Other children with the disorder, however, may smile, gesture, nod, and even giggle, although they do not talk.

Consensus regarding the most common causes of selective mutism has changed significantly over time. When the disorder was first studied, and for many years thereafter, it was thought to be caused by severe trauma in early childhood. Some of these causative traumas were thought to include rape, molestation, incest, severe physical or emotional **abuse**, and similar experiences. In addition, many researchers attributed selective mutism to family dynamics that included an overprotective mother and an abnormally strict or very distant father. As of 2002, these factors have not been completely eliminated as causes of selective mutism in most cases, but it is generally agreed that they are not the most common causes.

Instead, selective mutism is frequently attributed at present to high levels of social anxiety in children and not to traumatic events in their early years. Children with selective mutism have been found to be more timid and shy than most children in social situations, and to exhibit signs of depression, **obsessive-compulsive disorder**, and anxiety disorders. Some children have been reported to dislike speaking because they are uncomfortable with the sound of their own voice or because they think their voice sounds abnormal.

Many links have also been found between selective mutism and speech development problems. Language reception problems have also been documented in selectively mute children. Although there is no evidence indicating that selective mutism is the direct result of any of these difficulties in language development, possible connections are being explored.

Demographics

Selective mutism is generally considered a rare disorder. It is found in about 1% of patients in mental health settings, but it occurs in only about 0.01% of the general United States population. Some researchers maintain, however, that selective mutism occurs more frequently than these data suggest. There may be many unreported cases of selective mutism that resolve with time and require no **intervention**.

In terms of age grouping, selective mutism may appear at the very beginning of a child's social experience or may begin in later childhood. Some cases have been recorded in which selective mutism does not begin until high school. Onset in late adolescence is unusual, however; the most common age of onset for the disorder is the early elementary school years.

Selective mutism is often associated with **social phobia** in adult life. Children with selective mutism disorder may be more likely as adults to have a high level of social anxiety even if they do not meet the diagnostic criteria for social phobia. The disorder appears to run in families. Children whose parents are anxious in social settings, were exceptionally timid as children, or suffered from selective mutism themselves in childhood, are at greater risk for developing selective mutism.

Diagnosis

The criteria for diagnosing selective mutism disorder given by the reference manual, the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (*DSM-IV-TR*) include the failure to speak in some social situations even though the child may talk at other times. This criterion is not met if the child does not speak at all in any situation.

The child's inability to talk must interfere with the achievement of such relevant goals as schoolwork, play with friends, or communication of needs. In addition, the lack of speech must persist for at least one month. The first month of school should not be included in this measurement because many children are shy and unwilling to talk freely until they feel comfortable with their new teacher, classmates, and surroundings.

Furthermore, the child's lack of speech cannot be attributed to unfamiliarity with the language they must use in school or social settings. The **diagnosis** of selective mutism does not apply to children from immigrant families who may not feel comfortable conversing in a second language. Moreover, the child's inability to talk cannot be attributed to **stuttering** or similar speech disorders, which may make the child uncomfortable because they are aware that their speech sounds different from the speech of their peers. The lack of speech also must not be attributable to **schizophrenia**, **autism**, or other mental health disorders.

The disorder of selective mutism is usually noticed first by parents or teachers of affected children. It is often hard for doctors to diagnose selective mutism because it is unlikely that the child in question will talk to them. Therefore it may be difficult for a general practitioner to assess the existence of any underlying language or developmental problems that may be either causing or exacerbating the disorder. Tests that evaluate mental development without verbal responses from the patient may be used successfully to evaluate children with selective mutism.

There are also ways to test the child's speech development in the situations in which he or she does talk. One method involves interviews with the parents or whomever the child does speak to on a regular basis. This method can be fairly subjective, however. It is more useful for the doctor to obtain a tape or video recording of the child talking in a situation in which he or she feels comfortable. The child's hearing should be checked, as speech problems are often related to hearing disorders. Observing the child at play activities or asking him or her to draw pictures offer other effective ways to determine the child's reactions in social situations.

Treatments

A number of different approaches have been used in attempts to treat selective mutism. Recent opinion has moved away from the idea that it is caused by a trauma, and attempts to treat it have followed accordingly. The factors that are most intensively studied at present are underlying anxiety problems. In the few cases in which an underlying trauma is discovered to be the source of the problem, counseling to help treat the underlying problems is recommended. Treatments of any kind are generally found to be more effective when the family of the child is involved in decisions about his or her treatment.

Behavior modification

Selective mutism can be treated by using a **reinforcement** approach. This method gives positive rewards to the child in the form of praise, treats, privileges, or anything else that the child values. In general rewards are given for speech, and withheld for silence. The use of punishments alongside the rewards is not generally recommended because it would place more **stress** on children who are already severely anxious. The positive reinforcement technique is generally found to be at least partially successful in most cases.

Another technique for modifying behavior in children with selective mutism is known as stimulus fading. This technique sets goals of increasing difficulty for the child to meet. For example, the child might be encouraged to start talking by whispering, then work up gradually to talking at full volume. Alternately, the child could start by talking to one person who is not a family member and gradually add names until he or she feels comfortable talking to more than one person at a time. Stimulus fading has been found to be particularly effective when it is used in conjunction with positive reinforcement techniques.

Treatment with medications

In some cases, selective mutism is treatable with medication. **Fluoxetine** (Prozac), which is one of the selective serotonin reuptake inhibitors (SSRIs) is the drug that has been studied most often as a treatment for selective mutism. Treatment with medication is more successful in younger children. Overall, fluoxetine has been found to reduce the symptoms of selective mutism in about three-fourths of children. Other drugs used to treat anxiety and social phobia disorders may also be effective in certain cases.

Prognosis

Selective mutism is frequently treatable, in that many cases of the disorder are thought to resolve on their own. Sometimes reported cases do resolve with time, although treatment can be very effective. There is little information about the long-term outcome of selective mutism. Researchers have noted that while many children with the disorder do show improvement in speech, their anxiety in social situations persists.

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Self-control strategies

Definition

Self-control strategies are cognitive and behavioral skills used by individuals to maintain self-motivation and achieve personal goals. Initially the skills may be learned from a therapist, text, or self-help book. However, the individual is responsible for using these skills in real-life situations to produce the desired changes.

There are many varieties of self-control strategies. Other terms for self-control strategies are behavioral self-control training, cognitive self-regulation, and self-management techniques. In recent years, the term "self-management" has replaced "self-control," because self-control implies changing behavior through sheer willpower. Self-management, on the other hand, involves becoming aware of the natural processes that affect a particular behavior and consciously altering those processes, resulting in the desired behavior change.

Purpose

Most people who decide to use self-control strategies are dissatisfied with a certain aspect of their lives. For example, they may feel they smoke too much, exercise too little, or have difficulty controlling anger. Self-control strategies are useful for a wide range of concerns, including medical (such as diabetes, chronic pain, asthma, arthritis, incontinence, or **obesity**), addictions (such as drug and alcohol abuse, smoking, gambling, or eating disorders), occupational (such as study habits, organizational skills, or job productivity), and psychological (such as **stress**, anxiety, depression, excessive anger, hyperactivity, or shyness). If symptoms are severe, self-control strategies may be used in conjunction with other therapies, but should not be the only form of treatment.

The goal of self-control strategies is to reduce behavioral deficiencies or behavioral excesses. Behavioral deficiencies occur when an individual does not engage in a positive, desirable behavior frequently enough. The result is a missed future benefit. For example, a student who rarely studies may not graduate. Behavioral excesses occur when an individual engages in negative, undesirable behavior too often. This results in a negative future consequence. For example, a person who smokes may develop lung cancer.

In the case of behavioral deficiencies, one may fail to engage in a desirable behavior because it does not provide immediate gratification. With behavioral excesses, there is usually some type of immediate gratification and no immediate negative consequence. Self-control strategies help individuals to become aware of their own patterns of behavior and to alter those patterns (usually by creating artificial rewards or punishments) so that the behavior will be more or less likely to occur.

Description

Theoretical bases for self-control strategies

Self-control strategies are based primarily on the social cognitive theory of Albert Bandura. According to Bandura, one's behavior is influenced by a variety of fac-

KEY TERMS

Antecedents—Events that occur immediately before the target behavior.

Behavioral deficiency—Failure to engage in a positive, desirable behavior frequently enough.

Behavioral excess—Engaging in negative, undesirable behavior too often.

Competing behaviors—Behaviors that interfere with the target behavior because they are preferred by the individual.

Consequences—Events that occur immediately after the target behavior.

Contingencies—Naturally occurring or artificially designated reinforcers or punishers that follow a behavior.

Controlled behavior—The behavior to be changed by self-control strategies; also known as the target behavior.

Controlling behaviors—Self-control strategies used to change the controlled or target behavior.

Feedback loop—A naturally occurring process whereby individuals control their behavior by self-monitoring, self-evaluation, and self-reinforcement.

Outcome expectancies—What one believes will happen as a result of engaging in a certain behavior.

Punisher—Anything that causes a decrease of a particular behavior.

Reinforcer—Anything that causes an increase of a particular behavior.

Self-efficacy—One's belief about how well he or she can perform a given task, regardless of that person's actual ability.

Self-instructional training—Teaches individuals to become aware of their self-statements, evaluate whether these self-statements are helpful or hindering, and replace maladaptive self-statements with adaptive ones.

Short-circuiting of contingencies—The proper reinforcer or punisher for a given behavior is not administered.

Social cognitive theory—The theory that behavior is determined by an interaction between cognitive, behavioral, and environmental factors.

Target behavior—The specific behavior to be increased or decreased during treatment

tors, including one's own thoughts and beliefs, and elements in the environment. Bandura proposed that certain beliefs, self-efficacy and outcome expectancies, are important factors in determining which behaviors an individual will attempt, and how motivated the individual will be when engaging in those behaviors. Self-efficacy is one's belief about how well he or she can perform a given task, regardless of that person's actual ability. Outcome expectancies are what the person believes will happen as a result of engaging in a certain behavior. If self-efficacy and outcome expectancies are inaccurate, the individual may experience behavioral deficits or excesses.

Donald Meichenbaum developed the idea of self-instructional training, which is a major part of self-control strategies. Meichenbaum believed that learning to control behavior begins in childhood, based on parental instruction. Children eventually control their own behavior by mentally repeating the instructions of their parents. These internal instructions may be positive or negative. Self-instructional training teaches individuals to become aware of their self-statements, evaluate whether these self-statements are helpful or hindering, and replace maladaptive self-statements with adaptive ones.

Frederick Kanfer suggested that individuals achieve self-control by using a feedback loop consisting of continuous monitoring, evaluating, and reinforcing of their own behavior. This loop occurs naturally in everyone. However, the loop can be maladaptive if (a) only negative factors are noticed and positive factors are ignored during the monitoring phase, (b) standards are unrealistic during the evaluation phase, or (c) responsibility is accepted for negative behaviors but not for positive behaviors during the **reinforcement** phase. Self-control strategies help individuals to be aware of these phases and to make the appropriate changes in monitoring, evaluation, and reinforcement.

Development of a self-control program

Self-control strategies are often taught in treatment centers, group or individual therapies, schools, or vocational settings. However, self-control programs may also be designed without the help of a professional, especially if the problem being addressed is not severe. The use

of professionals, at least initially, may increase the likelihood that the program will succeed. Following are the necessary steps for creating a self-control program:

- Making a commitment. A plan cannot succeed unless one is committed to following through. Ways of increasing commitment level include listing the benefits of adhering to the program, telling others about one's intentions, posting written reminders of commitments around one's home, putting a significant amount of time and energy into designing the program, and planning ways to deal with obstacles ahead of time.
- Identifying the problem. The behavior in need of change is referred to as the target behavior or the controlled behavior. A precise definition of the target behavior is a crucial first step. This is usually done by keeping detailed records about when, where, and how the behavior occurs for one to two weeks. The record-keeping should also focus on other competing behaviors that may be interfering with the target behavior. For example, for a person who is trying to cut down on calorie consumption, a competing behavior would be eating high-calorie snack foods. It is important to note the antecedents and consequences of the target and competing behaviors; in other words, what typically occurs immediately before (antecedents) and after (consequences) these behaviors? The antecedents and consequences are factors that influence the occurrence of the behavior. Sometimes just the process of record-keeping alters the target behavior by increasing the individual's awareness of what he or she is doing.
- Setting a goal. Once the target behavior has been defined, the individual must decide in what way that behavior should be changed. The goal should be specific so that future progress can be measured. This may entail listing circumstances or behaviors that must be present, as well as to what degree they must be present, in order for a goal to be achieved. For example, a goal to "reduce hyperactivity" in a grade-school student is vague. "Remaining in seat for seven out of fourteen half-hour periods daily" is much more specific. Indicating a time frame in which the goal can realistically be achieved is also recommended. Goals should be realistic. It is better to set a small goal and progress to bigger goals than to set a big goal and become quickly discouraged.
- Applying self-control strategies. The self-control strategies are known as controlling behaviors. Choice of strategies will depend on the target behavior. Types of strategies are discussed later.
- Self-monitoring. While using the self-control strategies, one should continue to keep records regarding the occurrence of the target behavior. Keeping written records is essential for determining if the strategies are effective. If one is gradually meeting the goal requirements, the strategies can be assumed effective. If little progress towards the goal is evident, either the strategies are being used incorrectly, or the strategies are ineffective and should be changed. Self-monitoring can be done informally (for instance, by making notes on an index card) or formally (by using pre-designed data sheets). In any case, self-monitoring should gather the necessary information, but should not become too lengthy or complex. The individual will lose motivation to continue monitoring if the procedures are overly time-consuming or inconvenient.
- Making revisions as necessary. Based on the information gathered during self-monitoring, the individual decides if changes in the plan are necessary. One advantage of self-control programs is that the individual chooses the strategies that will work best for him or her. This freedom of choice increases the likelihood that the individual will adhere to the program. Therefore, self-control programs should always be flexible and adaptable.

Types of self-control strategies

Self-control strategies can be grouped into three broad categories:

ENVIRONMENTAL STRATEGIES. Environmental strategies involve changing times, places, or situations where one experiences problematic behavior. Examples include:

- changing the group of people with whom one socializes
- avoiding situations or settings where an undesirable behavior is more likely to occur
- changing the time of day for participating in a desirable behavior to a time when one will be more productive or successful

BEHAVIORAL STRATEGIES. Behavioral strategies involve changing the antecedents or consequences of a behavior. Examples include:

- increasing social support by asking others to work towards the same or a similar goal
- placing visual cues or reminders about one's goal in one's daily environment
- developing reinforcers (rewards) for engaging in desirable behaviors or punishers for engaging in undesirable behaviors
- eliminating naturally occurring reinforcers for undesirable behavior
- engaging in alternative, positive behaviors when one is inclined to engage in an undesirable behavior

- creating ways to make a desirable behavior more enjoyable or convenient
- scheduling a specific time to engage in a desirable behavior
- writing a behavioral contract to hold oneself accountable for carrying out the self-control program

COGNITIVE STRATEGIES. Cognitive strategies involve changing one's thoughts or beliefs about a particular behavior. Examples include:

- using self-instructions to cue oneself about what to do and how to do it
- using self-praise to commend oneself for engaging in a desirable behavior
- thinking about the benefits of reaching one's goal
- imagining oneself successfully achieving a goal or using imagery to distract oneself from engaging in an undesirable behavior
- substituting positive self-statements for unproductive, negative self-statements

In a therapeutic setting, self-control strategies are usually taught in weekly group sessions over a period of several weeks. The sessions typically include an educational lecture regarding a specific strategy, group discussion of how the strategy should be applied and how to cope with potential obstacles (relapse prevention), role-plays or rehearsal of the strategy, a review of the session, and a homework assignment for further practice. Sessions usually focus on one type of strategy at a time. Preferably, an individual should master one strategy before attempting another. After the series of training sessions are complete, the individual is responsible for implementing the strategies in daily life.

Aftercare

Relapse is a concern in any therapeutic situation. Current research suggests that individuals are more likely to continue using newly learned self-control strategies if they have periodic follow-up contact with a professional or other designated person. The contact serves at least three purposes: (1) a source of accountability, (2) review of strategy use to ensure proper application, and (3) discussion of problematic situations and development of plans to overcome these situations.

Risks

Self-control strategies are especially prone to short-circuiting of contingencies. This refers to the tendency for individuals to partake of reinforcers at inappropriate occasions, or to avoid punishers designated in their plan. If contingencies are short-circuited, the desired behavior change is unlikely to occur.

Relapse is another risk involved in self-control strategies. Causes of relapse include: (a) a poorly defined target behavior (progress cannot be recognized); (b) unrealistic or long-term goals without immediate sources of reinforcement; (c) failure to anticipate and plan for obstacles to goal-achievement; (d) overreaction to occasional setbacks; (e) negative self-talk, especially when one feels goals are not being satisfactorily met; (f) failure to use desirable or frequent reinforcers; (g) ineffective consequences for undesirable behavior; and (h) an inaccurate or unnecessarily complex monitoring system.

Normal results

Ideally individuals will use self-control strategies independently in their everyday surroundings to meet their designated goal. They will decrease behavioral deficiencies and excesses, engaging in desirable behaviors more often, or engaging in undesirable behaviors less frequently or not at all.

Abnormal results

If the self-control strategies are ineffective or used improperly, individuals may show no changes or increases in behavioral deficiencies or excesses.

See also Behavior modification; Bibliotherapy; Cognitive retraining techniques; Cognitive-behavioral therapy; Guided imagery therapy; Rational emotive therapy; Social skills training

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ORGANIZATIONS

Association for Behavioral Analysis. 213 West Hall, Western Michigan University, 1903 W. Michigan Avenue, Kalamazoo, Michigan 49008-5301. (616) 387-8341; (616) 384-8342. <http://www.wmich.edu/aba>.

Beck Institute for Cognitive Therapy. GSB Building, City Line and Belmont Avenues, Suite 700, Bala Cynwyd, Pennsylvania 19004-1610. (610) 664-3020. <http://www.beckinstitute.org>.

Cambridge Center for Behavioral Studies. 336 Baker Avenue, Concord, Massachusetts 01742-2107. (978) 369-2227. <http://www.behavior.org>.

Cognitive-Behavioral Therapy Institute. 211 East 43rd Street, Suite 1500, New York, New York 10017. (212) 490-3590. <http://www.cbtinstitute.com>.

Sandra L. Friedrich, M.A.

Self-help groups

Introduction

Self-help groups—also called mutual help or mutual aid groups—are composed of peers who share a similar mental, emotional, or physical problem, or who are interested in a focal issue, such as education or parenting. Historically, people banded together to improve their chances for survival by pooling their social and economic resources; however, contemporary groups are more likely to organize around a theme or problem.

Most self-help groups are voluntary, non-profit associations open to anyone with a similar need or interest; however, spin-off groups also exist to meet the needs of particular types of people; for example, the elderly, women, or Hispanics. Usually, groups are led by peers, have an informal structure, and are free (except for small donations to cover meeting expenses). However, professionals of various kinds lead some self-help groups.

In the past thirty years, the number of self-help organizations and groups operating in communities throughout the U.S. has dramatically risen; some organizations operate in several countries, primarily in the developed world. One of the reasons for the rapid proliferation of groups focusing on health problems may be the advent of managed health care. For individuals with insurance plans offering limited mental health coverage, self-help groups are an economical way to find emotional and social support.

Self-help groups and therapy

Because of the peer-led, informal, and democratic (as opposed to hierarchical and medical) structure, health professionals consider self-help groups for mental or emotional problems to be an adjunct to therapy. While there are therapeutic aspects associated with participation—principally, intimacy as a result of self-disclosure, personal growth in response to others' role modeling, and erosion of **denial** as a result of social confrontation—the primary value of contemporary groups is in the mutual aid offered by members to one another. Though the nature of self-help groups is outside of the medical realm, doctors and therapists see participation as a way to improve the outcome related to either ongoing or future formal treatment.

Another issue arguing against considering self-help groups as a type of therapy is that the variety of groups is extensive; groups available may include advocacy groups with a focus on legal or social remedies, groups organized around housing or employment needs, and groups focusing on racial or gender issues. Additionally, the self-help movement shares some characteristics with volunteerism and consumerism. In general, members who persevere have experience with other voluntary organizations and believe in the value of donating time and service; also, members may be thought of as consumers who participate in their own care and who have experience and knowledge of relevant goods and services.

Types of self-help groups

Twelve-step groups

The most popular type of self-help group is based on the Twelve Steps and Twelve Traditions of Alcoholics Anonymous (AA), founded in 1935. The Twelve Steps are a guide to recovery from alcoholism or **addiction**, whereas the Twelve Traditions are a code of ethics. AA and other 12-step programs are based on the spiritual premise that turning one's life and will over to a personally meaningful "higher power," such as God or Spirit, is the key to recovery. Another essential idea is that sobriety or recovery (not cure) depends on the admission of powerlessness with respect to alcohol or the sub-

stance(s) abused. This idea is offensive to critics of 12-step groups, but others believe that this admission accurately reflects the contemporary view of addiction as a disease. Furthermore, people with a familial, genetic vulnerability to addiction are particularly at risk. While some studies suggest that 20% of people suffering from alcoholism will experience remission without benefit of therapy or a 12-step group, most will suffer deteriorating health and dysfunctional, if not ruined, social relationships. In other words, most alcoholics need formal therapy or an informal self-help program to recover. While the dropout rate for AA groups during the first three months is high, alcoholics who persevere have a good chance of attaining and maintaining sobriety or abstinence. This is especially true if the person regularly attends a home group (90 meetings in the first 90 days, slowly diminishing to two or three times per week for years thereafter) and finds an experienced and sympathetic sponsor who also is in recovery.

In addition to AA and its sister organizations, Narcotics Anonymous (NA) and Cocaine Anonymous (CA), a number of 12-step organizations exist for a variety of disorders, such as Gambler's Anonymous (GA), Schizophrenics Anonymous (SA), Emotions Anonymous (EA), and Overeaters Anonymous (OA).

Other groups for health problems and diseases

Self-help organizations also provide support for individuals who are ill or have health problems. For example, support exists for people coping with weight management, HIV/AIDS, multiple sclerosis, muscular dystrophy, cancer, incontinence, and for the families of individuals who suffer from these conditions. Also, support exists for people who share interests or circumstances, such as groups for women who breast-feed (LaLeche League), singles, older adults, and new parents.

Self-help groups for family members are available since illness, addiction, and distress affect the entire family. Family members may unwittingly reinforce illness or addictive behaviors, or may need help coping with the person in distress. Al-Anon, an organization for friends and families of alcoholics, is a companion organization to AA, as is Alateen, a program for teenagers who have been hurt by the alcoholism of significant people in their lives. **Support groups** for caregivers of individuals with life-threatening or terminal illnesses, such as cancer, often meet at treatment centers and hospitals. One popular club for people with cancer, as well as for their friends and family, is Gilda's Club, founded by the actor/comedian Gene Wilder, Gilda Radner's husband. Gilda Radner, the well-known comedienne from Saturday Night Live, died at age 40 from ovarian cancer. Gilda's Clubs can be found in at least a half-dozen cities in the U.S., Canada, and London.

Online groups and clearinghouses

A growing trend in the self-help movement is the online support communities, as well as online resource centers and clearinghouses. Chat rooms, bulletin boards, and electronic mailing lists all provide convenient, around-the-clock access to peer support. Many large-scale, consumer health care web sites provide forums for discussions on numerous diseases and disorders, and major online commercial services, such as America Online (AOL), provide sites for health care and patient support. In some cases, professionals moderate online groups, although many are exclusively organized and populated by peers. There are self-help groups, such as LaLeche League, that hold some meetings online, often at their own web sites.

KEY TERMS

Clearinghouse—A centralized organization that is a repository of information and that facilitates access to information.

Cognitive restructuring—An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

Experiential knowledge—Knowledge gained from experience, often practical, in contrast with theoretical or professional knowledge.

Features of self-help groups

Accessibility

Accessibility and economy are appealing features of self-help groups. Since the groups are free, organizations such as AA and NA are very cost-effective. In addition, meetings are easy to locate through local newspaper announcements, hospitals, health care centers, churches, school counselors, and community agencies. For AA and sister organizations that encourage frequent attendance, hundreds of meetings may be held each week in large metropolitan areas. Furthermore, with the proliferation of online support communities and growth of connectivity to the Internet, self-help groups are becoming as accessible for individuals in rural areas as they are for those in large cities.

Anonymity

An important characteristic of 12-step groups is the preservation of anonymity by revealing first names only and by maintaining strict confidentiality of stories shared during meetings. Online self-help groups offer even more anonymity since the exchanges are not face to face. The virtual anonymity of online experience helps to reduce social discomfort and discrimination, or stereotyping otherwise associated with real-life perceptions of age, disabilities, race, gender, or culture.

Social support and mutual aid

Self-help groups provide an intact community and a sense of belonging. The social support and mutual aid available in a group may be critical to recovery, rehabilitation, or healthy coping. This is especially true for socially isolated people or people from dysfunctional families, who may have little or no emotional support. Participating in a social network of peers reduces social and emotional isolation and supports healthy behavior. Group members can offer unconditional support and, collectively, are a repository of helpful experiential knowledge.

Self-esteem and self-efficacy

Self-help groups promote self-esteem or self-respect by encouraging reciprocal caring; the concept of self-efficacy, or the belief that one is capable, is promoted by reinforcing appropriate behavior and beliefs and by sharing relevant information regarding the disease or condition. For example, there may be an exchange of information regarding how to cope with failed or disrupted relationships, about what is reasonable to expect from health care professionals, about how to manage pain or public embarrassment, about where to go and to whom for a variety of needs. In groups such as AA, self-efficacy also is promoted by sponsors who act as mentors and role models, and by encouraging rotating leadership roles.

Introspection and insight

Introspection, or contemplation, is another fundamental feature of many self-help groups, particularly for groups that follow a 12-step program of recovery. For example, the fourth step of AA states that members make "a searching and fearless moral inventory" of themselves, and the tenth step states that members continue "to take personal inventory" and admit wrongdoing. Introspection is particularly beneficial to individuals who are not entirely aware of the moral repercussions of and motivation for their behavior. In a sense, working through some of the 12 steps resembles the cognitive restructuring learned in **cognitive-behavioral therapy** (CBT), as maladaptive ideas and behaviors are transformed.

Spiritual recovery

The final step in a 12-step program recognizes that recovery entails a spiritual awakening; furthermore, recovering addicts are enjoined to spread the message to others suffering from addiction. Recovery depends on giving up both injurious self-will and denial of maladaptive behavior, and turning to a higher power. Members are urged to seek guidance or inspiration from this higher power. For many addicts, the key to recovery is a spiritually guided movement away from self-centeredness or self-absorption, and a turning towards the "Power greater than ourselves" through prayer and **meditation**.

Advocacy

Some self-help groups meet to advocate or promote social and legislative remedies with respect to the issue of concern. For example, HIV/AIDS groups have lobbied for improved access to prescription drugs. Groups lobby for reforms by identifying key legislators and policy makers; they submit papers or suggestions for more equitable laws and policies to these key people. They also conduct public education programs (including programs meant to redress the harm of stigmatization). There are groups that advocate for more funds for research and for improved services for people who suffer from one of many diseases or mental disorders. The most important grass roots organization of families and consumers of psychiatric services (former or current patients) is the National Alliance for the Mentally Ill (NAMI). This organization was founded in 1979, and blends self-help with advocacy efforts for the improvement of research, services, and public awareness of major mental illnesses. Their advocacy efforts target both the federal and state levels.

Limitations

Advocacy vs. mutual aid

In some organizations, there is a growing overlap between self-help efforts and community development. Critics maintain that focusing on issues such as crime prevention, affordable housing, and economic development drains time and effort from social support and mutual aid. Nevertheless, some organizations continue to develop both advocacy and support.

Lack of professional involvement

The absence of professional guidance may mean that a member in need of formal **psychotherapy** or treatment may be discouraged from seeking professional help. On the other hand, too much professional involvement in the group may compromise the quality of mutual aid.

The " thirteenth step"

There is a well-known risk associated with attending 12-step groups termed the "thirteenth step." Women new to the groups, especially young women, are at their most vulnerable in the early stages of recovery. Male sexual predators who attend meetings take advantage of the atmosphere of intimacy and mutual trust. To cope with the possibility of sexual exploitation, young females are encouraged to attend meetings with a family member or a trusted adult, and all women are encouraged to find a same-sex sponsor.

Substituting addictions

The early months of a 12-step program are especially difficult. Typically, an addict in early recovery either replaces an addictive substance with a new one, or intensifies his/her concurrent use of another substance.

It is not uncommon for people who are chemically dependent to also have an addictive sexual disorder. (When someone is addicted to sex, there is an intense desire to gratify sexual urges and fantasies or to behave in ways that cause clinically significant distress; sexual indulgence, often compulsive, is a major disruptive force with respect to social relationships.) In one four-year study of a treatment program, 33% of the chemically addicted patients also were sexually compulsive. Some physicians believe that the predatory "thirteenth step" is evidence of turning from one addiction to another—in this case, addictive sexual disorder.

Members at varying stages of recovery

Another common risk is associated with the varying levels of recovery in a self-help group—that of being actively involved in the abuse of alcohol and/or drugs. Newcomers need to realize that not all members are interested in supporting their recovery, and that people in later stages of recovery may be more reliable. Furthermore, some members are required to attend by disciplinary entities, such as employers or correctional authorities.

Ongoing meetings

One criticism of self-help groups, especially 12-step groups, is that in the eyes of families and friends, members who persevere and faithfully attend the seemingly endless number of meetings only to become "addicted" to the program. However, physicians who support self-help groups point out that since addiction is a disease, addicts are particularly vulnerable to relapse, and that ongoing involvement with a self-help community surely is better than suffering the recurring misery associated with active addiction.

An Alcoholics Anonymous meeting in progress. *(Larry Mulvehill. Photo Researchers, Inc. Reproduced by permission.)*

Rational alternatives to 12-step groups

For addicts who find the spirituality of 12-step groups offensive and irrational, and who believe that public proclamation of powerlessness at group meetings is demoralizing, alternative groups exist. For example, a well-known organization, Rational Recovery (RR), is based on the cognitive-behavioral principles of Albert Ellis. RR emphasizes self-reliance, rational thinking as a result of cognitive restructuring, and the development of a new repertoire of behaviors to respond effectively to events that trigger relapse.

Conclusion

Worldwide, self-help groups are becoming increasingly popular. They are effective in providing mutual support and are good resources for finding needed infor-

mation. However, when searching for an appropriate group, prospective members should ask their friends, physicians, and counselors for references, and then visit a few groups before deciding on which one to attend. Also, information clearinghouses on the Internet are a good first step.

See also Depression and depressive disorders; Disease concept of chemical dependency; Dual diagnosis; Group therapy; Pathological gambling; Poly-substance abuse; Sedatives and related disorders; Support groups

Resources

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ORGANIZATIONS

Aloholics Anonymous. Grand Central Station, PO Box 459, New York, NY 10163. <www.alcoholicsanonymous.org>.

Gilda's Club Worldwide. 322 Eighth Avenue, Suite 1402, New York, NY 10001. (888) GILDA-4-U. <http://www.gildasclub.org/>.

Narcotics Anonyomous World Service Office. PO Box 9999, Van Nuys, California 91409. Telephone: (818) 773-9999. Fax (818) 700-0700. <http://www.na.org/>.

National Self-Help Clearinghouse. Graduate School and University Center of The City University of New York, 365 5th Avenue, Suite 3300, New York, NY 10016. (212) 817-1822. <http://www.selfhelpweb.org>.

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Separation anxiety disorder

Definition

Like many childhood concerns, separation anxiety is normal at certain developmental stages. For example, when a child between the ages of eight and 14 months is separated from her mother or other primary caretaker, she may experience distress. This is normal. However, separation anxiety that occurs at later ages is considered a disorder because it is outside of normal developmental expectations, and because of the intensity of the child's emotional response. Separation anxiety disorder occurs most frequently from the ages of five to seven and from 11 to 14.

Environmental stimuli and internal cues from the child himself interact in the presentation of separation anxiety disorder. Separation anxiety disorder is defined by the primary expression of excessive anxiety that occurs upon the actual or anticipated separation of the child from adult caregivers—most often the parents. Significant problems in daily functioning for the child and parents can result from the disorder. Common fears observed in the presentation of separation anxiety include concerns about the parents' health or well-being (less frequently the child's own health), general catastrophes, natural disasters, or the child becoming lost/separated from the parents. Disrupted sleep, difficulty falling asleep alone, fear of monsters, or nightmares are also commonly experienced by children with separation anxiety disorder.

Family routines, parents' work schedules, and siblings' activities may all be negatively affected by the excessive anxiety and demands of the child with separation anxiety disorder. Family life is often disrupted by efforts to soothe the child. Parents can become stressed themselves as they try to maintain their daily routines and obligations, while attempting to manage their child's anxiety. The family's adjustment is often made more difficult due to the sudden appearance of symptoms.

Description

Children experiencing separation anxiety disorder display significant distress upon separation from the parent or other primary caregiver. Separation anxiety disorder often becomes problematic for families during elementary school, although it can also occur in older or younger children. The child appears fearful because he or she thinks something horrible will happen to the child or parent while they are apart. The child's responses to separation may include crying or becoming angry with the adult in an attempt to manipulate the situation. When

thwarted by the adult's appropriate boundaries, expectations, and structure (the child must attend school, for example), the child's distress may become displaced into other maladaptive or negative behaviors. The child may begin to exhibit behavioral problems at school or at home when there has been no previous history of such problems. The child may seek out a new, negative peer group in order to gain attention or avoid separation.

Many children are unable to describe their specific fear. The feelings may seem more general and engulfing, especially to the younger child, making description more difficult and the feelings more overpowering. Children, and even adolescents, may experience difficulty describing their internal thoughts and feelings, which is normal. The ability to self-monitor, or observe one's own behavior or decision-making process, does not develop until late in adolescence for some individuals. When caregivers press the child experiencing separation anxiety for explanations, the feelings of anxiety can actually become more overwhelming. The intensity of the child's emotional response, accompanied by a lack of explanation, can become very frustrating for parents. Children or adolescents with an angry or frustrated parent may create a reasonable explanation for their fears to appease caregivers, and to keep them from leaving. Lying to take the emphasis off their strong feelings may be one of the early behavioral changes that can accompany separation anxiety.

Although exposure to a specific stressor is not required for the development of separation anxiety disorder, in many cases, a specific incident may precipitate the onset of the disorder (the traumatic events of September 11, 2001, for example). Another common precipitant is the holiday or summer break from school. Some children experience significant difficulty returning to school after a relatively short break, but certainly after summer and holidays.

Causes and symptoms

Causes

- Environmental change. Separation anxiety disorder is often precipitated by change or **stress** in the child's life and daily routine, such as a move, death or illness of a close relative or pet, starting a new school, a traumatic event, or even a return to school after summer vacation.
- Genetic influence. Evidence suggests a genetic link between separation anxiety disorders in children and a history of **panic disorder**, anxiety, or depression in their parents. Infants with anxious temperaments may have a predisposition toward later development of anxiety disorders.

KEY TERMS

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

- Parent/child attachment. Quality of attachment between children and their parents has also been identified as a factor in separation anxiety disorder. If the child senses emotional distance, the behaviors may be an attempt to draw the parent in more closely. The problematic behaviors can also draw the attention and care of others as well.
- Developmental considerations. Children develop at different rates when compared to each other (boys mature slower than girls, for example). Furthermore, the rate of development within the same person can vary across different types of functioning (for example, a gifted child is advanced intellectually but may be behind developmental expectations for social and emotional areas of functioning). A slower rate of development in the intellectual, social, emotional, or physical arena can foster anxiety within the child, making the separation more difficult.
- Cognitive factors. Children repeatedly worry about what they are afraid of (getting lost or a parent getting hurt, for example). The thought patterns are repeated within the child's mind until his emotions are beyond his control. The child may feel he is unable to think about anything else other than his fears, which contributes to his anxiety and irrational behaviors.
- Behavioral factors. The child or adolescent's crying and clinging behaviors may be developed by the child to cope with the feelings of anxiety associated with certain people, environment, or situations, such as attending school. The behaviors serve to distract attention away from the child's negative feelings, while nurturing the anxiety and fear into a greater part of the child's daily experience. For children, the behavioral component often becomes the mode of expression for the anxiety. The behavior may appear manipulative at times, due to the quick disappearance of symptoms once the threat of separation passes.
- Stress factors and influence. Symptoms of separation anxiety disorder may be exacerbated by a change in routine, illness, lack of adequate rest, a family move, or change in family structure (such as death, divorce, parent illness, birth of a sibling). The child's symptoms may also be affected by a change in caregivers or changes in parents' response to the child in terms of

discipline, availability, or daily routine. Even if changes are positive or exciting, the change may feel uncomfortable and precipitate an anxious response in the child.

Symptoms

The *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*, a handbook for mental health professionals that aids in **diagnosis**, lists the following criteria for separation anxiety disorder.

- Recurrent excessive distress upon separation. The child may become focused on the separation long before the actual event, or simply at the time of the anticipated separation. The recurrent behavioral pattern does not respond to **intervention**. The child experiences extreme distress, a highly charged emotional response that is repeated when the child anticipates separation from the caregiver. The child's fears trigger more anxiety and the emotional response intensifies.
- Persistent and excessive worry. The content of the worry may include some type of harm occurring to the child himself or toward the parents, or it may focus on becoming lost or separated indefinitely from the parent or caregiver.
- Repetitive nightmares. The child may experience repeated nightmares with themes of being chased, harmed, or separated from her family. Some fears are age-appropriate, but in separation anxiety disorder, the intensity of the fears becomes overwhelming to the child, leaving little opportunity for the child to control her emotions or behaviors. Although dreams are often a way of exploring and making sense of daily life, children with separation anxiety disorder report nightmares that represent their irrational fears or preoccupation with disaster.
- Complaints of physical symptoms. The child may feign illness (headaches, stomachaches, etc.) to avoid separation, or the child may actually experience nausea upon separation. If allowed to continue, the child may develop psychosomatic symptoms (physical symptoms with a psychological origin) that prevent the child from attending or fully participating in school activities. In these cases, the separation anxiety may develop into a more serious hypochondriacal state in which the child complains of chronic pain, which results in the child getting what she wants (i.e., not attending school).
- Persistent reluctance or refusal to engage in age-appropriate activities. The child may refuse to attend school because of preoccupation about separation from the parent. The child may also experience reluctance to be alone at home or at school without another adult being immediately available. The child may resist sleep without an adult present. The disorder causes significant disruption in the child's daily routine and may decrease the ability to perform previously mastered tasks. The child may appear to have reverted to behaviors from a younger age. The intensity of her emotions blocks the child's ability to communicate her feelings in ways other than through behaviors. Examples include tantrums, hitting, or clinging. Crying is one of the primary behaviors associated with separation anxiety disorder. The crying can become quite intense, making it difficult for the child to regain composure.
- Enmeshment or unusual interest in parents' schedules. The child wants to know all the details of the daily routine, a behavior which minimizes the anxiety the child is feeling.
- Quick resolution of symptoms (upon meeting child's demands). It may be hard for parents to accept the reality of the disorder because the symptoms often disappear quickly when separation does not occur. It is this component that can feel manipulative to those in the child's life.

Demographics

Prevalence estimates of separation anxiety disorder are 4–5% of the population. Gender differences have not been observed, although girls do present more often with anxiety disorders in general. Of those diagnosed with separation anxiety disorder, approximately 75% experience school refusal. The most frequently observed ages for occurrence of separation anxiety disorder are in children ages five to seven years and again from ages 11 to 14 years. It is at these times the children may feel more challenged by the developmental tasks of entering school or beginning puberty.

Diagnosis

The mental health professional will usually make the diagnosis of separation anxiety disorder based on information gathered during an interview process involving the parent(s) and the child. It is usually preferable for the interviews with the parent and child to occur separately; however that may not be possible because of the child's intense anxiety about separation.

Separation anxiety disorder is generally diagnosed by history, including parental report; however, a few measures of general anxiety exist that can be used to supplement the history. These include Pediatric Anxiety Rating Scale, Children's Global Assessment Scale, Children's Anxiety Scale, Screen for Child Anxiety Related Emotional Disorders (SCARED-R), Multi-

Dimensional Anxiety Scale for Children, and Achenbach's Child Behavior Checklist.

Duration of disturbance prior to diagnosis is a minimum of four weeks, occurring prior to the age of 18 years.

The disorder is described as "early onset" prior to the age of six years, and is generally not diagnosed after the age of 18. However, some researchers are describing another type of separation anxiety experienced by parents when their adolescents leave home. Readers may recognize this stage of life as the "empty nest syndrome"; however, no such formal diagnosis exists for a parental form of separation anxiety.

Treatments

The most effective treatments for separation anxiety disorder involve parents, as well as school personnel when appropriate. Giving the child a sense of safety and security is key to successful treatment. Current treatment methods combine some form of group or individual cognitive behavioral intervention. A number of treatment options are discussed below.

Cognitive-behavioral therapy

Cognitive-behavioral therapy is a treatment approach designed to alter a person's thoughts, beliefs, and images as a way of changing behavior. In treating a child with separation anxiety disorder, the goal is to help the child label her fears and identify the irrational beliefs and assumptions underlying her fears. By confronting and correcting her false beliefs, a parent can help his or her child become less anxious about separation.

Imagery

With imagery, a child uses his imagination to see himself being successful in a stressful situation. For example, before heading off to school, a child could imagine how he will handle separation from mom. Instead of crying, he sees himself calmly saying goodbye to his mom. The use of positive mental pictures may help diminish some of the child's anxiety and fear before separation actually occurs.

Modeling

Parents and teachers can be helpful in **modeling** appropriate behaviors and coping mechanisms at home and at school. For example, parents can model being relaxed when saying goodbye to their children and other people.

Systematic desensitization

Systematic desensitization is a **behavior modification** technique in which a person is gradually exposed to an anxiety-provoking or fearful object or situation while learning to be relaxed. A child with separation anxiety disorder may be taught relaxation techniques for managing her anxiety, and, as a result, can spend longer and longer periods of time at school without a caregiver present by teaching her.

Positive role models

Using positive role models, whether in real life or in books, can also be helpful for children. Reading books about other children successfully separating from their caregiver can give the anxious child the confidence that he can do it, too. Watching his friends calmly separate from their caregivers can also empower the child to do the same.

Behavior modification

Behavior modification uses a system of rewards and reinforcements to change behavior. This method has been shown to be effective in a majority of cases involving children and separation anxiety disorder, even at one-year follow-up.

Reminders

Small items that remind the child of his bond with his parents can sometimes be helpful in managing the child's anxiety. Typical objects could include a smooth stone in the child's pocket, a picture of the family in the child's notebook, or a friendship bracelet. Allowing phone calls or contact throughout the day is generally not effective, as it provides a more direct reminder of the caregiver's absence.

Distraction and altruism

Distraction and altruism is another strategy that can be useful in treating separation anxiety disorder. Helping the child focus on things outside himself can provide a healthy distraction. For instance, the child may be asked to take care of a pet at school. Such distractions from the child's internal thoughts and feelings coupled with a "fun" responsibility can help the child move away from his internal state of anxiety.

Medication management

Medication is helpful in certain cases where the anxiety is so debilitating that the child is unable to participate in other forms of treatment, or go about his daily routine. Medication management most often involves some type

of anti-anxiety or anti-depressive drug. The newest classes include the SSRIs or selective serotonin re-uptake inhibitors that influence **neurotransmitters** in the **brain** to regulate emotional response. Before any medication is given, however, it is essential that a careful medical and psychiatric evaluation be performed by a trained health professional.

Prognosis

Over 60% of children participating with their parents in cognitive-behavioral treatment are successful in managing their symptoms without medication. Symptoms generally do not re-appear in exactly the same way as the initial presentation; however, the child may have a heightened sensitivity to normal life transitions, such as changing schools. Families can help children cope with these transitions by visiting the new school, meeting teachers, and getting to know some students.

Separation anxiety disorder has a poorer prognosis in environments where threat of physical harm or separation actually exist.

Existence of other conditions, such as **autism**, decrease the likelihood of a positive prognosis. Presence of separation anxiety disorder in childhood is sometimes associated with early onset panic disorder in adults.

Studies indicate a lower prevalence of alcohol use and suicidal ideation in children or adolescents who experience separation anxiety disorder. Depression is commonly associated with anxiety disorders. Developing social skills can also be negatively affected by separation anxiety disorder.

Prevention

Prevention can be enhanced through parent effectiveness training that emphasizes the child's positive and successful coping strategies when dealing with separation. Overly anxious parents may need to develop their own support mechanisms and systems to manage their feelings and avoid influencing their children negatively.

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Serax *see* **Oxazepam**

Serentil *see* **Mesoridazine**

Seroquel *see* **Quetiapine**

Serotonin *see* **Neurotransmitters**

Sertraline

Definition

Sertraline is an antidepressant that belongs to the class of drugs called selective serotonin reuptake inhibitors (SSRIs). In the in United States it is sold under the brand name Zoloft.

Purpose

Sertraline is used to treat depression, **obsessive-compulsive disorder**, **panic disorder**, and **post-traumatic stress disorder**.

Description

Serotonin, one of the **neurotransmitters**, is a **brain** chemical that carries nerve impulses from one nerve cell to another. Researchers think that depression and certain other mental disorders may be caused, in part, because there is not enough serotonin being released and transmitted in the brain. Like the other SSRI antidepressants, **fluvoxamine** (Luvox), **fluoxetine** (Prozac), and **paroxetine** (Paxil), sertraline increases the level of brain serotonin (also known as 5-HT). Increased serotonin levels in the brain may be beneficial in patients with obsessive-compulsive disorder, alcoholism, certain types of headaches, post-traumatic stress disorder (PTSD), pre-menstrual tension and mood swings, and panic disorder. Sertraline is not more or less effective than the other SSRI drugs although selected characteristics of each drug in this class may offer greater benefits in some patients. Fewer drug interactions have been reported with sertraline, however, than with other medications in the same class.

The benefits of sertraline develop slowly over a period of up to four weeks. Patients should be aware of this and continue to take the drug as directed, even if they feel no immediate improvement.

Sertraline is available in 25-mg, 50-mg and 100-mg tablets, or as a 20-mg per ml solution.

Recommended dosage

The recommended dosage of sertraline depends on the disorder being treated. The initial recommended dosage for depression and obsessive-compulsive disorder is 50 mg daily. This may be increased at intervals of at least one week to the maximum recommended dosage of 200 mg daily. For the treatment of panic disorder and post-traumatic stress disorder, the initial dose is 25 mg once daily. This dosage is increased to 50 mg daily after one week. If there is no therapeutic response, the dosage may be increased to the maximum of 200 mg daily at intervals of at least one week. These dosages may need to be reduced in elderly patients (over age 65) or in people with liver disease.

For the treatment of obsessive-compulsive disorder in the pediatric population, treatment should be initiated at a dose of 25 mg per day in children six to 12 years of age and 50 mg per day in children 13 to 17 years of age. Doses may be increased at one-week intervals to a total daily dose of 200 mg.

KEY TERMS

Obsessive-compulsive disorder—Disorder in which the affected individual has an obsession (such as a fear of contamination, or thoughts he or she doesn't like to have and can't control) and feels compelled to perform a certain act to neutralize the obsession (such as repeated hand-washing).

Panic disorder—An anxiety disorder in which an individual experiences sudden, debilitating attacks of intense fear.

Post-traumatic stress disorder—A disorder caused by an extremely stressful or traumatic event (such as rape, act of war, or natural disaster) in which the trauma victim is haunted by flashbacks. In the flashbacks, the event is re-experienced in the present. Other symptoms include nightmares and feelings of anxiety.

Serotonin syndrome—A condition characterized by at least three of the following symptoms: diarrhea, fever, extreme perspiration, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking. It is a result of too much serotonin in the body.

Precautions

A group of serious side effects, called serotonin syndrome, have resulted from the combination of antidepressants such as sertraline and members of another class of antidepressants known as monoamine oxidase (MAO)

inhibitors. Serotonin syndrome usually consists of at least three of the following symptoms: diarrhea, fever, sweatiness, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking. Because of this, sertraline should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting sertraline or any other antidepressant. The same holds true when discontinuing sertraline and starting an MAO inhibitor. Also, people should not take sertraline oral concentrate while using **disulfiram** (Antabuse). Sertraline should never be taken by people who are any other SSRI antidepressants.

Sertraline should be used with cautiously and with close physician supervision by people with a prior history of **seizures**, people who are at an increased risk of bleeding, and those for whom weight loss is undesirable. Sertraline may precipitate a shift to mania in patients with bipolar (formerly manic-depressive) disease.

Side effects

More than 5% of patients experience **insomnia**, dizziness, and headache. About 14% of men report delayed ejaculation while 6% report decreased sex drive while taking this drug. In order to reduce these sexual side effects, patients can wait for tolerance to develop (this may take up to 12 weeks), reduce the dose, have drug holidays (where the weekend dose is either decreased or skipped), or discus with their physician using a different antidepressant.

More than 10% of patients report nausea and diarrhea while taking sertraline. Other possible side effects include agitation, anxiety, rash, constipation, vomiting, tremors, or visual difficulty. Although most side effects eventually subside, it may take up to four weeks for people to adjust to the drug.

Interactions

Sertraline interacts with **St. John's Wort**, an herbal remedy for depression. The risk of seizures is increased in patients using tramadol and sertraline. Taking sertraline with MAO inhibitors may result in the serious side effects discussed above. Erythromycin, an antibiotic, may inhibit the breakdown of sertraline in the liver and cause increased central nervous system effects such as drowsiness and decreasing of mental alertness. Other antidepressants should not be taken by people using sertraline except in rare cases when prescribed by a physician. If a combination of antidepressants is considered beneficial, a low dose of tricyclic antidepressants (10–25 mg daily) should be used.

Sertraline should not be taken with grapefruit juice as the combination may increase sertraline levels in the body.

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Serzone *see* **Nefazodone**

Sexual abuse *see* **Abuse**

Sexual aversion disorder

Definition

Sexual aversion disorder is a disorder characterized by disgust, fear, revulsion, or lack of desire in consensual relationships involving genital contact.

Normal loss of desire

To understand sexual aversion disorder, one should first understand that there are circumstances in which it is normal for people to lose interest in sexual activity. The reader can then compare these situations to the loss of desire associated with serious sexual disorders, including sexual aversion disorder.

There are a number of reasons that people lose interest in sexual intercourse. It is normal to experience a loss of desire during menopause; directly after the birth of a child; before or during menstruation; during recovery from an illness or surgery; and during such major or stressful life changes as death of a loved one, job loss, retirement, or divorce. These are considered normal causes for fluctuations in sexual desire and are generally temporary. Changing roles, such as becoming a parent for the

first time or making a career change have also been found to cause loss of desire. Not having enough time for oneself or to be alone with one's partner may also contribute to normal and naturally reversible loss of desire. Loss of privacy resulting from moving a dependent elderly parent into one's home is a common cause of loss of desire in middle-aged couples. Depression, **fatigue**, or **stress** also contribute to lessening of sexual interest.

Description

Sexual aversion disorder represents a much stronger dislike of and active avoidance of sexual activity than the normal ups and downs in desire described above. Sexual aversion disorder is characterized not only by a lack of desire, but also by fear, revulsion, disgust, or similar emotions when the person with the disorder engages in genital contact with a partner. The aversion may take a number of different forms; it may be related to specific aspects of sexual intercourse, such as the sight of the partner's genitals or the smell of his or her body secretions, but it may include kissing, hugging, and petting as well as intercourse itself. In some cases the person with sexual aversion disorder avoids any form of sexual contact; others, however, are not upset by kissing and caressing, and are able to proceed normally until genital contact occurs.

There are several subclassifications of sexual aversion disorder. It may be lifelong (always present) or acquired after a traumatic experience; situational (with a specific partner or in a specific set of circumstances) or generalized (occurring with any partner and in all situations). Sexual aversion may be caused by psychological factors or by a combination of physical and psychological factors.

Causes and symptoms

There are a number of causes of sexual aversion disorder. The most common causes are interpersonal problems and traumatic experiences. Interpersonal problems generally cause situation-specific sexual aversion disorder, in which the symptoms occur only with a specific partner or under certain conditions. In such cases, underlying tension or discontent with the relationship is often the cause. Reasons for unhappiness with the relationship may include the discovery of marital infidelity; major disagreements over children, money, and family roles; domestic violence; lack of personal hygiene on the partner's side; or similar problems. Interpersonal problems are often the cause if intercourse was once enjoyed but is no longer desired.

Traumatic experiences have also been found to cause sexual aversion disorder, often of the generalized variety.

KEY TERMS

Coitus—Sexual intercourse.

Some possible traumas include rape, incest, molestation, or other forms of sexual **abuse**. The patient then associates intercourse with a painful experience or memory, possibly one that he or she is trying to forget. Sexual aversion disorder may also be caused by religious or cultural teachings that associate sexual activity with excessive feelings of guilt.

The symptoms of sexual aversion disorder can range from mild to severe. Mild symptoms include lack of interest and mild disgust. Severe symptoms can include panic attacks with all the symptoms of such an attack, including dizziness, shortness of breath, intense fear, and rapid heartbeat. People suffering from sexual aversion disorder often go out of their way to avoid situations that could end in sexual contact through any means they can think of, including going to bed at different times from the spouse, spending extra time at work, or trying to make themselves less sexually attractive.

Demographics

Both men and women can experience sexual aversion disorder. It is thought to be more common in women than in men, possibly because women are more likely than men to be victims of rape and other forms of sexual assault. There are relatively few statistics on the number of people with sexual aversion disorder because it is often confused with other disorders, or with the normal fluctuations in desire associated with life stress. Also, many people find sex a difficult subject to discuss even with a physician, so that the number of people who seek help are probably fewer than the number of people with the disorder overall.

Diagnosis

A **diagnosis** of sexual aversion disorder is usually made when the affected person or his or her partner mentions the problem itself or their dissatisfaction with the relationship to their family physician, gynecologist, or psychotherapist. An important first step in diagnosis is a thorough physical examination, preferably of both partners, to rule out physical causes of the disorder in the affected person, and to rule out a sexually transmitted disease, physical deformity, or lack of personal cleanliness in the partner that may contribute to the affected person's avoidance of sex.

According to the mental health professional's ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*) of the American Psychiatric Association, to meet criteria for a diagnosis of sexual aversion disorder the patient must not only avoid nearly all genital contact with his or her partner, but have strong negative feelings about such contact or its possibility. In addition, the problem must be causing serious difficulties and unhappiness either for the patient or for his or her partner. In addition, there must not be any underlying physical causes, such as certain disorders of the circulatory system, skin diseases, medication side effects, or similar problems that could cause a loss of desire. To be diagnosed with sexual aversion disorder, the affected person does not have to avoid all sexual contact, but must indicate that he or she is actively avoiding genital contact.

Many other sexual disorders have signs and symptoms similar to those of sexual aversion disorder, which complicates the diagnosis. Sexual aversion disorder is often found in conjunction with other sexual disorders; in some cases several diagnoses are appropriate for one patient.

One disorder similar in many aspects to sexual aversion disorder is hypoactive sexual disorder. Many of the signs, such as avoiding sexual contact in a variety of ways, are similar. The primary difference between the two disorders is that a patient with hypoactive sexual disorder is not interested in sex at all and does not have sexual fantasies of any variety. A patient with sexual aversion disorder, by comparison, may have normal sexual fantasies, and even function normally with some partners, although not with a specific partner. Also, a patient with hyposexual disorder will not enjoy or desire any anticipation in sexual activities including kissing and caressing. Some, though not all, people with sexual aversion disorder do enjoy sexual foreplay until the point of genital contact.

Sexual aversion disorder and hypoactive sexual disorder are both considered to be caused mainly by psychological factors and to manifest psychological symptoms. Another disorder that can have some similar symptoms is **female sexual arousal disorder** (FSAD). FSAD refers to a woman's recurrent inability to achieve or maintain an adequate lubrication-swelling response during sexual activity. Lack of lubrication is a physical problem that may have either physical or psychological causes. Women with FSAD find intercourse uncomfortable or even painful. As a result of the physical discomfort, the woman often will avoid intercourse and sexual activity with her partner that may lead to intercourse. Although FSAD is a disorder with physical symptoms as well as psychological ones, it is easily confused with sexual aversion disorder because it may manifest as a problem of interest or desire.

Treatments

Sexual aversion disorder is not thought to have any commonplace underlying physiological causes. The usual treatment is a course of **psychotherapy** for the psychological condition(s) that may be causing the problem. Marriage counseling, or **couples counseling**, is often appropriate if the disorder concerns a spouse. Medications can be used to treat some symptoms that may be associated with sexual aversion disorder, such as panic attacks, if they are severe enough to be causing additional distress.

Prognosis

When sexual aversion disorder is addressed as a psychological disorder, treatment can be very successful. Psychotherapy to treat the underlying psychological problems can be successful as long as the patient is willing to attend counseling sessions regularly. For sexual aversion disorder that is situational or acquired, psychotherapy for both the patient and his or her partner may help to resolve interpersonal conflicts that may be contributing to the disorder. Panic attacks caused by or associated with the disorder can be treated successfully by medication if the doctor considers this form of treatment necessary.

If sexual aversion disorder is not diagnosed, discussed, or treated, the result may be infidelity, divorce, or chronic unhappiness in the relationship or marriage.

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Tish Davidson, A.M.

Sexual deviance *see* **Paraphilias**

Sexual dysfunctions

Definition

Sexual dysfunction disorders are problems that interfere with the initiation, consummation, or satisfaction with sex. They occur in both men and women and are independent of sexual orientation.

Description

Probably nowhere in human health do the body and mind interact more than during sex. There are four generally recognized phases of sexual activity, involving both mental and physical responses and are applicable to both men and women. These phases are in sequence:

- desire or appetite—fantasies or thoughts about sex.
- excitement—physical changes to prepare the body for intercourse and accompanying sense of sexual pleasure
- orgasm—physical response that leads to the peak of physical pleasure and release of sexual tension
- resolution—physical relaxation accompanied by a feeling of well-being and satisfaction

Sexual dysfunction disorders can occur in any of these four phases. Their cause may be physiological or psychological. More than one sexual dysfunction disorder may appear simultaneously. The ***Diagnostic and Statistical Manual of Mental Disorders***, (*DSM-IV-TR*), produced by the American Psychiatric Association and used by most mental health professionals in North America and Europe to diagnose mental disorders, recognizes nine specific sexual dysfunctions:

- Disorders of desire: These interfere with the initiation of sex and include **hypoactive sexual desire disorder** (low interest in sex) and **sexual aversion disorder** (objections to having the genitals touched).
- Disorders of excitement or sexual arousal: These are **female sexual arousal disorder** (when a woman fails to have physiological responses associated with arousal), and male erectile disorder (when a man fails to get an adequate erection, also referred to as "**erectile dysfunction**").
- Disorders of the orgasm phase: These are **female orgasmic disorder** (when a woman fails to reach orgasm); and **male orgasmic disorder** (when a man fails to reach orgasm) and **premature ejaculation** (when a man reaches orgasm too soon).
- Sexual pain disorders (associated with intercourse and orgasm): These disorders are **vaginismus** (the outer part of a woman's vagina spasms causing pain) and **dyspareunia** (pain during intercourse in either men or women).

In addition, medications or illicit drugs may cause substance-induced sexual dysfunction and sexual dysfunction may be caused by a general medical condition such as diabetes or nerve damage. If the sexual dysfunction falls into none of the above areas, it is classified as sexual dysfunction not otherwise specified.

The causes of sexual dysfunction disorders are varied, as are their symptoms. In general, symptoms either prevent the initiation of sex or the completion of the sex act, or they interfere with satisfaction derived from sex. Almost everyone has some problem with sexual functioning or fulfillment at some point in their lives, but not all problems are considered sexual dysfunction disorders. Sexual satisfaction is very personal and individual, so that what may be an annoyance for one couple may be a serious problem for another. However, estimates suggest that roughly one-fourth of the adult population may have a sexual dysfunction disorder. More women than men report having sexual dysfunction disorders, but the difference may be that women are more open and active about seeking help with sexual problems than are men.

Diagnosis begins with a sexual and medical history, and often a physical examination and laboratory tests. Treatment must be individualized based on the cause and the specific dysfunction and includes physiological treatment, **psychotherapy**, and education and communication counseling. Most people can be helped to resolve their problems and improve their sex life. Generally, the sooner the person receives help, the easier the problem is to resolve. Support of a partner is often critical to successful resolution of the problem.

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ORGANIZATIONS

American Association of Sex Educators, Counselors, and Therapists (AASECT). P. O. Box 238, Mount Vernon, IA 53214-0238. (319) 895-8407. <www.aasect.org>.

Sexual Information and Education Council of the United States (SIECUS). West 42 Street, Suite 350, New York, NY 10036-7802. <www.siecus.org>.

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Sexual masochism

Definition

The essential feature of sexual masochism is the feeling of sexual arousal or excitement resulting from receiving pain, suffering, or humiliation. The pain, suffering, or humiliation is real and not imagined and can be physical or psychological in nature. A person with a **diagnosis** of sexual masochism is sometimes called a masochist.

The ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM*, is used by mental health professionals to diagnose specific mental disorders. In the 2000 edition of this manual (the Fourth Edition Text Revision also known as *DSM-IV-TR*) sexual masochism is one of several **paraphilias**. Paraphilias are intense and recurrent sexually arousing urges, fantasies, or behaviors.

Description

In addition to the sexual pleasure or excitement derived from receiving pain and humiliation, an individual with sexual masochism often experiences significant impairment or distress in functioning due to masochistic behaviors or fantasies.

With regard to actual masochistic behavior, the person may be receiving the pain, suffering, or humiliation at the hands of another person. This partner may have a diagnosis of **sexual sadism** but this is not necessarily the case. Such behavior involving a partner is sometimes referred to as sadomasochism.

Masochistic acts include being physically restrained through the use of handcuffs, cages, chains, and ropes. Other acts and fantasies related to sexual masochism include receiving punishment or pain by means of paddling, spanking, whipping, burning, beating, electrical shocks, cutting, rape, and mutilation. Psychological humiliation and degradation can also be involved.

Masochistic behavior can also occur in the context of a role-playing fantasy. For example, a sadist can play the role of teacher or master and a masochist can play the role of student or slave.

The person with sexual masochism may also be inflicting the pain or suffering on himself or herself. This can be done through self-mutilation, cutting, or burning.

The masochistic acts experienced or fantasized by the person sometimes reflect a sexual or psychological submission on the part of the masochist. These acts can range from relatively safe behaviors to very physically and psychologically dangerous behavior.

The *DSM* lists one particularly dangerous and deadly form of sexual masochism called hypoxyphilia. People with hypoxyphilia experience sexual arousal by being deprived of oxygen. The deprivation can be caused by chest compression, noose, plastic bag, mask, or other means and can be administered by another person or be self-inflicted.

Causes and symptoms

Causes

There is no universally accepted cause or theory explaining the origin of sexual masochism, or sadomasochism in general. However, there are some theories that attempt to explain the presence of sexual paraphilias in general. One theory is based on learning theory that paraphilias originate because inappropriate sexual fantasies are suppressed. Because they are not acted upon initially, the urge to carry out the fantasies increases and when they are finally acted upon, a person is in a state of considerable distress and/or arousal. In the case of sexual masochism, masochistic behavior becomes associated with and inextricably linked to sexual behavior.

There is also a belief that masochistic individuals truly want to be in the dominating role. This causes them to become conflicted and thus submissive to others.

Another theory suggests that people seek out sadomasochistic behavior as a means of escape. They get to act out fantasies and become new and different people.

Symptoms

Individuals with sexual masochism experience sexual excitement from physically or psychologically receiving pain, suffering, and/or humiliation. They may be receiving the pain, suffering, or humiliation at the hands of another person, who may or may not be a sadist, or they may be administering the pain, suffering, or humiliation themselves.

They experience distressed or impaired functioning because of the masochistic behaviors, urges, and fantasies. This distress or impairment can impact functioning in social, occupational, or other contexts.

Demographics

Although masochistic sexual fantasies often begin in childhood, the onset of sexual masochism typically occurs during early adulthood. When actual masochistic behavior begins, it will often continue on a chronic course for people with this disorder, especially when no treatment is sought.

Sadomasochism involving consenting partners is not considered rare or unusual in the United States. It often occurs outside of the realm of a mental disorder. More people consider themselves masochistic than sadistic.

Sexual masochism is slightly more prevalent in males than in females.

Death due to hypoxyphilia is a relatively rare phenomenon. Data indicate that less than two people per million in the United States and other countries die from hypoxyphilia.

Diagnosis

The *DSM* criteria for sexual masochism include recurrent intense sexual fantasies, urges, or behaviors involving real acts in which the individual with the disorder is receiving psychological or physical suffering, pain, and humiliation. The suffering, pain, and humiliation cause the person with sexual masochism to be sexually aroused. The fantasies, urges, or behaviors must be present for at least six months.

The diagnostic criteria also require that the person has experienced significant distress or impairment because of these behaviors, urges, or fantasies. The distress and impairment can be present in social, occupational, or other functioning.

Sexual masochism must be differentiated from normal sexual arousal, behavior, and experimentation. It should also be differentiated from sadomasochistic behavior involving mild pain and/or the simulation of more dangerous pain. When this is the case, a diagnosis of sexual masochism is not necessarily warranted.

Sexual masochism must also be differentiated from self-defeating or self-mutilating behavior that is performed for reasons other than sexual arousal.

Individuals with sexual masochism often have other sexual disorders or paraphilias. Some individuals, especially males, have diagnoses of both sexual sadism and sexual masochism.

Treatments

Behavior therapy is often used to treat paraphilias. This can include management and conditioning of arousal patterns and masturbation. Therapies involving cognitive restructuring and **social skills training** are also utilized.

Medication is also used to reduce fantasies and behavior relating to paraphilias. This is especially true of people who exhibit severely dangerous masochistic behaviors.

Treatment can also be complicated by health problems relating to sexual behavior. Sexually transmitted diseases and other medical problems, especially when the sadomasochistic behavior involves the release of blood, can be present. Also, people participating in hypoxyphilia and other dangerous behaviors can suffer extreme pain and even death.

Prognosis

Because of the chronic course of sexual masochism and the uncertainty of its causes, treatment is often difficult. The fact that many masochistic fantasies are socially unacceptable or unusual leads some people who may have the disorder not to seek or continue treatment.

Treating a paraphilia is often a sensitive subject for many mental health professionals. Severe or difficult cases of sexual masochism should be referred to professionals who have experience treating such cases.

Prevention

Because it is sometimes unclear whether sadomasochistic behavior is within the realm of normal experimentation or indicative of a diagnosis of sexual masochism, prevention is a tricky issue. Often, prevention refers to managing sadomasochistic behavior so it primarily involves only the simulation of severe pain and it always involves consenting partners familiar with each other's limitations.

Also, because fantasies and urges originating in childhood or adolescence may form the basis for sadomasochistic behavior in adulthood, prevention is made difficult. People may be very unwilling to divulge their urges and discuss their sadistic fantasies as part of treatment.

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Ali Fahmy, Ph.D.

Sexual sadism

Definition

The essential feature of sexual sadism is a feeling of sexual excitement resulting from administering pain, suffering, or humiliation to another person. The pain, suffering, or humiliation inflicted on the other is real; it is not imagined and may be either physical or psychological in nature. A person with a **diagnosis** of sexual sadism is sometimes called a sadist. The name of the disorder is derived from the proper name of the Marquis Donatien de Sade (1740-1814), a French aristocrat who became notorious for writing novels around the theme of inflicting pain as a source of sexual pleasure.

The ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM*, is used by mental health professionals to give diagnoses of specific mental disorders. In the 2000 edition of this manual— the Fourth Edition, Text Revision, also known as *DSM-IV-TR*— sexual sadism is listed as one of several **paraphilias**. The paraphilias are a group of mental disorders characterized by **obsession** with unusual sexual practices or with sexual activity involving nonconsenting or inappropriate partners (such as children or animals). The paraphilias may include recurrent sexually arousing urges or fantasies as well as actual behaviors.

Description

In addition to the sexual pleasure or excitement derived from inflicting pain and humiliation on another, a person diagnosed with sexual sadism often experiences significant impairment or distress in functioning due to actual sadistic behaviors or sadistic fantasies.

With regard to actual sadistic behavior, the person receiving the pain, suffering, or humiliation may or may not be a willing partner. Whether or not the partner is consenting, it is the very real suffering they are experiencing that is arousing to the sadist. When the sexual activity is consensual, the behavior is sometimes referred to as sadomasochism. The consenting partner may be given a diagnosis of **sexual masochism**. Like sadism, masochism is a term derived from a proper name; in this instance, from Leopold von Sacher-Masoch (1836-1895), an Austrian novelist who described the disorder in his books.

The sadistic acts performed or fantasized by a person with sadism often reflect a desire for sexual or psychological domination of another person. These acts range from behavior that is not physically harmful although it may be humiliating to the other person (such as being urinated upon), to criminal and potentially deadly behavior. Acts of domination may include restraining or imprisoning the partner through the use of handcuffs, cages, chains, or ropes. Other acts and fantasies related to sexual sadism include paddling, spanking, whipping, burning, beating, administering electrical shocks, biting, urinating or defecating on the other person, cutting, rape, murder, and mutilation.

In extreme cases, sexual sadism can lead to serious injury or death for the other person. According to the *DSM* these catastrophic results are more likely when the paraphilia is diagnosed as severe, and when it is associated with **antisocial personality disorder**, a personality disorder that may include psychotic symptoms.

Causes and symptoms

Causes

There is no universally accepted cause or theory explaining the origin of sexual sadism, or of sadomasochism. Some researchers attempt to explain the presence of sexual paraphilias in general as the result of biological factors. Evidence for this viewpoint comes from abnormal findings from neuropsychological and neurological tests of sex offenders.

Some researchers believe that paraphilias are related to such other problems as **brain** injury, **schizophrenia**, or another mental disorder. Often, people with sexual disorders or symptoms of paraphilia are diagnosed with other mental disorders.

Another theory about paraphilias is derived from learning theory. It suggests that paraphilias develop because the person is required to suppress, or squelch, inappropriate sexual fantasies. Because the fantasies are not acted out initially, the urge to carry them out increases. When the person finally acts upon the fantasies, they are in a state of considerable distress and/or arousal. This theory is not accepted by forensic experts at the Federal Bureau of Investigation (FBI) and other researchers who study sexual offenses. Rather than suppressing fantasies, most people who are eventually arrested for crimes involving sexual sadism begin with milder forms of acting on them and progressing to more harmful ways of

acting out. For example, the FBI's database indicates that these people— almost always males— start out by collecting pornographic materials that depict sadistic acts, or they may draw ropes and chains on the photographs of models in swimsuit or lingerie advertisements. They then typically progress to following women at a distance, to hiring a prostitute in order to act out the fantasy, and to asking a girlfriend or other willing partner to cooperate with their fantasy. In other words, the severity of sadistic acts tends to increase over time.

Symptoms

Individuals with sexual sadism derive sexual excitement from physically or psychologically administering pain, suffering, and/or humiliation to another person, who may or may not be a consenting partner.

They may experience distressed or impaired functioning because of the sadistic behaviors or fantasies. This distress or impairment may be due to the fact that the partner is not consenting.

Demographics

Although sadistic sexual fantasies often begin in the person's childhood, the onset of active sexual sadism typically occurs during early adult life. When actual sadistic behavior begins, it will often continue on a chronic course for people with this disorder, especially if they do not seek help.

Sexual sadism with consenting partners is much more common than with nonconsensual partners. When consenting partners are involved, the sadist and the masochist may be either male or female. When non-consenting partners are involved, the sadist is almost always a male.

Sadomasochism involving consenting partners is not considered rare or unusual in the United States. It often occurs outside of the realm of a mental disorder. Fewer people consider themselves sadistic than masochistic.

Diagnosis

The diagnosis of sexual sadism is complicated by several factors, beginning with the fact that most persons with the disorder do not enter therapy voluntarily. Some are referred to treatment by a court order. Some are motivated by fear of discovery by employers or family members, and a minority enter therapy because their wife or girl friend is distressed by the disorder. The diagnosis of sexual sadism is based on the results of a psychiatrist's interview with the patient. In some cases, a person with sexual sadism may be referred to a specialized clinic for the treatment of sexual disorders. In the clinic, he will be given questionnaires intended to measure the presence and extent of cognitive distortions regarding rape and other forms of coercion, aggression, and impulsivity.

DSM-IV-TR criteria for sexual sadism include recurrent intense sexual fantasies, urges, or behaviors involving real acts in which another person is suffering psychological or physical suffering, pain, and humiliation. The victim's suffering, pain, and humiliation cause the person with sexual sadism to become aroused. The fantasies, urges, or behaviors must be present for at least six months.

The diagnostic criteria also require either that the person has acted on these urges or fantasies with a non-consenting person, or that the person has experienced noticeable distress or interpersonal problems because of these urges or fantasies.

Sexual sadism must be differentiated from normal sexual arousal, behavior, and experimentation. Some forms of mild aggression, such as "love bites" or scratching, are within the range of normal behavior during sexual intercourse. Sadism should also be differentiated

KEY TERMS

Forensic—Pertaining to courtroom procedure or evidence used in courts of law.

Masochism—A mental disorder in which a person obtains sexual satisfaction through pain or humiliation inflicted by the self or by another person. The term is sometimes used more generally to refer to a tendency to find pleasure in submissiveness or self-denial.

Medroxyprogesterone acetate (MPA)—A female hormone that may be prescribed for male patients with sexual sadism or other paraphilias. MPA helps to control sexual urges in men by speeding up the clearance of testosterone from the bloodstream.

Paraphilias—A group of mental disorders that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Sadism—A mental disorder in which sexual arousal and gratification are obtained by inflicting pain or humiliation on another person.

from sadomasochistic behavior that involves only mild pain and/or the simulation of more dangerous pain. When these factors are present, a diagnosis of sexual sadism is not necessarily warranted.

Other mental disorders, such as the psychotic disorders, may include elements of sadism or other paraphilias. For example, patients with psychotic symptoms may perform sadistic acts for reasons other than sexual excitement. In these cases, an additional diagnosis of sexual sadism is not warranted.

Persons diagnosed with sexual sadism may have other sexual disorders or paraphilias. Some individuals, especially males, have diagnoses of both sexual sadism and sexual masochism.

Treatments

Behavior therapy is often used to treat paraphilias. This approach to treatment may include the management and conditioning of arousal patterns and masturbation. Therapies involving cognitive restructuring and **social skills training** are also often utilized.

Medication may be used to reduce fantasies and behavior relating to paraphilias. This form of treatment is especially recommended for people who exhibit sadistic behaviors that are dangerous to others. The medications that may be used include female hormones (most commonly medroxyprogesterone acetate, or MPA), which speed up the clearance of testosterone from the bloodstream; antiandrogen medications, which block the body's uptake of testosterone; and the selective serotonin reuptake inhibitors, or SSRIs.

Nonconsensual sadistic behavior often leads to problems with the criminal justice system. Issues related to legal problems may impair or delay the patient's treatment. Persons with sexual sadism may be reluctant to seek or continue treatment because they fear being reported to the police or being named in a lawsuit by an unwilling partner.

Treatment of sexual sadism may also be complicated by health problems related to sexual behavior. Sexually transmitted diseases and other medical problems may be present, especially when the sadistic behavior involves the release of blood or other body fluids.

Prognosis

Because of the chronic course of sexual sadism and the uncertainty of its causes, treatment is often difficult. The fact that many sadistic fantasies are socially unacceptable or unusual leads many people who may have the disorder to avoid or drop out of treatment. Treating a paraphilia is often a sensitive subject for many mental health professionals. Severe or difficult cases of sexual sadism should be referred to a specialized clinic for the treatment of sexual disorders or to professionals with experience in treating such cases.

As was noted previously, acts of sexual sadism tend to grow more violent or bizarre over time. As males with the disorder grow older, however, their ability to commit such acts begins to decrease. Sexual sadism is rarely diagnosed in men over 50.

Prevention

Because it is sometimes unclear whether sadomasochistic behavior is within the realm of normal experimentation or indicative of a diagnosis of sexual sadism, prevention is a tricky issue. Often, prevention refers to managing sadistic behavior so it never involves non-consenting individuals and it primarily involves the simulation of pain and not real pain.

Also, because fantasies and urges originating in childhood or adolescence may form the basis for sadomasochistic behavior in adulthood, prevention is made difficult. People may be very unwilling to divulge their urges and discuss their sadistic fantasies.

See also Cognitive-behavioral therapy; Sexual masochism; Sexual Violence Risk-20

Resources

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Sexual Violence Risk-20

Definition

The Sexual Violence Risk-20, also called the SVR-20, is an assessment instrument used by mental health professionals.

Purpose

The SVR-20 provides a structure for reviewing information important in characterizing an individual's risk of committing sexual violence and for targeting plans to manage that risk. The instrument's authors define sexual violence as, "Actual, attempted or threatened sexual contact with a person who is non-consenting or unable to give consent."

Precautions

SVR-20 results should be finalized and interpreted by a professional who is familiar with the scientific literature on sexual violence, and who is experienced in conducting individual assessments on sexual and violent offenders. The instrument is not capable of providing new information about past behavior or of profiling an examinee as a sexually violent offender. Rather, it helps provide a structure to follow in estimating risk of sexual violence under certain circumstances. The instrument should not be used as a stand-alone measure, and predictions derived from its use should be subject to critical review. It is especially important to place results in the contexts of the examinee's personal style, likely environmental conditions, and base rates of sexual violence in other offenders with similar characteristics.

Description

The SVR-20 is a tool that helps guide a professional in conducting a minimally comprehensive assessment of sexual violence risk. The assessment process is based on six principles:

- It is important to gather a depth of information about the examinee's personal, social, occupational, mental health, illegal, and other relevant behavior.
- Information should be gathered using a variety of sources and methods, including (and not limited to) record reviews, interviews, and psychological, physiological, and medical techniques.
- Information should be gathered from the examinee, his or her relatives and acquaintances, the victim(s), professionals who have interacted with the examinee, and any other sources likely to yield useful information.

KEY TERMS

High-density sex offenses—Several offenses within a short period of time.

Risk assessment—The process of gathering and interpreting data useful in estimating the probability that an individual will demonstrate sexual violence.

Risk management—Using the results of a risk assessment to tailor intervention strategies intended to reduce the likelihood that an individual will demonstrate sexual violence.

Sexual violence—Actual, attempted or threatened sexual contact with a person who is non-consenting or unable to give consent.

- The examinee's history and future exposure to risk factors should be considered.
- The examiner should critically weigh the accuracy, credibility and applicability of the data that has been gathered.
- The risk assessment process should be ongoing, with regular re-assessments for many examinees.

The content of the SVR-20 was developed following a comprehensive review of similar instruments and of the scientific literature on risk for sexual violence and re-offense. The SVR-20 materials consist of a reference manual and protocol sheets that are filled out by the examiner. The instrument includes three major sections: Psychosocial Adjustment, Sexual Offenses, and Future Plans. The SVR-20 items are coded based on presence (Yes or No) and if present, whether there has been a recent change in status regarding that factor (Exacerbation, No Change, Amelioration).

The Psychosocial Adjustment section includes 11 risk factors: sexual deviation, victim of child **abuse**, psychopathy, major mental illness, substance use problems, suicidal/homicidal ideation (ideas), relationship problems, employment problems, past non-sexual violent offenses, past non-violent offenses, and past supervision failure.

The Sexual Offenses section includes seven risk factors: high-density sex offenses, multiple sex offense types, physical harm to victim(s) in sex offenses, escalation in frequency and severity of sex offenses, extreme minimization or **denial** of sex offenses, and attitudes that support or condone sex offenses.

The Future Plans section includes two factors: lacks realistic plans, and negative attitude toward **intervention**. Aside from factors related to the examinee's thinking and personality, items found in the first and second sections reference fixed or relatively stable characteristics.

The first and third sections are relevant not only to sexual violence, but also to violence in general. There is also an unstructured supplementary section entitled "Other Considerations," that can be used to describe unique factors relevant to an examinee's probability of risk.

Results

The SVR-20 does not allow for the definite prediction of sexual violence. Prediction of risk is summarized using a rating of low, moderate or high. Although the instrument's authors did not provide decision-making guidelines for determining the appropriate rating, they did recommend five questions to consider in communicating a "Risk Message" derived from the results:

- What is the likelihood that the individual will engage in sexual violence, if no efforts are made to manage the risk?
- What is the probable nature, frequency, and severity of any future sexual violence?
- Who are the likely victims of any future sexual violence?
- What steps could be taken to manage the individual's risk for sexual violence?
- What circumstances might exacerbate the individual's risk for sexual violence?

Typically, answers to these and other questions are fashioned in the form of a report for those responsible for making decisions about the examinee.

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Association for the Treatment of Sexual Offenders. 4900 S.W. Griffith Drive, Suite 274, Beaverton, Oregon 97005. Telephone: (503) 643-1023. <http://www.atsa.com>.

British Columbia Institute Against Family Violence, 409 Granville Street, Suite 551, Vancouver, BC V6C 1T2. Telephone: (604) 669-7055 or Toll free (Canada): (877) 755-7055. <http://www.bcifv.org>.

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Shared psychotic disorder

Definition

Shared psychotic disorder, a rare and atypical psychotic disorder, occurs when an otherwise healthy person (secondary partner) begins believing the **delusions** of someone with whom they have a close relationship (primary partner) who is already suffering from a psychotic disorder with prominent delusions. This disorder is also referred to as "folie á deux."

Description

In cases of shared psychotic disorder, the primary partner is most often in a position of strong influence over the other person. This allows them, over time, to erode the defenses of the secondary partner, forcing their strange belief upon them. In the beginning, the secondary partner is probably healthy, but has such a passive or dependent relationship with the primary partner that imposition of the delusional system is but a matter of time. Most of the time, this disorder occurs in a nuclear family. In fact, more than 95% of the cases reported involved people in the same family. Without regard to the number of persons within the family, shared delusions generally involve two people. There is the primary, most often the dominant person, and the secondary or submissive person. This becomes fertile ground for the primary (dominant) partner to press for understanding and belief by others in the family.

Shared psychotic disorder has also been referred to by other names such as **psychosis** of association, contagious insanity, infectious insanity, double insanity, and communicated insanity. There have been cases

involving multiple persons, the most significant being a case involving an entire family of 12 people (folie á douze).

Causes and symptoms

Causes

Given the fact that the preponderance of cases occur within the same family, the theory about the origins of the disorder come from a psychosocial perspective. Approximately 55% of secondary cases of the disorder have first-degree relatives with psychiatric disorders, not including the primary partner. This is not true of individuals with the primary **diagnosis**, as they showed a roughly 35% incidence.

There are several variables which have great influence on the creation of shared psychotic disorder. For example, family isolation, closeness of the relationship to the person with the primary diagnosis, the length of time the relationship has existed, and the existence of a dominant-submissive factor within the relationship. The submissive partner in the relationship may be predisposed to have a mental disorder. Often the submissive partner meets the criteria for **dependent personality disorder**. Nearly 75% of the delusions are of the persecutory type.

An example of shared psychotic disorder involving the delusion of persecution, is that of a 52-year-old married female and her 48-year-old husband with multiple sclerosis, who believed that they were being harassed and watched by the Irish Republican Army (IRA). They were hospitalized and both became stable after two weeks on an antipsychotic medication. However, an interesting point in this case is that they were separated for that two-week period. The general consensus has been that, once separated, the submissive partner will let go of the delusion, that it would resolve itself simply due to separation. That did not happen in this case. Both partners had to be treated with proper medications before the delusion resolved.

In a case involving a middle-aged mother and an adolescent daughter, the delusions were multifaceted. The mother held the persecutory belief that someone in her neighborhood was manufacturing illegal drugs of some sort, and that they were periodically spraying something odorless, tasteless, and invisible into the air. The sprayed substance made her and her teen-aged daughter "act crazy." Oddly enough, the effects of the spraying began shortly after the husband left for work in the morning, and resolved shortly before he returned in the afternoon. The family raised ducks at their home, and the mother and daughter believed that the men making the illegal drugs were using the family ducks "as a food source" in order to stay near their hideout and avoid detection by police. Finally, mother and daughter also believed that occasional gunshots in their countryside landscape were meant as warnings to prevent anyone from learning about the misdeeds of the drug makers. This case was revealed when the daughter ran away from home, fearing that men with guns were coming to kill them. She was subsequently placed in the care of a child protective services agency, and the bizarre stories began to unfold. Both mother and daughter received psychiatric care.

KEY TERMS

Delusion—A false belief that is resistant to reason or contrary to actual fact.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Symptoms

The principal feature of shared psychotic disorder is the unwavering belief by the secondary partner in the dominant partner's delusion. The delusions experienced by both primary partners in shared psychotic disorder are far less bizarre than those found in schizophrenic patients; they are, therefore, believable. Since these delusions are often within the realm of possibility, it is easier for the dominant partner to impose his/her idea upon the submissive, secondary partner.

Demographics

Little data is available to determine the prevalence of shared psychotic disorder. While it has been argued that some cases go undiagnosed, it is nevertheless a rare finding in clinical settings.

Diagnosis

A clinical interview is required to diagnose shared psychotic disorder. There are basically three symptoms required for the determination of the existence of this disorder:

- An otherwise healthy person, in a close relationship with someone who already has an established delusion, develops a delusion himself/herself.

- The content of the shared delusion follows exactly or closely resembles that of the established delusion.
- Some other psychotic disorder, such as **schizophrenia**, is not in place and cannot better account for the delusion manifested by the secondary partner.

Treatments

The treatment approach most recommended is to separate the secondary partner from the source of the delusion. If symptoms have not dissipated within one to two weeks, antipsychotic medications may be in order.

Once stabilized, **psychotherapy** should be undertaken with the secondary partner, with an eye toward integrating the dominant partner, once he/she has also received medical treatment and is stable.

Prognosis

If the secondary partner is removed from the source of the delusion and proper medical and psychotherapeutic treatment are rendered, the prognosis is good. However, as stated above, the separation alone may not be successful. The secondary partner may require antipsychotic medication. Even after treatment, since this shared psychotic disorder is primarily found in families, the family members tend to reunite following treatment and release. If family dynamics return to pretreatment modes, a relapse could occur. Periodic monitoring by a social services agency is advised for as long as a year following treatment.

Prevention

In an effort to prevent relapse, **family therapy** should also be considered to re-establish the nuclear family and to provide social support to modify old family dynamics. The family cannot continue in isolation as it did in the past, and will require support from community agencies.

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Simple phobia *see* **Specific phobias**

Sinequan *see* **Doxepin**

Single photon emission computed tomography

Definition

Single photon emission **computed tomography** (SPECT) is an imaging study that uses radioactive materials injected through a vein that will pass into the **brain** generating a high-resolution brain image.

Description

SPECT is used to diagnose head trauma, epilepsy, **dementia**, and cerebrovascular disease. Development of a radiotracer called Tc99m label has increased the resolution of brain images generated from SPECT. The images yield very accurate spatial and contrast resolutions. The resulting sharp images enable the clinician to visualize very small structures within the brain. The accuracy of SPECT brain images makes it a very useful clinical and research tool.

Clinically, SPECT is useful for diagnosing the following disease states:

- Cerebrovascular disease or **stroke**: SPECT is useful to detect ischemia (reduced blood flow), determination of stroke causes, evaluate transient ischemia, determine prognosis, and monitor treatment.
- Dementia such as in Alzheimer's disease: SPECT studies can be used effectively to rule out other medical causes of dementia.
- Head trauma: Evidence suggests that SPECT is useful to detect greater number of lesions following the period after head trauma. It seems that the high resolution and accurate brain images of SPECT can detect lesions in the brain that are not possible to visualize using other techniques such as **positron emission tomography**

(PET) scanning. SPECT images can give clinicians important information concerning prognosis (also sometimes called outcome) and treatment of persons affected with head injury.

- Epilepsy: The radioactive material injected before SPECT imaging concentrates at the seizure locus (the region that contains nerve cells that generate an abnormal impulse). This can help identify the location of **seizures** and assist clinicians concerning management and outcomes.
- SPECT allows clinicians to visualize a specific area of the brain called the striatum, which contains a neurotransmitter (a chemical that communicates nerve impulse from one nerve cell to another) called dopamine. Circuitry in the striatum and interaction with dopamine can help provide valuable information concerning **movement disorders**, **schizophrenia**, mood disorders, and hormone diseases (since hormones require control and regulation from the brain in structures called the pituitary gland and hypothalamus).

As a research tool, SPECT imaging seems to be sensitive tool to measure blood flow through the brain (cerebral blood flow), in persons who have psychological disorder such as **obsessive-compulsive disorder** (higher blood flow) and alcoholism (lower blood flow).

Other dianostic indications and procedures are similar to other **imaging studies** such as computed tomography, **magnetic resonance imaging**, and PET.

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Skills training *see* **Social skills training**

Sleep disorders

Definition

Sleep disorders are chronic disturbances in the quantity or quality of sleep that interfere with a person's ability to function normally.

Description

An estimated 15% of Americans have chronic sleep problems, while about 10% have occasional trouble sleeping. Sleep disorders are listed among the clinical syndromes in Axis I of the American Psychiatric Association's ***Diagnostic and Statistical Manual of Mental Disorders***, also known as the *DSM*. They may be either primary (unrelated to any other disorder—medical or psychological) or secondary (the result of physical illness, psychological disorders, or drug or alcohol use).

In the revised fourth edition of the *DSM* (*DSM-IV-TR*), the primary sleep disorders are categorized as either dyssomnias or parasomnias. Dyssomnias pertain to the amount, quality, or timing of sleep, whereas parasomnias pertain to abnormal behavioral or physiological events that occur while sleeping. Dyssomnias include:

- Primary insomnia—difficulty getting to sleep or staying asleep. Sleep loss is so severe that it interferes with daytime functioning and well-being. Three types of **insomnia** have been identified (although a single person can have more than one): sleep-onset insomnia (difficulty falling asleep); sleep-maintenance insomnia (difficulty staying asleep); and terminal insomnia (waking early and not being able to go back to sleep). While insomnia can occur at any stage of life, it becomes increasingly common as people get older.
- Primary **hypersomnia**—excessive sleepiness either at night or during the day.
- **Narcolepsy**—sudden attacks of REM sleep during waking hours. Many narcoleptics experience additional symptoms including cataplexy (a sudden loss of muscle tone while in a conscious state), **hallucinations** and other unusual perceptual phenomena, and sleep paralysis, an inability to move for several minutes upon awakening. The disorder is caused by a physiological **brain** dysfunction that can be inherited or develop after trauma to the brain from disease or injury.
- **Breathing-related sleep disorder**—abnormalities in breathing cause sleep disruptions. Sleep apnea consists of disrupted breathing which wakens a person repeatedly during the night. Though unaware of the problem while it is occurring, people with sleep apnea are unable to get a good night's sleep and feel tired and

sleepy during the day. The condition is generally caused either by a physical obstruction of the upper airway or an impairment of the brain's respiration control centers.

- **Circadian rhythm sleep disorder**—environmental disruptions to an individual's internal 24-hour-clock affect his or her sleep patterns. This disorder has four subtypes: delayed sleep phase type, jet lag type, shift work type, and unspecified type.

Parasomnias include:

- **Nightmare disorder**—nightmares repeatedly awaken the affected individual.
- **Sleep terror disorder**—affected individual is repeatedly awakened from sleep and remains awake and frightened for a short period of time (about 10 minutes or so, usually less), and during that time, the individual is difficult to awaken or comfort. No dream is recalled, and the person often does not remember the event the following day.
- **Sleepwalking disorder**—repeated episodes of motor activity during sleep, including getting out of bed and walking around.

Other features of parasomnias not listed in the *DSM-IV-TR* include bruxism (teeth grinding) and **enuresis** (bed-wetting). Both are often stress-related, although enuresis may also be caused by genitourinary disorders, neurological disturbances, or toilet training problems. A parasomnia only identified in the late twenieth century is REM sleep behavior disorder. Those affected by this condition—usually middle-aged or older men—engage in vigorous and bizarre physical activities during REM sleep in response to dreams, which are generally of a violent, intense nature. As their actions may injure themselves or their sleeping partners, this disorder, thought to be neurological in nature, has been treated with hypnosis and medications, including **clonazepam** and **carbamazepine**.

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ORGANIZATIONS

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Sleep terror disorder

Definition

Sleep terror disorder is defined as repeated temporary arousal from sleep, during which the affected person appears and acts extremely frightened.

Description

Sleep terror disorder is sometimes referred to as *pavor nocturnus* when it occurs in children, and *incubus* when it occurs in adults. Sleep terrors are also sometimes called night terrors, though sleep terror is the preferred term, as episodes can occur during daytime naps as well as at night. Sleep terror is a disorder that primarily affects children, although a small number of adults are affected as well.

Causes and symptoms

Causes

The causes of sleep terror are for the most part unknown. Some researchers suggest that sleep terrors are caused by a delay in the maturation of the child's central nervous system. Such factors as sleep deprivation, psychological **stress**, and fever may also trigger episodes of sleep terror.

Symptoms

The symptoms of sleep terror are very similar to the physical symptoms of extreme fear. These include rapid heartbeat, sweating, and rapid breathing (hyperventilation). The heart rate can increase up to two to four times the person's regular rate. Sleep terrors cause people to be jolted into motion, often sitting up suddenly in bed. People sometimes scream or cry. The person's facial expression may be fearful.

People experiencing sleep terror disorder sometimes get out of bed and act as if they are fighting or fleeing something. During this time injuries can occur. Cases have been reported of people falling out of windows or falling down stairs during episodes of sleep terror.

People experiencing sleep terror are not fully awake. They are nearly impossible to bring to consciousness or comfort, and sometimes respond violently to attempts to console or restrain them. In many cases, once the episode is over the person returns to sleep without ever waking fully. People often do not have any recollection of the episode after later awaking normally, although they may recall a sense of fear.

Episodes of sleep terror usually occur during the first third of a person's night sleep, although they can occur even during naps taken in the daytime. The average sleep terror episode lasts less than 15 minutes. Usually only one episode occurs per night, but in some cases terror episodes occur in clusters. It is unusual for a person to have many episodes in a single night, although upwards of 40 have been reported. Most persons with the disorder have only one occurrence per week, or just a few per month.

Demographics

Sleep terror disorder is much more common in children than it is in adults. It is estimated that approximately 1%–6% of children in the United States experience sleep terror at some point in their childhood. For most children, sleep terrors begin between the ages of four and 12. The problem usually disappears during adolescence. Sleep terror disorder appears to be more common in boys than in girls; some studies have reported that preadolescent boys are the group most commonly affected. No figures are available for the rates of the disorder in different racial or ethnic groups. Sleep terrors in children are not associated with any psychological disorders.

Fewer than 1% of adults have sleep terror disorder. For most adults, sleep terrors begin in their 20s or 30s, although it is possible for someone to suffer from episodes of sleep terror from childhood onward. In the adult population, sleep terrors affect both sexes equally. They are, however, often associated with psychological disorders, most commonly anxiety, personality, or posttraumatic disorders. People who have a family history of sleep terrors or **sleepwalking disorder** are about 10 times more likely to develop sleep terror disorder than those who do not.

Diagnosis

Sleep terror is diagnosed most often in children when parents express concern to the child's pediatrician. A fact sheet from the American Academy of Child and Adolescent Psychiatry suggests that parents consult a child **psychiatrist** if the child has several episodes of sleep terror each night, if the episodes occur every night for weeks at a time, or if they interfere with the child's daytime activities. The **diagnosis** is usually made on the basis of the child's and parents' description of the symptoms. There are no laboratory tests for sleep terror disorder. In adults, the disorder is usually self-reported to the patient's family doctor. Again, the diagnosis is usually based on the patient's description of the symptoms.

KEY TERMS

Hypnotic—A type of medication that induces sleep.

Pavor nocturnus—The Latin medical term for sleep terror disorder.

Sleep terror is characterized by an abrupt arousal from sleep followed by symptoms of extreme fear. The symptoms often include screams, rapid heartbeat, heavy breathing, and sweating, as well as a subjective feeling of terror. According to the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (*DSM-IV-TR*), which is the standard reference work used by mental health professionals to diagnose mental disorders, people with sleep terror disorder do not respond to attempts to comfort or awaken them. In order to meet criteria for the diagnosis, the patients must not be able to recall their dreams, and they must not remember the episode itself. In addition, the episodes may not be attributed to a medical condition or drug use.

Sleep terror disorder is frequently confused with **nightmare disorder**. The two are similar in the sense that both are related to bad dreams. Nightmare disorder, however, involves a significantly smaller amount of physical movement than does sleep terror. Normally, people experiencing nightmare disorder do not get out of bed.

Moreover, people experiencing nightmare disorder often have problems going back to sleep because of the nature of their dream. Most people experiencing sleep terrors, however, go back to deep sleep without ever having fully awakened. People experiencing nightmares can generally remember their dreams and some of the events in the dream leading up to their awakening. People often awake from nightmares just as they are about to experience the most frightening part of a disturbing dream. People experiencing sleep terrors, however, can sometimes recall a sense of profound fear, but often do not remember the episode at all.

Treatments

If sleep terror episodes are infrequent, then treatment may not be necessary as long as the episodes are not

interfering significantly with the person's life. Some people may want to rearrange their bedroom furniture to minimize the possibility of hurting themselves or others if they get out of bed during a sleep terror episode. To keep children from becoming overly worried about their sleep terrors, experts suggest that parents avoid placing unnecessary emphasis on the episodes. **Psychotherapy** is often helpful for adults concerned about the specific triggers of sleep terror episodes.

Several different medications have been used to treat sleep terror disorder, with varying degrees of success. One of the most common is **diazepam** (Valium). Diazepam is a hypnotic (sleep-inducing medication), and is thought to be useful in the prevention of sleep terror episodes because it acts as a nervous system depressant. There are many different types of hypnotics, and choosing one for a patient depends on other drugs that the patient may be taking, any medical or psychological conditions, and other health factors. Most studies of medications as treatments for sleep terror disorder have been done on adult patients; there is little information available on the use of medications to treat the disorder in children.

Prognosis

In most children, sleep terror disorder resolves before or during adolescence without any treatment. Adults often respond well to diazepam or another hypnotic. Psychotherapy and avoidance of stressors that may precipitate terror episodes may be helpful as well. Episodes of sleep terrors often decrease with age. This decrease is due to the fact that the amount of slow-wave sleep, which is the sleep phase during which terror episodes usually occur, declines with age.

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Tish Davidson, A.M.

Sleepwalking disorder

Definition

Sleepwalking disorder, also called somnambulism, is characterized by repeating episodes of motor activity during sleep such as sitting up in bed, rising, and walking around, among others. The person appears to be awake because their eyes are usually open and they can maneuver around objects, but is considered asleep.

Sleepwalking disorder is one of several **sleep disorders** listed in the ***Diagnostic and Statistical Manual of Mental Disorders***, often called *DSM-IV-TR*, produced by the American Psychiatric Association and used by most mental health professionals in North America and Europe to diagnose mental disorders.

Description

Sleepwalking episodes usually occur during the first third of the night during the deepest phase of sleep. The episodes can last anywhere from a few minutes up to one hour, with five to 15 minutes being average. Sleepwalkers appear to be awake but are typically unresponsive to individuals who attempt to communicate with them. Persons who sleepwalk typically have no memory or awareness of their actions or movement upon waking.

Causes and symptoms

Causes

There appears to be a genetic component for individuals who sleepwalk. The condition is 10 times more likely to occur in close relatives of known sleepwalkers than in the general public. These families also tend to be deep sleepers.

Sleepwalking may also be triggered by fever, which directly affects the nervous system, general illness, alcohol use, sleep deprivation, and emotional **stress**. Hormonal changes that occur during adolescence, menstruation, and pregnancy can be also be triggers for sleepwalking. Sleepwalking episodes are more likely during times of physiological or psychological stress.

Certain classes of medication have also been shown to precipitate sleepwalking episodes in some individuals. These include: Anti-anxiety or sleep-inducing drugs, antiseizure medications, stimulants, antihistamines, and anti-arrhythmic heart drugs.

Symptoms

The *DSM-IV-TR* specifies six diagnostic criteria for sleepwalking disorder:

- Repeated episodes of rising from bed during sleep: These episodes may include sitting up in bed, looking around, and walking, and usually occur during the first third of the night.
- Is unresponsive to attempts at communication: During sleepwalking, the person typically has eyes open, dilated pupils, a blank stare, and does not respond to another's attempts at communication. Affected persons typically are only awakened with great difficulty.
- No recollection of the sleepwalking incident: Upon waking, the person typically has no memory of the sleepwalking events. If the individual does awaken from the sleepwalking episode, they may have a vague memory of the incident. Often, sleepwalkers will return to bed, or fall asleep in another place with no recall as to how they got there.
- No impairment of mental activity upon waking: If an individual awakens during a sleepwalking episode, there may be a short period of confusion or disorientation, but there is no impairment of mental activity or behavior.
- Causes significant distress to life situations: Sleepwalking causes significant disruption of social and occupational situations, or affects other abilities to function.
- Not due to substance use or abuse: Sleepwalking disorder is not diagnosed if the cause is related to drug abuse, medication, or a general medical condition.

Demographics

Sleepwalking can occur at any age but is most common in children, with the first episodes usually between the ages of four and eight years. The peak of sleepwalking behavior occurs at about 12 years of age. Between 10 and 30% of children have had at least one episode of sleepwalking. Sleepwalking disorder is seen in only 1–5% of children and occurs more frequently in boys. Adults who sleepwalk typically have a history of sleepwalking that stems back to childhood. Sleepwalking events occur in approximately 1–7% of adults while sleepwalking disorder occurs in about 0.5%.

Diagnosis

The line that separates periodic sleepwalking from sleepwalking disorder is not clearly defined. Individuals or families most often seek professional help when the episodes of sleepwalking are violent, pose a risk for injury, or impair the person's ability to function. For a **diagnosis** of sleepwalking disorder to be made, the person must experience a significant amount of social, occupational, or other impairment related to the sleepwalking problem. Episodes that have a long history extending from childhood through adolescence and especially into adulthood are more likely to be diagnosed with sleepwalking disorder.

Since the individual cannot recall the sleepwalking activity, diagnosis by means of interview is of little benefit, unless it involves someone who has witnessed the sleepwalking behavior. The preferred method for accurate diagnosis is through **polysomnography**. This technique involves hooking electrodes to different locations on the affected person's body to monitor **brain** wave activity, heart rate, breathing, and other vital signs while the individual sleeps. Monitoring brain-wave patterns and physiologic responses during sleep can usually give sleep specialists an accurate diagnosis of the condition and determine the effective means of treatment, if any.

Sleepwalking disorder can be difficult to distinguish from **sleep terror disorder**. In both cases, the individual has motor movement, is difficult to awaken, and does not remember the incident. The primary difference is that sleep terror disorder typically has an initial scream and signs of intense fear and panic associated with the other behaviors.

Treatments

Treatment for sleepwalking is often unnecessary, especially if episodes are infrequent and pose no hazard to the sleepwalker or others. If sleepwalking is recurrent, or daytime **fatigue** is suspected to result from disturbed sleep patterns, polysomnography may be recommended to determine whether some form of treatment may be helpful. If stress appears to trigger sleepwalking events in adults, stress management, **biofeedback** training, or relaxation techniques can be beneficial. Hypnosis has been used help sleepwalkers awaken once their feet touch the floor. **Psychotherapy** may help individuals who have underlying psychological issues that could be contributing to sleep problems.

Medications are sometimes used in the more severe cases with adults. Benzodiazepines—anti-anxiety drugs—such as **diazepam** (Valium) or **alprazolam** (Xanax) can be used to help relax muscles, although these may not result in fewer episodes of sleepwalking. When medications are used, they are typically prescribed in the lowest dose necessary and only for a limited period.

Prognosis

Most cases of sleepwalking subside over time. Sleepwalking in childhood usually disappears without treatment by age 15. If sleepwalking episodes persist into early adulthood, treatment is recommended. With an accurate diagnosis and appropriate treatment, episodes of sleepwalking can be greatly reduced and, in some cases, eliminated.

Prevention

In children, sleepwalking is relatively common and is not cause for concern. The major risk associated with sleepwalking is accidental injury. Parents should take precautions to block stairways, lock windows, keep floors cleared of harmful objects, etc.

If taking certain medications, a medical condition, or exposure to significant stressors are suspected triggers of sleepwalking episodes, a doctor should be consulted for a complete assessment.

Resources

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Gary Gilles, M.A.

Smoking *see* **Nicotine and related disorders**

Social phobia

Definition

Social phobia is defined by *DSM-IV-TR* as an anxiety disorder characterized by a strong and persistent fear of social or performance situations in which the patient might feel embarrassment or humiliation. Generalized social phobia refers to a fear of most social interactions combined with fear of most performance situations, such as speaking in public or eating in a restaurant. Persons who are afraid of only one type of performance situation or afraid of only a few rather than most social situations may be described as having nongeneralized, circumscribed, or specific social phobia.

Social phobia, which is also known as social anxiety disorder, is a serious mental health problem in the United States. In any given year, social phobia affects 3.7% of the American population between the ages of 18 and 54, or about 5.3 million people. It is the third most common psychiatric condition after depression and alcoholism. Patients diagnosed with social phobia have the highest risk of alcohol abuse of all patients with anxiety disorders; in addition, they suffer from worse impairment than patients with major medical illnesses, including congestive heart failure and diabetes.

KEY TERMS

Amygdala—An almond-shaped brain structure in the limbic system that is activated in acute stress situations to trigger the emotion of fear. Some studies suggest that social phobia may be related to changes in or overfunctioning of the amygdala.

Behavioral inhibition—A set of behaviors that appear in early infancy that are displayed when the child is confronted with a new situation or unfamiliar people. These behaviors include moving around, crying, and general irritability, followed by withdrawing, and seeking comfort from a familiar person. These behaviors are associated with an increased risk of social phobia and panic disorder in later life. Behavioral inhibition in children appears to be linked to anxiety and mood disorders in their parents.

Cognitive restructuring—An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

Exposure therapy—A form of cognitive-behavioral therapy in which patients suffering from phobias are exposed to their feared objects or situations while accompanied by the therapist. The length of exposure is gradually increased until the association between the feared situation and the patient's experienced panic symptoms is no longer present.

Limbic system—A group of structures in the brain that includes the amygdala, hippocampus, olfactory bulbs, and hypothalamus. The limbic system is associated with homeostasis and the regulation and arousal of emotions.

Mutism—Inability to speak due to conscious refusal or psychogenic inhibition. Mutism is a common symptom of social phobia in children.

Performance anxiety—A subcategory of circumscribed social phobia in which the patient's fear is limited to performing certain activities or tasks in public. Common areas of performance anxiety include public speaking, acting on stage, solo singing, and playing instrumental solos.

Phobia—Irrational fear of places, things, or situations that lead to avoidance.

Social modeling—A process of learning behavioral and emotional response patterns from observing one's parents or other adults. Some researchers think that social modeling plays a part in the development of social phobia.

Temperament—A person's natural disposition or inborn combination of mental and emotional traits. Children with a shy or withdrawn temperament are at increased risk of developing social phobia in adolescence.

Description

Social phobia varies in its development and initial presentation. In some young people, the disorder grows out of a long-term history of shyness or social inhibition. In others, social phobia becomes apparent following a move to a new school or similar developmental challenge. In adults, circumscribed social phobia may be associated with a change of occupation or job promotion, the most common example being the emergence of the disorder with regard to public speaking in a person whose previous jobs did not require them to make presentations or speeches in front of others. The onset of social phobia may be insidious, which means that it gets worse by slow degrees. About half of all patients, however, experience a sudden onset of social phobia following a particularly humiliating or frightening experience. For example, in one British case study the patient's social phobia developed abruptly after her father's sudden death. The patient had had an argument with him one morning and he was killed in an accident later in the day. The onset of social phobia almost always occurs in childhood or the mid-teens; onset after age 25 is unusual. The disorder is often a lifelong problem, although its severity may diminish in adult life.

Adults and adolescents with social phobia, as well as many children with the disorder, have sufficient insight to recognize that their fears are excessive or unwarranted. This factor often adds to their distress and feelings of inferiority.

Social phobia is of major concern to society as a whole for two reasons. One reason is the disorder's very high rate of comorbidity with such other mental health problems as major depression and substance abuse. In comparison with patients diagnosed with other anxiety disorders, patients with social phobia have higher averages of concurrent anxiety disorders (1.21 versus 0.45); comorbid depression or other disorders (2.05 versus 1.19); and lifetime disorders (3.11 versus 2.05). The most

common comorbid disorders diagnosed in patients with social phobia are major depression (43%); **panic disorder** (33%); **generalized anxiety disorder** (19%); PTSD (36%); alcohol or substance abuse disorder (18%); and attempted **suicide** (23%).

The second reason is the loss to the larger society of the gifts and talents that these patients possess. Social phobia can have a devastating effect on young people's intellectual life and choice of career, causing them to abandon their educations, stay stuck in dead-end jobs, refuse promotions involving travel or relocation, and make similar self-defeating choices because of their fear of classroom participation, job interviews, and other social interactions in educational and workplace settings. One sample of patients diagnosed with social phobia found that almost half had failed to finish high school; 70% were in the bottom two quartiles of socioeconomic status (SES); and 22% were on welfare. In addition to their academic and employment-related difficulties, people with social phobia have limited or nonexistent social support networks. They are less likely to marry and start families of their own because of their fear of interpersonal relationships. Many continue to live at home with their parents even as adults, or remain in unfulfilling relationships.

Causes and symptoms

Causes

The causes of social phobia appear to be a combination of physical and environmental factors.

NEUROBIOLOGICAL FACTORS. There is some evidence as of 2002 that social phobia can be inherited. A group of researchers at Yale has identified a genetic locus on human chromosome 3 that is linked to **agoraphobia** and two genetic loci on chromosomes 1 and 11q linked to panic disorder. Because social phobia shares some traits with panic disorder, it is likely that there are also genes that govern a person's susceptibility to social phobia. In addition, researchers at the National Institute of Mental Health (NIMH) have identified a gene in mice that appears to govern fearfulness.

Positron emission tomography (PET) scans of patients diagnosed with social phobia indicate that blood flow is increased in a region of the **brain** (the amygdala) associated with fear responses when the patients are asked to speak in public. In contrast, PET scans of control subjects without social phobia show that blood flow during the public speaking exercise is increased in the cerebral cortex, an area of the brain associated with thinking and evaluation rather than emotional arousal. The researchers have concluded that patients with social phobia have a different neurochemical response to certain social situations or challenges that activates the limbic system rather than the cerebral cortex.

TEMPERAMENT. A number of researchers have pointed to inborn temperament (natural predisposition) as a broad vulnerability factor in the development of anxiety and mood disorders, including social phobia. More specifically, children who manifest what is known as behavioral inhibition in early infancy are at increased risk for developing more than one anxiety disorder in adult life, particularly if the inhibition remains over time. Behavioral inhibition refers to a group of behaviors that are displayed when the child is confronted with a new situation or unfamiliar people. These behaviors include moving around, crying, and general irritability, followed by withdrawing, seeking comfort from a familiar person, and stopping what one is doing when one notices the new person or situation. Children of depressed or anxious parents are more likely to develop behavioral inhibition. One study of preadolescent children diagnosed with social phobia reported that many of these children had been identified as behaviorally inhibited in early childhood.

PSYCHOSOCIAL FACTORS. The development of social phobia is also influenced by parent-child interactions in a patient's family of origin. Several studies have found that the children of parents with major depression, whether or not it is comorbid with panic disorder, are at increased risk of developing social phobia. Children of parents with major depression and comorbid panic disorder are at increased risk of developing more than one anxiety disorder. A family pattern of social phobia, however, is stronger for the generalized than for the specific or circumscribed subtype.

It is highly likely that the children of depressed parents may acquire certain attitudes and behaviors from their parents that make them more susceptible to developing social phobia. One study of children with social phobia found that their cognitive assessment of ambiguous situations was strongly negative, not only with regard to the dangerousness of the situation but also in terms of their ability to cope with it. In other words, these children tend to overestimate the threats and dangers in life and to underestimate their strength, intelligence, and other resources for coping. This process of learning from observing the behavior of one's parents or other adults is called social **modeling**.

Still another psychosocial factor related to the development of social phobia in children and adolescents is the general disintegration in the social fabric of the developed countries since World War II. A number of social theorists as well as physicians and therapists have noted

that children are exposed more frequently to both real-life and media depictions of aggressive behavior and abrasive language than earlier generations. Children also learn about frightening or unpleasant social realities at earlier and earlier ages. The increased rate of social phobia and school refusal among adolescent girls has been linked to the greater crudity of teasing from boys in junior high and high school. The American Association of University Women released a study in 1998 that reported that 70% of girls experience verbal sexual harassment in high school and 50%, unwanted sexual touching. In addition, the fortress mentality reflected in the architecture of high-rise apartment buildings and gated communities for those who can afford them also sends children the message that other people are to be feared. While trends in the larger society may not directly cause social phobia (or other mental disorders), they are nonetheless important indirect influences.

Symptoms

The symptoms of social phobia are somewhat different in children and adults, in that the early onset of social phobia typically means that children with the disorder fail to achieve at their predicted level, whereas adults and adolescents show declines from previously achieved levels of functioning.

SYMPTOMS IN CHILDREN. Symptoms of social phobia in children frequently include tantrums, crying, "freezing," clinging to parents or other familiar people, and inhibiting interactions to the point of refusing to talk to others (mutism).

SYMPTOMS IN ADULTS. The symptoms of social phobia in adults include a range of physical signs of anxiety as well as attitudes and behaviors.

- blushing, sweating, nausea, diarrhea, dry mouth, tremors, and other physical indications of anxiety
- difficulties with self-assertion
- extreme sensitivity to criticism, rejection, or negative evaluations
- intense preoccupation with the reactions and responses of others
- heightened fears of being embarrassed or humiliated
- avoidance of the feared situation(s) and anticipatory anxiety

In adults, there is often a "vicious circle" quality to the symptoms, in that the anxiety and symptoms lead to actual or perceived poor performances, which in turn increase the anxiety and avoidance. A common example is performance anxiety related to musical instruments; the person who is afraid of having to play the piano in a recital, for example, may become so anxious that the muscles in the hands become tense, thus producing frequent mistakes in fingering and sound production during the recital performance.

Social phobia is an anxiety disorder characterized by a strong and persistent fear of social or performance situations in which the patient might feel embarrassment or humiliation. Performance situations are those such as speaking in front of an audience. *(Bill Varie/CORBIS. Photo reproduced by permission.)*

Not all adults with social phobia appear shy or outwardly nervous to other people. Some adults are able to force themselves to attend social events, give public presentations, or interact with others by self-medicating with alcohol or limiting the time period of their interactions. These strategies, however, prevent the underlying fears and disabilities from being addressed.

Demographics

The prevalence of social phobia in the general United States population is difficult to evaluate because researchers differ in their estimation of the threshold of "significant interference" with the person's occupational or educational functioning. In addition, different studies have focused on different subtypes of social phobia. One study found that about 20% of the adults surveyed reported high levels of anxiety related to public speaking or other types of public performance, but only 2% indicated sufficient distress to meet the diagnostic criteria of social phobia. Because of these differences in measurement, epidemiological and community-based studies give figures for a lifetime prevalence of social phobia that fall between 3% and 13%.

The types of situations associated with social phobia are different in the general population as contrasted with clinical populations. Surveys of adults in the general pop-

ulation indicate that most people diagnosed with social phobia are afraid of public speaking; only 45% report being afraid of meeting new people or having to talk to strangers. Fears related to eating, drinking, or writing in public, or using a public restroom, are much less common in this group of patients. By contrast, people being treated for social phobia in outpatient clinics are more likely to be afraid of a range of social situations rather than just one. Social phobia accounts for 10%–20% of the anxiety disorders diagnosed in patients in outpatient clinics, but it is rarely the reason for hospitalizing a patient.

The same difference between general and clinical populations affects the sex ratios given for social phobia. Community-based studies suggest that social phobia is more common in women, but in most samples of clinical patients, the sex ratio is either 1:1 or males are in the majority. A study of social phobia in prepubertal children found that girls were more likely to verbalize anxiety than boys, but the researchers who observed the children interact with adults and with one another did not observe any behavioral differences between boys and girls. The researchers concluded that the apparently higher rates of social phobia in women may simply reflect women's greater openness about their feelings.

With regard to race, the same study found no statistically significant difference in the incidence of social phobia between Caucasian and African American children. This finding was consistent with a 1995 study that failed to find differences based on race in lists of children's top 10 fears. Further research, however, is necessary in order to determine whether social phobia has different symptom patterns or rates of development in different racial or ethnic groups.

The demographics of social phobia in young children are particularly difficult to determine because of changes in diagnostic categories and criteria in successive editions of *DSM*. Social phobia was introduced as a diagnostic category in *DSM-III*, which was published in 1980. Neither *DSM-III* nor its 1987 revision restricted social phobia to adults, but the disorder was rarely diagnosed in children—most likely because *DSM-III* and *DSM-III-R* listed two diagnoses for children, overanxious disorder and avoidant disorder of childhood, whose symptoms overlapped with those of social phobia. Statistics based on *DSM-III-R*'s criteria for social phobia placed the prevalence of the disorder in children in the general population at about 1%. The revisions of the diagnostic criteria in *DSM-IV*, however, have led to an apparent dramatic increase in the prevalence of social phobia in children. One study done in 1997 reported that 18% of the children in a clinical sample met *DSM-III-R* criteria for social phobia, but that 40% of the children in the same sample had social phobia according to *DSM-IV* criteria.

Diagnosis

The **diagnosis** of social phobia is usually made on the basis of the patient's history and reported symptoms. The doctor may also decide to administer diagnostic questionnaires intended to rule out other phobias, other anxiety disorders, and major depression. In diagnosing a child, the doctor will usually ask the child's parents, teachers, or others who know the child well for their observations.

Children and adolescents

A doctor who is evaluating a child for social phobia must take into account that children do not have the freedom that adults usually have to avoid the situations that frighten them. As a result, they may not be able to explain why they are anxious. It is important to evaluate the child's capacity for social relationships with people that he or she knows; and to assess his or her interactions with peers for indications of social phobia, not only his or her behavior around adults.

A semi-structured interview that a doctor can use to assess social phobia in children is the Anxiety Disorders Interview Schedule for Children, or ADIS-C. A newer clinician-administered test is the Liebowitz Social Anxiety Scale for Children and Adolescents, or LSAS-CA. Self-report inventories for children include the **Child Depression Inventory**, or CDI, and the Social Phobia and Anxiety Inventory for Children, or SPAI-C. Parents can be asked to complete the Child Behavior Checklist (CBL), and teachers may be given the Teacher's Report Form (TRF).

Adults

Diagnostic instruments for assessing social phobia in adults are more problematic. Some general screeners that are used in primary care settings, such as the Structured Clinical Interview for DSM-IV-Screen (SCID-Screen), do include questions related to social phobia but can take as long as 25 minutes to administer. Others, such as the Primary Care Evaluation of Mental Disorders, or Prime-MD, are not specific for social phobia. Instruments designed to measure social phobia by itself, such as the Fear of Negative Evaluation Scale and the Social Avoidance and Distress Scale, are lengthy and generally more useful for monitoring the progress of therapy. Another clinician-administered interview for social phobia in adults, the Liebowitz Social Anxiety Scale (LSAS), is not yet in widespread use.

Many physicians, however, have found that the addition of a few selected questions to a general screener for mental disorders is helpful in detecting social phobia. One study found that giving patients three specific state-

ments with yes/no answers detected 89% of cases of social phobia:

- Being embarrassed or looking stupid are among my worst fears.
- Fear of embarrassment causes me to avoid doing things or speaking to people.
- I avoid activities in which I am the center of attention.

As of 2002 there are no laboratory tests or brain imaging techniques that can help to diagnose social phobia in adults.

Treatments

Social phobia responds well to proper treatment; however, patients with social phobia have a distinctive set of barriers to treatment. Unlike persons with some other types of mental disorders, they are unlikely to deny that they have a problem. What researchers have found is that in comparison to persons suffering from other disorders, persons with social phobia are significantly more likely to say that financial problems, uncertainty over where to go for help, and fear of what others might think prevent them from seeking treatment. The researchers concluded that providing better information about community services as well as easing the psychological and financial burdens of patients with social phobia would significantly improve their chances of recovery. Left untreated, social phobia can become a chronic, disabling disorder that increases the patient's risk of suicide.

Medications

About 53% of patients diagnosed with social phobia are treated with medications. Drug treatment has proven beneficial to patients with this disorder; however, no one type of medication appears to be clearly superior to others. Selection of a medication depends on the subtype of the patient's social phobia; the presence of other mental disorders; and the patient's occupation and personal preferences.

Specific medications that are used to treat social phobia include:

- Benzodiazepine tranquilizers. These are often prescribed for patients who need immediate relief from anxiety. They have two major drawbacks, however; they are habit-forming, and they are unsuitable for patients with comorbid alcohol or substance abuse disorders. Benzodiazepines are, however, sometimes prescribed for patients who have a low risk for substance abuse and have not responded to other medications.
- Monoamine oxidase inhibitors (MAOIs). About two-thirds of patients with social phobia show significant improvement when treated with these drugs. MAOIs, however, have the disadvantage of requiring patients to stick to a low-tyramine diet that excludes many popular foods, and requiring them to avoid many over-the-counter cold and cough preparations.
- Selective serotonin reuptake inhibitors (SSRIs). About 50%–75% of patients with social phobia benefit from treatment with SSRIs. The SSRIs appear to work best in patients with comorbid major depression or panic disorder. **Sertraline** (Zoloft) has been recommended for patients with generalized social phobia.
- Newer drugs. A recent placebo-controlled study indicates that **gabapentin** (Neurontin) shows promise as a treatment for social phobia.
- **Beta blockers**. These medications, which include **propranolol** (Inderal), are given to patients with mild to moderate circumscribed performance anxiety. The patient takes the medication on an as-needed basis rather than a standing dosage. Beta-blockers do not appear to be helpful for patients with generalized social phobia.

Psychotherapy

The type of **psychotherapy** most commonly recommended for treatment of social phobia is **cognitive-behavioral therapy** (CBT). Mild to moderate cases of social phobia often show considerable improvement with CBT alone; patients with more severe social phobia benefit from a combination of CBT and an appropriate medication. Cognitive-behavioral treatment of adults diagnosed with social phobia usually combines exposure therapy with cognitive restructuring techniques. In exposure therapy, the patient is exposed to small "doses" of the feared situation that are gradually lengthened in time. The chief drawback to exposure therapy for social phobia is that some feared situations are easier to replicate for purposes of treatment than others. Patients who are afraid of public speaking or musical performance can practice performing in front of any group of people that can be collected to help; but it is not so easy to arrange exposure sessions for a patient who is afraid of interactions with a specific teacher, employer, or supervisor. The other aspect of CBT that is used in treating social phobia in adults is cognitive restructuring. This approach challenges the patient to reconsider and then replace the biased cognitions that have led him or her to overestimate the dangers in social situations and to underestimate his or her own resources for coping with them.

Several trial programs of CBT **group therapy** have been used with adolescents with social phobia. One pilot program situated the group meetings in the school rather than in a clinic, on the grounds that most of the fears of adolescents with social phobia revolve around school

activities. Another CBT group for adolescents was conducted in a clinical setting. Both programs included **social skills training** alongside exposure therapy and cognitive restructuring, and both were reported to be moderately successful at one-year follow-up.

Other

Other approaches that have been used to treat social phobia include **family therapy** and relaxation techniques.

Prognosis

The prognosis for recovery from social phobia is good, given early diagnosis and appropriate treatment. The prognosis for persons with untreated social phobia, however, is poor. In most cases, these individuals become long-term underachievers, at high risk for alcoholism, major depression, and suicide.

Prevention

Given that some of the factors implicated in social phobia are neurobiological or genetic, the best preventive strategy as of 2002 is early identification of children with behavioral inhibition and developing techniques for assisting their social development.

See also Child Depression Inventory; Exposure Treatment

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Social skills training

Definition

Social skills training (SST) is a form of behavior therapy used by teachers, therapists, and trainers to help persons who have difficulties relating to other people.

Purpose

Goals

A major goal of social skills training is teaching persons who may or may not have emotional problems about the verbal as well as nonverbal behaviors involved in social interactions. There are many people who have never been taught such interpersonal skills as making "small talk" in social settings, or the importance of good eye contact during a conversation. In addition, many people have not learned to "read" the many subtle cues contained in social interactions, such as how to tell when someone wants to change the topic of conversation or shift to another activity. Social skills training helps patients to learn to interpret these and other social signals, so that they can determine how to act appropriately in the company of other people in a variety of different situations. SST proceeds on the assumption that when people improve their social skills or change selected behaviors, they will raise their self-esteem and increase the likelihood that others will respond favorably to them. Trainees learn to change their social behavior patterns by practicing selected behaviors in individual or **group therapy** sessions. Another goal of social skills training is improving a patient's ability to function in everyday social situations. Social skills training can help patients to work on specific issues—for example, improving one's telephone manners—that interfere with their jobs or daily lives.

Treatment of specific disorders

A person who lacks certain social skills may have great difficulty building a network of supportive friends and acquaintances as he or she grows older, and may become socially isolated. Moreover, one of the consequences of loneliness is an increased risk of developing emotional problems or mental disorders. Social skills training has been shown to be effective in treating patients with a broad range of emotional problems and diagnoses. Some of the disorders treated by social skills trainers include shyness; adjustment disorders; marital and family conflicts, anxiety disorders, **attention-deficit/hyperactivity disorder**, **social phobia**, alcohol dependence; depression; **bipolar disorder**; **schizophrenia**; developmental disabilities; **avoidant personality disorder**; **paranoid personality disorder**; **obsessive-compulsive disorder**; and **schizotypal personality disorder**.

A specific example of the ways in which social skills training can be helpful includes its application to alcohol dependence. In treating patients with alcohol dependence, a therapist who is using social skills training focuses on teaching the patients ways to avoid drinking when they go to parties where alcohol is served, or when they find themselves in other situations in which others may pressure them to drink.

KEY TERMS

Cue—Any behavior or event in a person's environment that serves to stimulate a particular response. For example, the smell of liquor may be a cue for some people to pour themselves a drink. In social skills training, the term is usually used to refer to social signals, whether spoken or unspoken.

Feedback—A reaction or response from others to a particular behavior or activity.

Generalization—A person's ongoing use of new behaviors that were previously modeled for him or her. Generalization is also called transfer of training or maintenance.

Modeling—A type of teaching method used in social skills training. Therapists who use this method may offer positive and negative examples of the behaviors that make up a social skill.

Psychoeducation—An approach to treatment that combines instruction with various therapeutic techniques.

Reinforcement—In social skills training, responding to a client's changed behavior in ways that will make the client want to perform the behavior again.

Role-playing—A technique used in social skills training and therapy in which participants act out roles relevant to real-life situations in order to change their attitudes and behaviors.

Shaping—A technique used in teaching social skills by prompting and reinforcing behaviors that come close to the desired behavior.

Social perspective-taking—A skill that involves a person's capacity to perceive or recognize other people's thoughts and feelings.

Another example is the application of social skills training to social phobia or shyness. People who suffer from social phobia or shyness are not ignorant of social cues, but they tend to avoid specific situations in which their limitations might cause them embarrassment. Social skills training can help these patients to improve their communication and social skills so that they will be able to mingle with others or go to job interviews with greater ease and self-confidence. Some studies indicate that the social skills training given to patients with shyness and social phobia can be applied to those with avoidant personality disorder, but more research is needed to differentiate among the particular types of social skills that benefit specific groups of patients, rather than treating social skills as a single entity. When trainers apply social skills training to the treatment of other **personality disorders**, they focus on the specific skills required to handle the issues that emerge with each disorder. For example, in the treatment of **obsessive-compulsive personality disorder** (OCD), social skills trainers focus on helping patients with OCD to deal with heavy responsibilities and **stress**.

People with disabilities in any age group can benefit from social skills training. Several studies demonstrate that children with developmental disabilities can acquire positive social skills with training. Extensive research on the effects of social skills training on children with attention-deficit/hyperactivity disorder shows that SST programs are effective in reducing these children's experiences of school failure or rejection as well as the aggressiveness and isolation that often develop in them because they have problems relating to others.

SST can be adapted to the treatment of depression with a focus on **assertiveness training**. Depressed patients often benefit from learning to set limits to others, to obtain satisfaction for their own needs, and to feel more self-confident in social interactions. Research suggests that patients who are depressed because they tend to withdraw from others can benefit from social skills training by learning to increase positive social interactions with others instead of pulling back.

There has been extensive research on the effective use of social skills training for the treatment of schizophrenia, in outpatient clinics as well as inpatient units. SST can be used to help patients with schizophrenia make better eye contact with other people, increase assertiveness, and improve their general conversational skills.

Social skills training in combination with other therapies

Social skills training is often used in combination with other therapies in the treatment of mental disorders. For example, in the treatment of individuals with alcohol dependence, social skills training has been used together with cognitive restructuring and coping skills training. Social skills training has also been integrated with exposure therapy, cognitive restructuring, and medication in the treatment of social phobia. Social skills training has been used within **family therapy** itself in the treatment of marital and family conflicts. Moreover, SST works well together with medication for the treatment of depression. For the treatment of schizophrenia, social skills training has often been combined with pharmacotherapy, family therapy, and assertive **case management**.

Precautions

Social skills training should rest on an objective assessment of the patient's actual problems in relating to other people.

It is important for therapists who are using SST to move slowly so that the patient is not overwhelmed by trying to change too many behaviors at one time. In addition, social skill trainers should be careful not to intensify the patient's feelings of social incompetence. This caution is particularly important in treating patients with social phobia, who are already worried about others' opinions of them.

An additional precaution is related to the transfer of social skills from the therapy setting to real-life situations. This transfer is called generalization or maintenance. Generalization takes place more readily when the social skills training has a clear focus and the patient is highly motivated to reach a realistic goal. In addition, social skills trainers should be sure that the new skills being taught are suitable for the specific patients involved.

Description

Techniques in social skills training

Therapists who use social skills training begin by breaking down complex social behaviors into smaller portions. Next, they arrange these smaller parts in order of difficulty, and gradually introduce them to the patients. For example, a therapist who is helping a patient learn to feel more comfortable at parties might make a list of specific behaviors that belong to the complex behavior called "acting appropriately at a party," such as introducing oneself to others; making conversation with several people at the party rather than just one other guest; keeping one's conversation pleasant and interesting; thanking the host or hostess before leaving; and so on. The patient would then work on one specific behavior at a time rather than trying to learn them all at once.

Such specific techniques as instruction, **modeling**, role-playing, shaping, feedback, and **reinforcement** of positive interactions may be used in SST. For example, instruction may be used to convey the differences among assertive, passive, and aggressive styles of communication. The technique of monitoring may be used to ask patients to increase their eye contact during a conversation. In role-playing exercises, group members have the opportunity to offer feedback to one another about their performances in simulated situations. For example, two members of the group may role-play a situation in which a customer is trying to return a defective purchase to a store. The others can then give feedback about the "customer's" assertiveness or the "clerk's" responses.

Content of social skills training

SST may be used to teach people specific sets of social competencies. A common focus of SST programs is communication skills. A program designed to improve people's skills in this area might include helping them with nonverbal and assertive communication and with making conversation. It might also include conversational skills that are needed in different specific situations, for example job interviews, informal parties, and dating. The skills might be divided further into such subjects as beginning, holding, and ending conversations, or expressing feelings in appropriate ways.

Another common focus of SST programs involves improving a patient's ability to perceive and act on social cues. Many people have problems communicating with others because they fail to notice or do not understand other people's cues, whether verbal or nonverbal. For example, some children become unpopular with their peers because they force their way into small play groups, when a child who has learned to read social signals would know that the children in the small group do not want someone else to join them, at least not at that moment. Learning to understand another person's spoken or unspoken messages is as important as learning conversational skills. A social skills program may include skills related to the perceptual processing of the conversation of other individuals.

Scheduling

Social skills training may be given as an individual or as a group treatment once or twice a week, or more often depending upon the severity of a patient's disorder and the level of his or her social skills. Generally speaking, children appear to gain more from SST in a peer group setting than in individual therapy. Social skill training groups usually consist of approximately 10 patients, a therapist, and a co-therapist.

Culture and gender issues

Social skills training programs may be modified somewhat to allow for cultural and gender differences. For example, eye contact is a frequently targeted behavior to be taught during social skills training. In some cultures, however, downcast eyes are a sign of respect rather than an indication of social anxiety or shyness. In addition, girls or women in some cultures may be considered immodest if they look at others, particularly adult males, too directly. These modifications can usually be made without changing the basic format of the SST program.

Generalization or transfer of skills

Current trends in social skills training are aimed at developing training programs that meet the demands of specific roles or situations. This need developed from studies that found that social skills acquired in one setting or situation are not easily generalized or transferred to another setting or situation. To assist patients in using their new skills in real-life situations, trainers use role-playing, teaching, modeling, and practice.

Preparation

Preparation for social skills training requires tact on the therapist's part, as patients with such disorders as social phobia or paranoid personality disorder may be discouraged or upset by being told that they need help with their social skills. One possible approach is through reading. The social skills therapist may recommend some self-help books on social skills in preparation for the treatment. Second, the therapist can ease the patient's self-consciousness or embarrassment by explaining that no one has perfect social skills. An additional consideration before starting treatment is the possibility of interference from medication side effects. The therapist will usually ask the patient for a list of all medications that he or she takes regularly.

One of the most critical tasks in preparation for social skills training is the selection of suitable target behaviors. It is often more helpful for the therapist to ask the patient to identify behaviors that he or she would like to change, rather than pointing to problem areas that the therapist has identified. The treatment should consider the patient's particular needs and interests. Whereas social skills training for some patients may include learning assertiveness on the job, training for others may include learning strategies for dating. Therapists can prepare patients for homework by explaining that the homework is the practice of new skills in other settings; and that it is as relevant as the therapy session itself.

Aftercare

Some studies strongly suggest the need for follow-up support after an initial course of social skills training. One study showed that follow-up support doubled the rate of employment for a group of patients with schizophrenia, compared to a group that had no follow-up.

Normal results

Outcome studies indicate that social skills training has moderate short-term effects, but limited long-term effects. SST programs that include social perspective-taking may have greater long-term effects than traditional SST programs based on cognitive-behavioral models. In general, social skills training tends to generalize or transfer to similar contexts rather than to contexts that are not similar to the training. SST programs for patients with developmental disabilities should include programming for generalization, so that the patients can transfer their newly acquired skills more effectively to real-life settings. One approach to improving generalization is to situate the training exercises within the patient's work, living, or social environment.

The benefits of social skills training programs include flexibility. The treatment can take place either as individual or group therapy, and new trainers can learn the techniques of SST fairly quickly. An additional advantage of SST is that it focuses on teaching skills that can be learned rather than emphasizing the internal or biological determinants of social adequacy. Future research should explore the integration of social skills training with the needs of families from different cultural backgrounds; the relationship between social skills training and different categories of mental disorders; the transfer of skills from therapeutic contexts to daily life; and improving patients' long-term gains from SST.

See also Assertiveness training; Bibliotherapy; Cognitive problem-solving skills training; Conduct disorder; Modeling; Peer groups

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Social workers

Definition

A social worker is a helping professional who is distinguished from other human service professionals by a focus on both the individual and his or her environment. Generally, social workers have at least a bachelor's degree from an accredited education program and in most states they must be licensed, certified, or registered. A Master's in Social Work is required for those who provide **psychotherapy** or work in specific settings such as hospitals or nursing homes.

Description

Social workers comprise a profession that had its beginnings in 1889 when Jane Addams founded Hull House and the American settlement house movement in Chicago's West Side. The ethics and values that informed her work became the basis for the social work profession. They include respect for the dignity of human beings, especially those who are vulnerable, an understanding that people are influenced by their environment, and a desire to work for social change that rectifies gross or unjust differences.

The social work profession is broader than most disciplines with regard to the range and types of problems addressed, the settings in which the work takes place, the levels of practice, interventions used, and populations served. It has been observed that social work is defined in its own place in the larger social environment, continuously evolving to respond to and address a changing world. Although several definitions of social work have been provided throughout its history, common to all definitions is the focus on both the individual and the environment, distinguishing it from other helping professions.

Social workers may be engaged in a variety of occupations ranging from hospitals, schools, clinics, police departments, public agencies, court systems to private practices or businesses. They provide the majority of mental health care to persons of all ages in this country, and in rural areas they are often the sole providers of services. In general, they assist people to obtain tangible services, help communities or groups provide or improve social and health services, provide counseling and psychotherapy with individuals, families, and groups, and participate in policy change through legislative processes. The practice of social work requires knowledge of human development and behavior, of social, economic and cultural institutions, and of the interaction of all these factors.

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Sodium amobarbital *see* **Barbiturates**

Sodium pentobarbital *see* **Barbiturates**

Somatization and somatoform disorders

Definition

Somatization is a term that describes the expression of psychological or mental difficulties through physical symptoms. Somatization takes a number of forms, ranging from preoccupation with potential or genuine but mild physical problems to the development of actual physical pain, discomfort, or dysfunction. The *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*, the professional handbook clinicians use to diagnose mental disorders, describes seven disorders under the category of somatoform disorders. These disorders are **somatization disorder**, **undifferentiated somatoform disorder**, **conversion disorder**, **pain disorder**, **hypochondriasis**, **body dysmorphic disorder**, and somatoform disorder not otherwise specified. Somatization appears to be fairly common, and a somatoform disorder **diagnosis** is not warranted unless symptoms cause significant distress or disability.

Description

Somatization disorder is characterized by a history of multiple unexplained medical problems or physical

KEY TERMS

Dysmorphic—Malformed.

Somatization—The expression of mental or psychological experiences through physical symptoms.

Somatoform—Referring to physical symptoms with a psychological origin.

complaints beginning prior to age 30. In the nineteenth and early twentieth centuries, somatization disorder was known as Briquet's syndrome or hysteria—a more generic term for such a condition. People with somatization disorder report symptoms affecting multiple organ systems or physical functions, including pain, gastrointestinal distress, sexual problems, and symptoms that mimic neurological disorders. Although medical explanations for their symptoms cannot be identified, individuals with somatization disorder experience genuine physical discomfort and distress. Review of their medical histories will usually reveal visits to a number of medical specialists, and many patients take numerous medications prescribed by different doctors, running the risk of dangerous drug interactions.

Undifferentiated somatoform disorder is similar to somatization disorder, but may involve fewer symptoms, have a shorter duration or begin after the age of 30. Common symptoms include chronic **fatigue**, loss of appetite, gastrointestinal distress or problems involving the genitals or urinary tract. This diagnosis is appropriate for patients with symptoms of somatization disorder who do not meet all diagnostic criteria.

Conversion disorder is marked by unexplained sensory or motor symptoms that resemble those of a neurological or medical illness or injury. Common symptoms include paralysis, loss of sensation, double vision, **seizures**, inability to speak or swallow and problems with coordination and balance. Symptoms often reflect a naive understanding of the nervous system, and physicians often detect conversion disorder when symptoms do not make sense anatomically. For instance, a patient may report loss of both touch and pain sensation on one side of the body, when, in fact, a genuine lesion would result in loss of touch and pain sense on opposite sides of the body. The name conversion disorder reflects a theoretical understanding of the disorder as a symbolic conversion of a psychological conflict into a concrete physical representation. Interestingly, patients with conversion disorder may not express the level of distress one would expect from someone with a disabling neurological condition. This phenomenon is traditionally called *la belle indifference*.

The primary feature of pain disorder is physical pain that causes significant distress or disability or leads an individual to seek medical attention. Pain may be medically unexplained, or it may be associated with an identifiable medical condition but far more severe than the condition would warrant. Common symptoms include headache, backache and generalized pain in muscles and joints. Pain disorder can be severely disabling, causing immobility that prevents patients from working, fulfilling family responsibilities or engaging in social activities. Like patients with somatization disorder, people with pain disorder often have a history of consultations with numerous physicians.

Hypochondriasis is diagnosed when a person is excessively concerned by fears of having a physical disease or injury. Individuals with hypochondriasis usually do not complain of disabling or painful symptoms. Instead, they tend to overreact to minor physical symptoms or sensations, like rapid heartbeat, sweating, small sores or fatigue. Many people with hypochondriasis develop fears in response to the illness or death of a friend or family member or after reading about a condition or seeing a feature on television. Hypochondriacal fears can be confined to a single disease or may involve a number of different physical concerns. Individuals with hypochondriasis seek frequent reassurance by consulting physicians or talking about their fears, yet these efforts provide only temporary relief from their fears. Although hypochondriasis is usually not as disabling as somatoform disorders involving the development of actual physical symptoms, it can put **stress** on relationships or reduce work productivity through time lost to frequent medical appointments and tests.

Body dysmorphic disorder is characterized by preoccupation with a defect in physical appearance. Often the defect of concern is not apparent to other observers, or if there is a genuine defect it is far less disfiguring than the patient imagines. Common preoccupations include concerns about the size or shape of the nose, skin blemishes, body or facial hair, hair loss, or "ugly" hands or feet. Individuals with body dysmorphic disorder may be extremely self-conscious, avoiding social situations because they fear others will notice their physical defects or even make fun of them. They may spend hours examining the imagined defect or avoid mirrors altogether. Time-consuming efforts to hide the defect, such as application of cosmetics or adjustments of clothing or hair, are common. Many people with body dysmorphic disorder undergo procedures like plastic surgery or cosmetic dentistry, but are seldom satisfied with the results.

Somatoform disorder, not otherwise specified, is diagnosed when somatoform symptoms are present but criteria for another somatoform disorder are not met. *DSM-IV-TR* includes several examples of symptoms that could merit this diagnosis, including false pregnancy, and hypochondriacal fears or unexplained physical symptoms of recent onset or short duration.

There is some disagreement among researchers about the *DSM-IV-TR* somatoform disorders category. Some have argued that hypochondriasis and body dysmorphic disorder are more similar to **obsessive-compulsive disorder** than to other somatoform disorders, while others think hypochondriasis may be more appropriately classified with the anxiety disorders.

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Somatization disorder

Definition

Somatization disorder is a psychiatric condition marked by multiple medically unexplained physical, or somatic, symptoms. In order to qualify for the **diagnosis** of somatization disorder, somatic complaints must be serious enough to interfere significantly with a person's ability to perform important activities, such as work, school or family and social responsibilities, or lead the person experiencing the symptoms to seek medical treatment.

Somatization disorder has long been recognized by psychiatrists and psychologists, and was originally called Briquet's syndrome in honor of Paul Briquet, a French physician who first described the disorder in the nineteenth century. It is included in the category of somatoform disorders in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR),* the professional handbook that aids clinicians in diagnosing patients' mental disorders. The term "somatoform" means that the physical symptoms have a psychological origin.

KEY TERMS

Somatization—Conversion of mental experiences into physical symptoms.

Description

Individuals with somatization disorder suffer from a number of vague physical symptoms, involving at least four different physical functions or parts of the body. The physical symptoms that characterize somatization disorder cannot be attributed to medical conditions or to the use of drugs, and individuals with somatization disorder often undergo numerous medical tests (with negative results) before the psychological cause of their distress is identified. They often use impressionistic and colorful language to describe their symptoms, describing burning sensations, pains that move from place to place, strange tastes on the tongue, tingling, or tremors. While many symptoms resemble those associated with genuine diseases, some of the symptoms reported by people with somatization disorder are not. The individual usually visits many different physicians, but the information they provide about the patient's symptoms can be inconsistent. It is important to note that while the physical symptoms of somatization disorder frequently lack medical explanations, they are not intentionally fabricated. The typical person with somatization disorder has suffered from physical pain, discomfort, and dysfunction for an extended period of time and consulted several doctors; they are hopeful that they one can be found who can identify the cause of their illness and provide relief.

Somatization disorder can be dangerous, since patients may end up taking several different medications, thereby risking harmful drug interactions.

Causes and symptoms

Causes

DEFENSE AGAINST PSYCHOLOGICAL DISTRESS. One of the oldest theories about the cause of somatization disorder suggests that it is a way of avoiding psychological distress. Rather than experiencing depression or anxiety, some individuals will develop physical symptoms.

According to this model, somatization disorder is a defense against psychological pain that allows some people to avoid the **stigma** of a psychiatric diagnosis. While getting the care and nurturing they need from doctors and others who are responsive to their apparent medical illnesses, many patients are encouraged to continue their manipulative behavior.

Many patients described by Sigmund Freud would be diagnosed today with somatization disorder. His patients were usually young women who complained of numerous physical symptoms. In the process of speaking with Freud, they would often recall a number of distressing memories; discussing these memories frequently led to the relief of physical symptoms. These cases formed the foundation of Freud's psychoanalytic treatment. Although this theory offers a plausible explanation for somatization disorder, research indicates that people with multiple physical symptoms are actually more likely to report psychiatric symptoms than those with few physical problems. These findings appear to support a connection between psychological and physical distress, but are inconsistent with the idea that physical symptoms offer a defense against overt psychiatric symptoms.

HEIGHTENED SENSITIVITY TO PHYSICAL SENSATIONS. An alternative theory suggests that somatization disorder arises from a heightened sensitivity to internal sensations. People with somatization disorder may be keenly aware of the minor pains and discomforts that most people simply ignore. A similar theory has been offered to account for **panic disorder**. Studies have shown that people with panic disorder are particularly sensitive to internal sensations like breathing rate and heartbeats, which may lead them to react with intense fear to minor internal changes. The physiological or psychological origins of this hypersensitivity to internal sensations and their relevance to somatization disorder are still not well understood.

CATASTROPHIC THINKING ABOUT PHYSICAL SENSATIONS. According to these thoughts, somatization disorder results from negative beliefs and exaggerated fears about the significance of physical sensations. Individuals with somatization disorder are thus more likely to believe that vague physical symptoms are indicators of serious disease and to seek treatment for them. For instance, someone with somatization disorder may fear that a headache signals a **brain** tumor, or that shortness of breath indicates the onset of asthma. When their doctors can find no medical explanation for the symptoms, the patients may fear that they have a rare disease; they frantically look for specialists who can provide a diagnosis. Anxiety causes them to focus even more intensely on their symptoms, which in turn become more disabling. Many people with somatization disorder reduce or eliminate many activities out of fear that exertion will worsen their symptoms. With fewer activities to distract them from their symptoms, they spend more time worrying about physical problems, resulting in greater distress and disability.

Symptoms

Gastrointestinal (GI) complaints, such as nausea, bloating, diarrhea, and sensitivities to certain foods are common, and at least two different GI symptoms are required for the diagnosis. Sexual or reproductive symptoms, including pain during intercourse, menstrual problems, and **erectile dysfunction** are also necessary features for a diagnosis for somatization disorder. Other frequent symptoms are headaches, pain in the back or joints, difficulty swallowing or speaking, and urinary retention. To qualify for the diagnosis, at least one symptom must resemble a neurological disorder, such as **seizures**, problems with coordination or balance, or paralysis.

Demographics

According to the *DSM-IV-TR*, somatization disorder is rare in males in the United States, although higher rates are seen among males from some cultural and ethnic groups. The *DSM-IV-TR* estimates that between 0.2% and 2% of women, and less than 0.2% of men, suffer from somatization disorder in the U.S. Sex ratios may arise from different rates of seeking treatment. However, studies of unexplained somatic symptoms in the general population find less striking differences in rates between men and women. Specific symptoms may vary across cultures. For example, the *DSM-IV-TR* notes that the sensation of worms in the head or ants crawling under the skin are sometimes reported in African and South Asian countries, but rarely seen in North American patients.

Diagnosis

To receive a diagnosis of somatization disorder, the individual must have a history of multiple physical complaints that began before age 30 and that continued for several years (*DSM-IV-TR*). These symptoms must cause significant impairment to social, occupational or other areas of functioning—or lead the patient to seek medical treatment.

Each of the following four criteria must be met.

- The individual must report a history of pain affecting at least four different parts or functions of the body. Examples include headaches, back, joint, chest or abdominal pain, or pain during menstruation or sexual intercourse.

- A history of at least two gastrointestinal symptoms, such as nausea, bloating, vomiting, diarrhea, or food intolerance must be reported.
- There must be a history of at least one sexual or reproductive symptom, such as lack of interest in sex, problems achieving erection or ejaculation, irregular menstrual periods, excessive menstrual bleeding, or vomiting throughout pregnancy.
- One symptom must mimic a neurological condition. Examples include weakness, paralysis, problems with balance or coordination, seizures, **hallucinations**, loss of sensations such as touch, seeing, hearing, tasting, smelling—or difficulty swallowing or speaking, or **amnesia** and loss of consciousness. Pseudo-neurologic symptoms like these are the primary characteristics of another somatoform disorder known as "conversion disorder."

If a thorough medical evaluation reveals no evidence of an underlying medical- or drug- or medication-induced condition, the diagnosis of somatization disorder is likely. People with genuine medical conditions can qualify for the diagnosis if the level of functional impairment reported is more than would be expected based on medical findings. The symptoms must not be intentionally produced. If the patient is feigning symptoms, a diagnosis of **factitious disorder** or **malingering** would most likely be considered.

Treatments

Cognitive behavior therapy

Cognitive-behavioral therapy (CBT) for somatization disorder focuses on changing negative patterns of thoughts, feelings, and behavior that contribute to somatic symptoms. The cognitive component of the treatment focuses on helping patients identify dysfunctional thinking about physical sensations. With practice, patients learn to recognize catastrophic thinking and develop more rational explanations for their feelings. The behavioral component aims to increase activity. Patients with somatization disorder have usually reduced their activity levels as a result of discomfort or out of fear that activity will worsen symptoms. CBT patients are instructed to increase activity gradually while avoiding overexertion that could reinforce fears. Other important types of treatment include relaxation training, sleep hygiene, and communication skills training. Preliminary findings suggest that CBT may help reduce distress and discomfort associated with somatic symptoms; however, it has not yet been systematically compared with other forms of therapy.

Medications

Antidepressant medications may help to alleviate symptoms of somatization disorder. According to one study, patients with somatization disorder who took the antidepressant **nefazodone** (Serzone) showed reductions in physical symptoms, increased activity levels, and lower levels of anxiety and depression at the end of treatment.

Prognosis

Untreated somatization disorder is usually a chronic condition, though specific symptoms can come and go and overall severity may fluctuate over time. Somatization disorder poses a serious problem for society, since many who suffer from it become functionally disabled and unable to work. In addition, patients with unexplained physical symptoms strain already overburdened health care resources. Unexplained physical symptoms are extremely common among patients visiting general practitioners, with some estimates suggesting that over two-thirds of general medical patients have symptoms that cannot be explained by medical tests. Fortunately, there is preliminary evidence that **psychotherapy** and medication can effectively reduce symptoms and disability.

Prevention

Greater awareness of somatization disorder, particularly among physicians, can help them identify individuals with somatization disorder, and help these patients get appropriate psychological or psychiatric treatment.

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Somnambulism *see* **Sleepwalking disorder**

Somnote *see* **Chloral hydrate**

Sonata *see* **Zaleplon**

Specific phobias

Definition

Specific phobia is a type of disorder in which the affected individual displays a marked and enduring fear of specific situations or objects. Individuals with specific phobias experience extreme fear as soon as they encounter a defined situation or object, a phobic stimulus. For example, an individual with a specific phobia of dogs will become anxious when coerced to confront a dog. The specific phobia triggers a lot of distress or significantly impairs an affected individual.

Mental health professionals use the ***Diagnostic and Statistical Manual of Mental Disorders*** (the *DSM*) to diagnose mental disorders. The 2000 edition of this manual (the Fourth Edition Text Revision, also called the *DSM-IV-TR*) classifies specific phobia as a type of anxiety disorder. Formerly, specific phobia was known as simple phobia. In the last few years, mental health professionals have paid more attention to specific phobias.

Description

Specific phobia has a unique position among the anxiety disorders in that individuals with this disorder do not experience pervasive anxiety nor do they seek treatment as readily as individuals with other anxiety disorders. Unlike individuals with other anxiety disorders, the fear of individuals with specific phobias is limited to defined situations or objects. Individuals with specific phobias experience impairment or a significant amount of anguish. They may lead restricted lifestyles depending upon the phobia type. Adults and adolescents with specific phobias recognize that their fear is unreasonable. Children, on the other hand, may not recognize that their fear of the phobic stimulus is unreasonable or extreme.

The types of specific phobias include situational, object, and other. The situational type is diagnosed if an individual's fear is cued by a defined situation. Examples include situations such as flying, enclosed places, tunnels, driving, bridges, elevators, or public transportation. Object types include animal, natural environment, and blood-injection-injury types. Animal type is diagnosed if an individual's fear is cued by animals or insects. Natural environment type is diagnosed if an individual's fear is cued by storms, water, or heights. Blood-injection-injury type is diagnosed if an individual's fear is cued by seeing an injury or blood or by an injection or other invasive medical treatment. Other type is diagnosed if an individual's fear is cued by other stimuli such as fears of vomiting, choking, becoming ill, and falling down if far from a means of physical support, and a child's fears of loud noises or characters in costumes.

Researchers have found that the frequency of type for adults in clinical settings, from least to most frequent, is: animal, blood-injection-injury, natural environment, and situational. The most common phobias for community samples, however, include phobias of heights, mice, spiders, and insects.

Causes and symptoms

Causes

The development of a specific phobia may be determined by a variety of factors. Behavioral, cognitive, and social theories of learning and conditioning, psychodynamic models such as the psychoanalytic theory of Freud, physiological studies of the **brain**, family background and genetic predisposition, variations in sociocultural themes, and theories on trauma can influence the development of specific phobia disorder. Some theorists propose that biological researchers have ignored specific phobias because pharmacological treatment is not the treatment of choice for this disorder.

LEARNING AND CONDITIONING CAUSES. As of 2002, research on phobias focuses on information-processing, learning, and conditioning themes. Learning to experience fear is the core of a conditioning perspective. Informational and instructional factors can result in the formation of fears. For example, an individual who frequently hears of plane crashes in the news may develop a specific phobia of flying. Research shows that individuals with specific phobias pay more attention to information about danger than do individuals who do not have specific phobias. Vicarious acquisition occurs when an individual witnesses a traumatic event or sees another individual behave with fear when confronting a phobic stimulus. Direct conditioning occurs when an individual is frightened by a phobic stimulus.

A major determinant of specific phobias is conditioning. Association and avoidance are types of conditioning. In association conditioning, a stimulus that was initially neutral begins to trigger an anxiety response. For example, if an individual was driving one day and experienced a strong anxiety response, an association may form between driving and anxiety. Individuals do not learn to become phobic until they begin to avoid. In avoidance conditioning, individuals learn to avoid a stimulus that triggers anxiety. Every time individuals avoid the phobic stimulus—driving, for example—they are rewarded by the relief from anxiety.

TRAUMATIC CAUSES. A determinant of specific phobias includes traumas. For example, individuals who have been attacked by a dog may develop a specific phobia disorder and become conditioned to fear dogs. Individuals who observe others experiencing a trauma (the others are "modeling" behavior for the individual who will be affected) may become predisposed to developing specific phobia disorder. For example, individuals who witness people falling from a building may develop a specific phobia disorder. Phobias with a traumatic origin may develop acutely, or, in other words, have a more sudden onset than other phobias that develop more gradually.

PSYCHODYNAMIC CAUSES. Psychodynamic theorists explain that phobias emerge because individuals have impulses that are unacceptable, and they repress these impulses. More specifically, Freud proposed that phobias emerge because of an unresolved oedipal conflict. According to Freud's theory, an oedipal conflict is a developmental conflict that emerges during the third (or oedipal) stage of Freud's psychosexual development stages. During this stage, a conflict emerges with regard to the triad of father, mother, and child. The conflict concerns the sexual impulses that the child has toward the parent of the opposite gender and the hostile impulses that the child has towards the parent of the same gender. During this stage, the developmental conflict concerns a resolution of oedipal issues. Psychoanalysts propose that when repression does not work, individuals with phobias displace their anxiety connected to the unresolved oedipal conflict upon a situation or object that is less relevant. The feared situation or object symbolizes the source of the conflict. For example, a specific phobia may be connected to an individual's conflict about aggressive or sexual thoughts and feelings. In one sense, a phobia protects individuals from realizing their emotional issues.

KEY TERMS

Axis I—Axis I offers mental health professionals a diagnostic coding domain for listing disorders or conditions that are not classified as personality disorders and mental retardation.

Oedipal conflict—A developmental conflict that emerges during the third or oedipal stage of Freud's psychosexual development stages. During this stage, a conflict emerges with regard to the triad of father, mother, and child. The conflict concerns the sexual impulses that the child has toward the parent of the opposite gender and the hostile impulses that the child has towards the parent of the same gender. During this stage, the developmental conflict concerns a resolution of oedipal issues.

The case of Hans, a boy with a horse phobia, is Freud's paradigm example of a phobia. Freud attributed Hans' fear of horses to an oedipal conflict that was not resolved, and he explained that Hans repressed his sexual feelings for his mother and his wish that his father would die. Freud proposed that Hans feared that his father would discover his wish, repressed his wish to attack his father, and displaced his fear of his father's aggression onto horses. The young boy resolved the conflict of loving and hating his father by hating horses rather than admitting that he had aggressive feelings towards his father. Hans was better able to avoid the feared horses than his father. Thus, the phobia in the case of Hans represents a compromise of intrapsychic movement.

PHYSIOLOGICAL CAUSES. Some research has suggested that the high activation of brain pathways that correspond to the cognitive and emotional constituents of anxiety biologically predispose individuals to specific phobias.

GENETIC AND FAMILY CAUSES. Although specific phobia is frequently attributed to environmental issues such as **modeling**, learning by association, and negative **reinforcement**, genetic predisposition can influence this

disorder. An individual who has a family member with a specific phobia is at an increased risk for developing this disorder. Some research indicates that the pattern of types are similar within families. For example, a first-degree biological relative of individuals with a situational type is likely to have phobias of situations. Studies indicate that the blood and injury phobias have strong familial patterns.

SOCIOCULTURAL CAUSES. There is a paucity of information about cultural differences in specific phobias. Phobia content may vary by culture. Fear of a phobic stimulus such as magic or spirits, present in several cultures, is diagnosed as a specific phobia only if the fear is excessive for a particular culture and if the fear triggers major distress or interferes with functioning. Some research indicates that African Americans are more likely than whites to report specific phobias. Some studies show that specific phobias are less common among whites born in the U.S. or immigrant Mexican-Americans than among Mexican-Americans born in the U.S. Research suggests mixed data with regard to socioeconomic level, with some data associating specific phobia disorder with a lower socioeconomic level.

PERSONAL VARIABLES. Studies suggest a relationship between age and specific phobia. Research indicates some connections between the age of individuals with specific phobias and insight into the extreme quality of their fears. Insight increases with age. Children, unlike adults and adolescents, often do not report feelings of distress about having phobias. Insight into the unreasonable nature of the fear is not required for a **diagnosis** of specific phobia in children. The animal and natural environment types of specific phobia are common and generally transitory in children. Some studies indicate a connection between gender and specific phobia. Research shows that specific phobias from the animal type are more common among women. Some studies suggest that women are more likely to report specific phobias and to seek treatment than men.

Symptoms

DSM-IV-TR delineates seven diagnostic criteria for specific phobia:

- Significant and enduring fear of phobic stimulus: Patients with specific phobia display marked and enduring fear when they encounter a defined situation or object, the phobic stimulus.
- Anxiety response to phobic stimulus: Patients with specific phobia display anxiety as soon as they confront the phobic stimulus. When they confront the phobic stimulus, a defined situation or object, patients with specific phobia may experience a **panic attack** related to the specific situation. Children may cry, cling, freeze, or display tantrums when they express their anxiety in the face of the phobic stimulus.
- Recognition: Although adolescents and adults realize that their fear is unreasonable and disproportionate to the situation, children may not recognize that their fear is excessive.
- Avoidance: Individuals with specific phobia avoid the phobic stimulus or endure it with deep distress and anxiety.
- Impairment and distress: Individuals with specific phobia display avoidance, distress, and anxious anticipation when they encounter the phobic stimulus. Their avoidance reactions interfere with their daily functioning, or they express significant distress about having a phobia.
- Duration: To diagnose specific phobia in a patient who is under 18 years of age, the duration of the disorder needs to be at least six months.
- Not accounted for by another disorder: A diagnosis of specific phobia is assigned if the phobic avoidance, panic attacks, or anxiety related to the defined situation or object are not better accounted for by other disorders.

Demographics

General United States population

Specific phobias are common. The prevalence rates of specific phobia in community samples range from 4% to 8%. Over the course of a lifetime, the prevalence estimates in community samples range from 7.2% to 11.3%.

High-risk populations

Individuals whose family members have specific phobia are at a higher risk for developing this disorder.

Cross-cultural issues

Prior to assigning a diagnosis of specific phobia, clinicians need to consider whether a patient's fear is extreme in the context of a particular culture and whether the phobia causes difficulties in daily functioning or triggers a lot of distress. Further research is needed on the effects of culture upon the symptoms of specific phobia.

Gender issues

There are twice as many women with specific phobia than there are men with this disorder. The gender ratio variable varies depending upon the type of specific phobia. Approximately 75%–90% of people with the animal, situational, and natural environment types are female.

Approximately 55%–70% of people with the blood-injection-injury subtype are female. For height phobias, there are fewer women than men than for other specific phobia types; however, illness phobias are more common in men.

Diagnosis

The diagnosis of specific phobia is complicated by factors such as degree of impairment and differential diagnosis. Although fears of specified situations or objects are common, a diagnosis of specific phobia relies on the degree of sufficient impairment.

With regard to differentiating specific phobia types, factors such as the focus of fear and the predictability and timing of the reaction to the phobic stimulus across the specific phobia types can assist clinicians to differentiate. With regard to differentiating specific phobia from other disorders, there are several disorders with similar symptoms. They include **panic disorder** with **agoraphobia**, **social phobia**, **post-traumatic stress disorder**, **obsessive-compulsive disorder**, **hypochondriasis**, **schizophrenia**, delusional, and other psychotic disorders. Generally, a diagnosis of specific phobia rather than panic disorder is made when there are no spontaneous panic attacks and no fear of panic attacks. It is often difficult to differentiate specific phobia, situational type, from panic disorder with agoraphobia. Specific phobia, situational type, is commonly diagnosed when an individual displays situational avoidance without unexpected and recurrent panic attacks. On the other hand, panic disorder with agoraphobia is diagnosed if an individual experiences an initial onset of panic attacks that are not anticipated and subsequently experiences avoidance of several situations considered triggers of panic attacks. Although individuals with specific phobia, unlike individuals with panic disorder with agoraphobia, do not display enduring anxiety, anxious anticipation may occur when confrontation with a phobic stimulus is more likely to occur. *DSM-IV-TR* outlines differentiating factors as the type and number of panic attacks, the number of avoided contexts, and the focus of the fear. At times, both diagnoses, specific phobia and panic disorder with agoraphobia, need to be assigned.

Psychological measures

Measures used to diagnose specific phobia include behavioral observation, clinical interviews, physiological evaluation, and self-report measures. The Behavioral Avoidance Task (BAT) is a common behavioral observation method used to assess specific phobia. Often, the diagnosis of specific phobia is made on the basis of an individual's responses to semistructured interviews such as the Anxiety Disorders Interview Schedule for *DSM-IV* (ADIS-IV) and the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-IV). To assist in differential diagnosis between specific phobias and other disorders with similar characteristics, clinicians use the Anxiety Disorders Interview Schedule for *DSM-IV* (ADIS-IV). Physiological evaluations usually include heart rate monitors. Self-report questionnaires include measures such as the SUDS (subjective units of discomfort/distress scale), the most frequently used self-report measure, the Fear Survey Schedule (FSS-III), and the Mutilation Questionnaire, specifically for measuring fear of the blood type of specific phobia.

This woman suffers from claustrophobia— the fear of enclosed spaces. *(Nathan Benn/CORBIS. Photo reproduced by permission.)*

Time of onset/symptom duration

Generally, the initial symptoms of specific phobia occur when an individual is a child or a young adolescent. The type of phobia determines the age of onset. The blood, animal, and natural environment types begin when an individual is a child; however, many new cases of the natural environment type occur when an individual is a young adult. The onset for the height type begins in adolescence. The onset age for the situational type occurs in childhood, but peaks again in the mid-twenties. There is no specific onset age for phobias with a traumatic origin.

Individual variations in specific phobia

Classification systems distinguish between individuals with different types of specific phobias. The types of specific phobia, situation, object, and other, relate to particular features such as the age, gender, and culture of an individual. Some researchers propose that to distinguish individual differences in treatment planning, it is more

helpful to simply name the specific phobia rather than to use the type classification system. For example, researchers have found that for the animal type, some animals such as a tiger or a bear did not trigger disgust for tiger-phobic or bear-phobic individuals, but other animals such as a spider triggered disgust for some spider phobic individuals, but did not trigger disgust for other spider phobic individuals.

Dual diagnoses

Specific phobia often occurs with other disorders of mood and anxiety, and with substance-related disorders. When specific phobias occur with other disorders in clinical contexts, the primary diagnosis is associated with greater distress than is the specific phobia. The blood-injury-injection type of specific phobia may occur with physical symptoms such as vasovagal fainting. The vasovagal fainting response is characterized by a short heart rate acceleration and blood pressure elevation. Then, the heart rate decelerates and the blood pressure drops. Research shows that individuals who have one specific phobia type are more likely to have other phobias of the same type.

Treatments

Specific phobias are highly treatable. They are most effectively treated by psychological rather than biological treatments. The primary goal of most treatments of specific phobias is to reduce fear, phobic avoidance, impairment, and distress. Approximately 12%–30% seek treatment for specific phobias.

Cognitive-behavioral therapy

Cognitive-behavioral therapy has been effective in treating specific phobias. There has not been much research on the effects of cognitive therapy alone on specific phobias. Cognitive therapists challenge fearful thoughts and replace them with more positive thoughts. Although some studies show benefits in that cognitive therapy may assist patients to decrease anxiety related to their exposure exercises, research indicates that cognitive therapy alone is probably not an effective treatment for specific phobia. Researchers suggest adding panic management strategies such as cognitive restructuring to assist with behavioral treatments.

Several studies indicate that real-life (in vivo) desensitization or exposure is the most effective and long-lasting treatment for a broad range of specific phobias. **Systematic desensitization** includes a process by which individuals unlearn the association between the phobic stimulus and anxiety. Incremental exposure involves the patient's gradual facing of the phobic stimulus through a series of graded steps. Wolpe's imagery desensitization is suggested so that patients with specific phobias can face the fear in imagery prior to attempting in vivo exposure. Unlike many of the other treatments, the treatment gains of in vivo exposure are maintained upon follow-up. Some desensitization treatments employ flooding as a useful strategy. When flooding is used, patients maintain a high anxiety level without retreating. Similar to desensitization, flooding can be used both in imagination and in vivo. Flooding is not suggested for most individuals because it can trigger a higher level of sensitization and fear reinforcement. For in vivo treatment, a patient needs to be highly motivated because the treatment may lead to temporary discomfort. The primary reasons for poor **compliance** with cognitive-behavioral treatment include lack of time, anxiety, and low motivation.

Psychodynamic therapy

Psychodynamic psychotherapy, or insight-oriented therapy assists patients to become more aware of the symbolic nature of their anxiety and to explore traumatic past events. Insight-oriented therapy is a psychodynamic therapy that aims to expose and reduce patients' unconscious conflicts, increase patients' understanding of their underlying thoughts, and assist patients to gain conscious control over their psychological conflicts. In psychodynamic therapy, for example, patients may discover that their anxiety may be connected to aggressive or sexual feelings and thoughts.

Group therapy

There is little research on **group therapy** for specific phobia disorder. Some studies suggest that group treatment has been effective for dental and spider phobias.

Medications

There has been a paucity of research on the relationship between medication and specific phobia. Generally, pharmacotherapy has not been considered to be a treatment of choice for individuals with specific phobias. Benzodiazepines, however, (medications that slow the central nervous system to ease nervousness and tension) may decrease anticipatory anxiety prior to an individual's entrance into a phobic situation. A low dose of a benzodiazepine such as **clonazepam** (Klonopin) or **alprazolam** (Xanax) is indicated to decrease some fear arousal prior to in vivo exposure. The reduction of symptoms, however, may interfere with the treatment. Prior to beginning in vivo exposure, an antidepressant such as **sertraline** (Zoloft) or **paroxetine** (Paxil) is suggested to increase motivation for undertaking an uncomfortable

treatment. **Beta blockers** can assist individuals to confront the specific phobia.

Alternative therapies

Research shows some benefits for specific phobias with applied relaxation. Relaxation training includes abdominal breathing and muscle relaxation on a regular basis. Studies have indicated that applied muscle tension has been highly effective for individuals with blood type phobias who faint in that the treatment triggers an early response. When using applied tension, therapists request that patients tense their muscles several times. The repeated muscle tensing results in a temporary increase in blood pressure and prevents fainting when patients see blood. Similar to in vivo exposure, the gains from applied tension are maintained upon follow-up. Some alternative therapies include immersive virtual reality, **hypnotherapy**, eye-movement desensitization and reprocessing (EMDR), and energy balance approaches such as massage and **acupuncture**.

Prognosis

If specific phobias exist in adolescence, they have a greater chance of persisting in early adulthood. Specific phobias that continue into adulthood generally become chronic if they are not treated. Furthermore, there is a greater chance for an individual diagnosed with specific phobia to develop new phobias as a young adult. Phobias contracted during childhood or adolescence that continue when individuals become young adults remit approximately 20% of the time. Individuals with specific phobias do not often seek treatment. For those who seek treatment, research suggests that compared to individuals with specific phobias whose fear diminishes slowly during exposure, individuals with specific phobias whose fear diminishes more rapidly have a better prognosis for recovery.

A consideration of prognosis takes into account the distinction between fear onset and phobia onset. Studies indicate that individuals with specific phobias of animal, blood, heights, and driving had a fear onset nine years earlier than their phobia onset. Some studies have shown that generalized anxiety level, severity of symptoms, and prior experience with the phobic stimulus are factors that have been associated with treatment outcome.

Although most mental health professionals consider specific phobia that begins in childhood to be a benign disorder, it can last for years if left untreated. Some studies indicate, however, that specific phobia does not become worse and usually diminishes as an individual ages. Without treatment, the prognosis is poor for an individual who has several phobias.

Prevention

Early detection is a key to assisting individuals with mild cases of specific phobia to seek treatment to prevent the development of full-blown cases of the disorder. Individuals who are at risk for developing specific phobia as well as individuals who already have been diagnosed with specific phobia need to avoid caffeine because caffeine can increase arousal. Further research is needed to discover variables that predict the reason that only certain individuals will develop specific phobias after conditioning or acquiring information that leads to fear.

See also Anxiety-reduction techniques; Generalized anxiety disorder; Exposure treatment

Resources

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American Psychological Association. 750 First Street NE, Washington, D.C. 20002-4242. (202) 336-5500. <http://www.apa.org>.

Anxiety Disorders Association of America (ADAA). 11900 Parklawn Drive, Suite 100, Rockville, MD. 20852-2624. (301) 231-9350. <http://www.adaa.org>.

Phobics Anonymous. P.O. Box 1180, Palm Springs, CA. 92213. (760) 322-COPE.

Judy Koenigsberg, Ph.D.

SPECT *see* **Single photon emission computed tomography**

Speech therapy *see* **Speech-language pathology**

Speech-language pathology

Definition

Speech-language pathology is the treatment for the improvement or cure of communication disorders, including speech, language, and swallowing disorders. The term used to describe professionals in this discipline is speech and language pathologist (SLP).

Description

The discipline of speech-language pathology includes professionals that are trained in the techniques, strategies, and interventions designed to improve or correct communication disorders. Communication disorders include disorders of speech, language, and swallowing.

In 2000, there were nearly 88,000 speech-language pathologists in the United States certified by the American Speech-Language-Hearing Association (ASHA), and an additional 13,000 audiologists, who often work with speech pathologists to diagnose disorders. Speech disorders treated by speech-language pathologists include voice disorders (abnormalities in pitch, volume, vocal quality, or resonance or duration of sounds), articulation disorders (problems producing speech sounds), and fluency disorders (impairment in speech fluency, such as **stuttering**). Language disorders include developmental or acquired conditions that lead to difficulties in understanding or producing language. Speech-language pathologists participate in the screening, assessment, and treatment of patients who experience one or a combination of these disorders.

A young girl repeats sounds after the speech pathologist. *(Photo Researchers, Inc. Reproduced by permission.)*

Persons with isolated speech sound disorders are often helped by articulation therapy, in which they practice repeating specific sounds, words, phrases, and sentences. For individuals experiencing voice disorders, a combination of medical and behavioral treatments are often helpful. For stuttering and other fluency disorders, treatment approaches usually help individuals develop techniques to both reduce the severity of stuttering and allow the individual to produce more fluent speech. For all of these therapies, individuals are taught to cope more effectively with their speech in progressively difficult situations, starting with speaking alone to the pathologist and ending with speaking to a group of people. In treating children with developmental language disorders, treatment often focusses on **modeling** and stimulation of correct productions of language. This type of approach may also be useful for adults with language disorders, secondary to a **stroke** or degenerative neurological disorder. For people with severe communication disorders, those due to either a speech or language problem, speech pathologists can assist with alternate means of communication, such as manual signing and computer-synthesized speech. Finally, speech-language pathologists have become increasingly involved with the assessment and treatment of individuals with swallowing disorders, or dysphagia.

The majority of speech-language pathologists work in public schools. They are also found at both residential health care facilities and outpatient clinics that specialize in communication disorders. Finally, speech-language pathologists are often employed at hospitals and universities. Professional training programs in speech-language pathology are offered at both the undergraduate and graduate levels. Undergraduate training may include classes in

biology, anatomy, psychology, linguistics, education, and special education. Graduate training, at both the masters and doctoral level, provides much deeper opportunities to study communication disorders and their treatment. To receive the Certificate of Clinical Competence (CCC) in speech-language pathology, individuals must hold a master's degree in communications sciences and disorders from a program accredited by the ASHA and complete their Clinical Fellowship Year (CFY).

Resources

ORGANIZATIONS

American Academy of Private Practice in Speech-Language Pathology and Audiology. 7349 Topanga Canyon Boulevard, Canoga Park, CA 91303.

American Speech-Language-Hearing Association. 10801 Rockville Pike, Rockville, MD 20785.

National Black Association for Speech, Language and Hearing. 3542 Gentry Ridge Court, Silver Spring, MD 20904.

Rodney Gabel, Ph.D.

Split personality disorder *see* **Dissociative identity disorder**

St. John's wort

Definition

St. John's wort is a perennial, yellow-flowering plant that grows in the wild throughout Europe and is now found also in North America. The plant tends to be in blossom in the month of June, around the day considered to be the birthday of John the Baptist; hence its popular name. The plant's Latin name is *Hypericum perforatum.*

St. John's wort has been used as a popular herbal folk remedy for centuries. More recently, practitioners of conventional Western medicine have been exploring its utility for treating depression and anxiety.

Purpose

Writings since the Middle Ages have described using St. John's wort as treatment for inflammation, injuries, burns, muscle pain, anxiety, high blood pressure, stomach problems, fluid retention, **insomnia**, hemorrhoids, cancer, and depression. Research conducted over the 1990s in Europe studied the efficacy of St. John's wort for the treatment of depression and anxiety. Research protocols have been developed in the United States to study the same issues, to determine appropriate dosages, to develop standard formulations, and to define whether it can be used for all forms of depression or only for more mild forms of the condition.

KEY TERMS

Immunosuppressant—Medications that suppress or lower the body's immune system, primarily used to help the body accept a transplanted organ.

Monoamine oxidase inhibitors—A group of antidepressant drugs that decrease the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

Reserpine—Medication to treat high blood pressure. Brand names include Serpalan, Novoreserpine, and Reserfia.

Theophylline—A medication used to treat asthma. Sold under many brand names, including Aerolate Sr, Respbid, and Theolair.

Warfarin—A medication that helps to prevent the formation of clots in the blood vessels. Sold as Coumadin in the U.S.

Description

Research has yet to completely explain how St. John's wort affects the **brain** in depression. It is, however, thought to change the balance of chemicals in the brain in much the same way as selective serotonin reuptake inhibitors (SSRIs) such as **fluoxetine** (Prozac), and monoamine oxidase inhibitors (MAOIs). The active ingredients are thought to be compounds called hypericin and pseudohypericin, although researchers are attempting to identify other chemicals that may be involved in the herb's effectiveness.

The leaves and flowers of St. John's wort are both used. St. John's wort is available as pills, capsules, extracts, dried herbs for tea, and oil infusions for skin applications.

Recommended dosage

Because dosages of herbal preparations are not always standardized, it is important to discuss with a knowledgeable practitioner the most reliable form of St. John's wort. Recommendations call for 300–500 mg (of a standardized 0.3% hypericin extract) three times daily. It can take four to six weeks to notice the antidepressant effects of this preparation.

Alternatively, one to two teaspoons of dried St. John's wort can be put into a cup of boiling water and

St. John's wort flowers. *(Photo Researchers, Inc. Reproduced by permission.) See color insert for color version of photo.*

steeped for 10 minutes to make tea. The recommended dosage of tea is one to two cups daily. Again, four to six weeks may be necessary in order to notice improvement in symptoms of depression.

Precautions

The following precautions should be considered and discussed with a knowledgeable practitioner before St. John's wort is taken:

- Some people may become more sensitive to the sun.
- Patients taking MAOIs must carefully avoid taking St. John's wort due to serious adverse effects of combining the two.
- Because the effects of St. John's wort are still being studied, pregnant and breast-feeding women should avoid its use.
- Depression can be a serious, even life-threatening, condition; therefore, it is imperative that depressed patients using St. John's wort are carefully monitored.

Side effects

People taking St. John's wort may develop one or all of the following side effects:

- skin rash due to sun sensitivity—the most common side effect
- headache, dizziness, dry mouth, constipation
- abdominal pain, confusion, sleep problems, and high blood pressure are less frequently experienced

Interactions

Again, a knowledgeable professional should be consulted before St. John's wort is taken to determine the appropriateness of its use and avoid serious interactions. Interactions include:

- Possible decrease in effectiveness of reserpine, warfarin, theophylline, immunosuppressant medications such as cyclosporine, and antiviral drugs such as indinavir.
- Dangerous interactions when used with other antidepressant medicines (especially MAOIs), digoxin, and loperamide.
- Interactions with oral birth control pills. St. John's wort may interfere with the effectiveness of birth control pills, increasing the risk of pregnancy; an alternative form of birth control should be considered while taking St. John's wort. In addition, women taking both birth control pills and St. John's wort may notice bleeding between menstrual periods.

See also Depression and depressive disorders

Resources

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Rosalyn Carson-DeWitt, M.D.

Stanford-Binet Intelligence Scale

Definition

The Stanford-Binet Intelligence Scale: Fourth Edition (SB: FE) is a standardized test that measures intelligence and cognitive abilities in children and adults, from age two through mature adulthood.

Purpose

The Stanford-Binet Intelligence Scale was originally developed to help place children in appropriate educational settings. It can help determine the level of intellectual and cognitive functioning in preschoolers, children, adolescents and adults, and assist in the **diagnosis** of a learning disability, developmental delay, **mental retardation**, or giftedness. It is used to provide educational planning and placement, neuropsychological assessment, and research. The Stanford-Binet Intelligence Scale is generally administered in a school or clinical setting.

Precautions

The Stanford-Binet Intelligence Scale is considered to be one of the best and most widely used **intelligence tests** available. It is especially useful in providing intellectual assessment in young children, adolescents, and young adults. The test has been criticized for not being comparable for all age ranges. This is because different age ranges are administered different subtests. Additionally, for very young preschoolers, it is not uncommon to receive a score of zero due to test difficulty or the child's unwillingness to cooperate. Consequently, it is difficult to discriminate abilities in this age group among the lower scorers.

Administration and interpretation of results of the Stanford-Binet Intelligence Scale requires a competent examiner who is trained in psychology and individual intellectual assessment, preferably a **psychologist**.

Description

The Stanford-Binet Intelligence Scale has a rich history. It is a descendant of the Binet-Simon scale which was developed in 1905 and became the first intelligence test. The Stanford-Binet Intelligence Scale was developed in 1916 and was revised in 1937, 1960, and 1986. The present edition was published in 1986. The Stanford Binet Intelligence Scale is currently being revised and the Fifth Edition is expected to be available in the spring of 2003.

Administration of the Stanford-Binet Intelligence Scale typically takes between 45 to 90 minutes, but can take as long as two hours, 30 minutes. The older the child and the more subtests administered, the longer the test generally takes to complete. The Stanford-Binet Intelligence Scale is comprised of four cognitive area scores which together determine the composite score and factor scores. These area scores include: Verbal Reasoning, Abstract/Visual Reasoning, Quantitative Reasoning, and Short-Term Memory. The composite score is considered to be what the authors call the best estimate of "g" or "general reasoning ability" and is the sum of all of subtest scores. General reasoning ability or "g" is considered to represent a person's ability to solve novel problems. The composite score is a global estimate of a person's intellectual functioning.

Abstract and visual reasoning are analyzed in Stanford-Binet intelligence tests. This blindfolded subject is matching shapes by touch. *(Richard Nowitz. Photo Researchers, Inc. Reproduced by permission.)*

The test consists of 15 subtests, which are grouped into the four area scores. Not all subtests are administered to each age group; but six subtests are administered to all age levels. These subtests are: Vocabulary, Comprehension, Pattern Analysis, Quantitative, Bead Memory, and Memory for Sentences. The number of tests administered and general test difficulty is adjusted based on the test taker's age and performance on the subtest that measures word knowledge. The subtest measuring word knowledge is given to all test takers and is the first subtest administered.

The following is a review of the specific cognitive abilities that the four area scores measure. The Verbal Reasoning area score measures verbal knowledge and understanding obtained from the school and home learning environment and reflects the ability to apply verbal skills to new situations. Examples of subtests comprising this factor measure skills which include: word knowledge, social judgment and awareness, ability to isolate the inappropriate feature in visual material and social intelligence, and the ability to differentiate essential from non-essential detail.

The Abstract/Visual Reasoning area score examines the ability to interpret and perform mathematic operations, the ability to visualize patterns, visual/motor skills, and problem-solving skills through the use of reasoning.

An example of a subtest which determines the Abstract/Visual Reasoning score is a timed test that involves tasks such as completing a basic puzzle and replicating black and white cube designs.

The Quantitative Reasoning area score measures: numerical reasoning, concentration, and knowledge and application of numerical concepts. The Quantitative Reasoning area is combined with the Abstract/Visual Reasoning area score to create an Abstract/Visual Reasoning Factor Score.

The Short-Term Memory score measures concentration skills, short-term memory, and sequencing skills. Subtests comprising this area score measure visual short-term memory and auditory short term memory involving both sentences and number sequences. In one subtest that measures visual short-term memory, the participant is presented with pictures of a bead design, and asked to replicate it from memory.

Results

The Stanford-Binet Intelligence Scale is a standardized test, which means that a large sample of children and adults were administered the exam as a means of developing test norms. The population in the sample was representative of the population of the United States based on age, gender, race or ethnic group, geographic region, community size, parental education, educational placement (normal versus special classes), etc. From this sample, norms were established. Norms are the performance of a comparison group of subjects—that nature of the group should be specified, and this usually constitutes a normal group so that the performance of the tested individual can be compared to this group and thus evaluated.

The numbers of correct responses on the given subtests are converted to a SAS score or Standard Age Score which is based on the chronological age of the test subject. This score is similar to an I.Q. score. Based on these norms, the Area Scores and Test Composite on the Stanford-Binet Intelligence Scale each have a mean or average score of 100 and a standard deviation of 16. For this test, as with most measures of intelligence, a score of 100 is in the normal or average range. The standard deviation indicates how above or below the norm a child's score is. For example, a score of 84 is one standard deviation below the norm score of 100. Based on the number of correct responses on a given subtest, an age-equivalent is available to help interpret the person's level of functioning.

Test scores provide an estimate of the level at which a child is functioning based on a combination of many different subtests or measures of skills. A trained psychologist is needed to evaluate and interpret the results, determine strengths and weaknesses, and make overall recommendations based on the findings and observed behavioral observations.

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The American Psychological Association. 750 First St., NE, Washington, DC 20002-4242. (202) 336-5500 <www.apa.org>.

The National Association of School Psychologists. 4340 East West Highway, Suite 402, Bethesda, MD 20814. (301) 657-0270. <www.nasponline.com>.

Jenifer P. Marom, Ph.D.

Stelazine *see* **Trifluoperazine**

Stereotypic movement disorder

Definition

Stereotypic movement disorder is a disorder characterized by repeated, rhythmic, purposeless movements or activities such as head banging, nail biting, or body rocking. These movements either cause self-injury or severely interfere with normal activities. Until 1994, the American Psychiatric Association referred to stereotypic movement disorder as stereotypy/habit disorder.

Description

Stereotypic movements were first described as a psychiatric symptom in the early 1900s. Since then, they have been recognized as a symptom of both psychotic and neurological disorders. They may also arise from unexplained causes. These movements may include:

- head banging
- nail biting

- playing with hair (but not hair pulling, which is considered the separate disorder of **trichotillomania**)
- thumb sucking
- hand flapping
- nose picking
- whirling
- body rocking
- picking at the body
- self-biting
- object biting
- self-hitting
- compulsive scratching
- eye gouging
- teeth grinding (bruxism)
- breath holding
- stereotyped sound production

The precise definition of stereotypic movement disorder has changed over the past 20 years. Today, it limits the disorder to repetitive movements that cause physical harm or severely interfere with normal activities. These movements cannot be better described by another psychiatric condition such as anxiety disorder, a general medical condition such as Huntington's disease, or as the side effect of a medication or illicit drug (for example, cocaine use).

Stereotypic movements occur in people of any age, including the very young, but they are most prevalent in adolescence. People may exhibit only one particular stereotyped movement or several. The movements may be slow and gentle, fast and frenetic, or varied in intensity. They seem to increase with boredom, tension, or frustration, and it appears that the movements are self-stimulatory and sometimes pleasurable. The root causes are unknown.

Stereotypic movements are common in infants and toddlers. Some estimates suggest that 15–20 percent of children under age three exhibit some kind of rhythmic, repetitive movements. Certainly thumb sucking and body rocking are common self-comforting mechanisms in the very young. This type of repeated movement is temporary, and usually ends by age three or four. It is not the same as stereotypic movement disorder.

Causes and symptoms

Causes

Stereotypic movements can be caused by:

- sensory deprivation (blindness or deafness)
- drug use (cocaine, amphetamines)
- brain disease (**seizures**, infection)
- major psychiatric disorders (anxiety disorder, **obsessive-compulsive disorder**, **autism**)
- **mental retardation**

It has also been suggested that inadequate caregiving may cause the disorder. Although many situations can give rise to stereotypic movements, the root cause of stereotypic movement disorder is unknown. Different theories propose that the causes are behavioral, neurological, and/or genetic. Although there are many theories to account for this disorder, no hard evidence clearly supports one line of reasoning or specific cause.

Symptoms

Symptoms of stereotypic movement disorder include all the activities listed above. It should be noted that many of these activities are normal in infants. They usually begin between five and 11 months, and disappear on their own by age three. In fact, about 55% of infants grind their teeth. These passing phases of repetitive movement in infants are not the same as stereotypic movement disorder. They do not cause harm, and often serve the purpose of self-comforting or helping the child learn a new motor skill.

People with stereotypic movement disorder often hurt themselves. They may pick their nail cuticles or skin until they bleed. They may repeatedly gouge their eyes, bite or hit themselves causing bleeding, bruising, and sometimes, as in the case of eye gouging or head banging, even more severe damage. Some people develop behaviors such as keeping their hands in their pockets, to prevent these movements. In other cases those who hurt themselves appear to welcome, rather than fight, physical restraints that keep them safe. However when these restraints are removed, they return to their harmful behaviors.

Demographics

Stereotypic movement disorder is most strongly associated with severe or profound mental retardation, especially among people who are institutionalized and perhaps deprived of adequate sensory stimulation. It is estimated that 2–3% of people with mental retardation living in the community have stereotypic movement disorder. About 25% of all people with mental retardation who are institutionalized have the disorder. Among those with severe or profound retardation, the rate is about 60%, with 15% showing behavior that causes self-injury.

Stereotypic movements are common among children with **pervasive developmental disorders** such as autism, childhood degenerative disorder, and **Asperger's disorder**. These movements can also be seen in people with Tourette's disorder or with tics. Head banging is estimated to affect about 5% of children, with boys outnumbering girls three to one, although other stereotypic behaviors appear to be distributed equally between males and females. Despite its association with psychiatric disorders, there are some people with normal intelligence and adequate caregiving who still develop stereotypic movement disorder.

Diagnosis

Stereotypic movements are diagnosed by the presence of the activities mentioned above. Young children rarely try to hide these movements, although older children may, and the first sign of them may be the physical harm they cause (bleeding skin, chewed nails). Often parents mention these repetitive movements when the physician takes a history of the child.

The difficulty in diagnosing stereotypic movement disorder comes from distinguishing it from other disorders where rhythmic, repetitive movements occur. To be diagnosed with stereotypic movement disorder, the following conditions must be met:

- The patient must show repeated, purposeless motor behavior.
- The patient must experience physical harm from this behavior or it must seriously interfere with activities.
- If the patient is mentally retarded, the behavior must be serious enough to need treatment.
- The behavior must not be a symptom of another psychiatric disorder.
- The behavior must not be a side effect of medicinal or illicit substance use.
- The behavior must not be caused by a diagnosed medical condition.
- The behavior must last at least four weeks. The disorder may be classified as either with self-injurious behavior or without self-harm.

This definition of stereotypic movement disorder rules out many people who show repetitive movement because of autism or other pervasive developmental disorders. It also rules out those with obsessive-compulsive disorder, where movements are apt to be ritualistic and follow rigid rules or patterns. In addition, specific disorders such as trichotillomania (hair pulling) do not fall under the **diagnosis** of stereotypic movement disorder, nor do developmentally appropriate self-stimulatory behavior among young children, such as thumb sucking, rocking or transient pediatric head banging.

Treatments

There are few successful treatments for stereotypic movement disorder. When the patient harms himself, physical restraints may be required. In less severe situations, behavioral modifications using both rewards and punishments may help decrease the intensity of the behavior. Drugs that have been used with some success to treat stereotypic movement disorder include **clomipramine** (Anafranil), **desipramine** (Norpramin), **haloperidol** (Haldol) and **chlorpromazine** (Thorazine).

Prognosis

Stereotypic movements peak in adolescence, then decline, and sometimes disappear. Although **behavior modification** may reduce the intensity of the stereotypic movements, rarely does it completely eliminate them. **Stress** and physical pain may bring on these movements, (which may come and go for years), especially among those patients with severe mental retardation.

Prevention

Stereotypic movement disorder cannot be prevented. Interventions should be done to prevent self-injury.

Resources

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Tish Davidson, A.M.

Stigma

Definitions

The 1999 report on mental health by the Surgeon General of the United States was regarded as a landmark document in the United Kingdom, as well as the United States. This was because of its straightforward identifica-

tion of the stigma associated with mental illness as the chief obstacle to effective treatment of persons with mental disorders. *Stigma* (plural, stigmata) is a Greek word that in its origins referred to a kind of tattoo mark that was cut or burned into the skin of criminals, slaves, or traitors in order to visibly identify them as blemished or morally polluted persons. These individuals were to be avoided or shunned, particularly in public places. The word was later applied to other personal attributes that are considered shameful or discrediting.

Social psychologists have distinguished three large classes, or categories, of stigma:

- Physical deformities. These include extremes of height and weight and such conditions as albinism and facial disfigurements or missing limbs. In the developed countries, this category also includes such signs of aging as gray hair, wrinkles, and stooped posture.
- Weaknesses or defects of individual character. This category includes biographical data that are held to indicate personal moral defect, such as a criminal record, **addiction**, divorce, treatment for mental illness, unemployment, **suicide** attempts, etc.
- Tribal stigma. This type of stigma refers to a person's membership in a race, ethnic group, religion, or (for women) gender that is thought to disqualify all members of the group.

The nature of stigma

Origins

One explanation for the origin of stigmata is that its roots in the human being's concern for group survival at earlier times in their evolutionary journey. According to this theory, stigmatizing people who were perceived as unable to contribute to the group's survival, or who were seen as threats to its well-being, were stigmatized in order to justify being forced out or being isolated.

The group survival theory is also thought to explain why certain human attributes seem to be universally regarded as stigmata, while others are specific to certain cultures or periods of history. Mental illness appears to be a characteristic that has nearly always led to the stigmatization and exclusion of its victims. The primary influences on Western culture, the classical philosophical tradition of Greece and Rome, and the religious traditions of Judaism and Christianity indicate that mental illness was a feared affliction that carried a heavy stigma. The classical philosopher's definition of a human being as a "rational animal" excluded him or her who had lost the use of reason and was no longer regarded as fully human; most likely he or she was under a divine curse. This attitude was summarized in the well-known saying of Lucretius, "Whom the gods wish to destroy, they first make mad."

KEY TERMS

Stigma—A mark or characteristic trait of a disease or defect; by extension, a cause for reproach or a stain on one's reputation.

In the Bible, both the Old and the New Testaments reflect the same fear of mental illness. In 1 Samuel 21, there is an account of David's pretending to be insane in order to get away from the king of a neighboring territory. "He changed his behavior before [the king's servants]; he pretended to be mad in their presence. He scratched marks on the doors of the gate, and let his spittle run down his beard." The king, who was taken in by an act that certainly fits the ***Diagnostic and Statistical Manual of Mental Disorders*** criteria for **malingering**, quickly sent David on his way. In the New Testament, one of Jesus' most famous miracles of healing (Mark 5:1-20) is the restoration of sanity to a man so stigmatized by his village that he was hunkered down in the graveyard (itself a stigmatized place) outside the village when Jesus met him. Mark's account also notes that the villagers had tried at different times to chain or handcuff the man because they were so afraid of him. One important positive contribution of Biblical heritage, however, is a sense of religious obligation toward the mentally ill. Among Christians, the New Testament's account of Jesus' openness to all kinds of stigmatized people—tax collectors, prostitutes, and physically deformed people, as well as the mentally ill—became the basis for the establishment of the first shelters and hospitals for the mentally ill.

Contemporary contexts

The core feature of stigma in the modern world is defined by social psychologists as the possession of an attribute "that conveys a devalued social identity within a particular context." Context is important in assessing the nature and severity of **stress** that a person suffers with regard to stigma. Certain attributes, such as race or sex, affect an individual's interactions with other people in so many different situations that they have been termed "master status" attributes. These have become the classic identifying characteristic of the person who possesses them. Dorothy Sayers' essay, "Are Women Human?" is not only a witty satire on the way men used to describe a woman's job or occupation (with constant reference to feminine qualities), but a keen social analysis of the problems created by master status attributes for persons who are stigmatized.

Other forms of devalued social identity are relative to specific cultures or subcultures. In one social context, a person who is stigmatized for an attribute devalued by a particular group may find acceptance in another group that values the particular attribute. A common example is that of an artistically or athletically talented child who grows up in a family that values only intellectual accomplishment. When the youngster is old enough to leave the family of origin, he or she can find a school or program for other students who share the same interest. A less marked contrast, but one that is relevant to the treatment of mental illness, is the cultural differences with regard to the degree of response to certain symptoms of mental illness. A study conducted in the early 21st century assessed the reaction of family members to elderly people who were diagnosed with **Alzheimer's disease** (AD). Findings pointed to considerable variation across racial and ethnic groups. Asian Americans were most affected by feelings of shame and social stigma relative to the memory loss of a family member, while African Americans were the least affected.

One additional complicating feature of stigma is the issue of overlapping stigmata. Many people belong to several stigmatized groups or categories, and it is not always easy to determine which category triggers the unkind or discriminatory treatment encountered. For example, one study of the inadequate medical treatment that is offered to most HIV-positive Native Americans noted that the stigma of Acquired Immune Deficiency Syndrome (AIDS) provides a strong motivation for not seeking treatment. The study protocol, however, did not seek to investigate whether young Native American men are afraid of being stigmatized for their sexual orientation, their race, their low socioeconomic status, or all three.

Stigma and mental illness

Stigma and specific disorders

The stigma that is still attached to mental illness in the developed countries does not represent a simple or straightforward problem. Public health experts who have studied the stigmatization of mental illness in recent years have noted that the general public's perception of mental illness varies, depending on the nature of the disorder. While in general the stigma of mental illness in contemporary society is primarily associated with the second of the three categories of stigma listed above,—supposed character failings—it also spills over into the first category. Mental disorders that affect a person's physical appearance—particularly weight gain—are more heavily stigmatized than those that do not.

The stigma related to certain types of mental disorders has declined since the 1950s, most notably in regard to depression and the anxiety disorders. It is thought that the reason for this change is that people are more likely nowadays to attribute these disorders to stress, with which most people can identify. On the other hand, the stigma associated with psychotic disorders appears to be worse than it was in the 1950s. Changes in public attitude are also reflected in age-group patterns in seeking or dropping out of treatment for mental disorders. One study demonstrated that older adults being treated for depression were more likely than younger adults to drop out of treatment because they felt stigmatized. The difference in behavior is related to public attitudes toward mental illness that were widespread when the older adults were adolescents.

In 2002, the types of mental disorders that carry the heaviest stigma fall into the following categories:

- Disorders associated in the popular mind with violence and/or illegal activity. These include **schizophrenia**, mental problems associated with HIV infection, and substance abuse disorders.
- Disorders in which the patient's behavior in public may embarrass family members. These include **dementia** in the elderly, **borderline personality disorder** in adults, and the autistic spectrum disorders in children.
- Disorders treated with medications that cause weight gain or other visible side effects.

The role of the media

The role of the media in perpetuating the stigmatization of mental illness has received increasing attention from public health researchers, particularly in Great Britain. In 1998, the Royal College of Psychiatrists launched a five-year campaign intended to educate the general public about the nature and treatment of mental illness. Surveys conducted among present and former mental patients found that they considered media coverage of their disorders to be strongly biased toward the sensational and the negative. One-third of patients said that they felt more depressed or anxious as a result of news stories about the mentally ill, and 22% felt more withdrawn. The main complaint from mental health professionals, as well as patients, is that the media presented mentally ill people as "dangerous time bombs waiting to explode" when in fact 95% of murders in the United Kingdom are committed by people with no mental illness. The proportion of homicides committed by the mentally ill has decreased by 3% per year since 1957, but this statistic goes unreported. Much the same story of unfair stigmatization in the media could be told in the United States, as the Surgeon General's report indicates.

Physicians' attitudes toward mental illness

Physicians' attitudes toward the mentally ill are also increasingly recognized as part of the problem of stigmatization. The patronizing attitude of moral superiority toward the mentally ill in the early 1960s, specifically in mental hospitals, has not disappeared. This was reported by Erving Goffman in his classic study. A Canadian insurance executive told a conference of physicians in May 2000 that they should look in the mirror for a picture of the ongoing stigmatization of the mentally ill. The executive was quoted as saying, "Stigma among physicians deters the detection of mental disorders, defers or pre-empts correct **diagnosis** and proper treatment and, by definition, prolongs suffering." An American physician who specializes in the treatment of substance addicts cites three reasons for the persistence of stigmatizing attitudes among his colleagues: their tendency to see substance abuse as a social issue, rather than a health issue; their lack of training in detecting substance abuse; and their mistaken belief that no effective treatments exist. A similar lack of information about effective treatments characterizes many psychiatrists' attitudes toward borderline personality disorder.

Stigma as cause of mental illness

It is significant that researchers in the field of social psychology have moved in recent years to analyzing stigma in terms of stress. Newer studies in this field now refer to membership in a stigmatized group as a stressor that increases a person's risk of developing a mental illness. The physiological and psychological effects of stress caused by racist behavior, for example, have been documented in African Americans. Similar studies of obese people have found that the stigmatization of **obesity** is the single most important factor in the psychological problems of these patients. To give still another example, the high rates of depression among postmenopausal women have been attributed to the fact that aging is a much heavier stigma for women than for men in contemporary society.

Stigma has a secondary effect on rates of mental illness in that members of stigmatized groups have less access to educational opportunities, well-paying jobs, and adequate health care. They are therefore exposed to more environmental stressors in addition to the stigma itself.

Stigma as effect

Stigma resulting from mental illness has been shown to increase the likelihood of a patient's relapse. Since a mental disorder is not as immediately apparent as race, sex, or physical handicaps, many people with mental disorders undergo considerable strain trying to conceal their condition from strangers or casual acquaintances. More seriously, the stigma causes problems in the job market, leading to stress that is related to lying to a potential employer and fears of being found out. Erving Goffman reported in the 1960s that a common way around the dilemma involved taking a job for about six months after discharge from a mental institution, then quitting that job and applying for another with a recommendation from the first employer that did not mention the history of mental illness.

The stigmatization of the patient with mental illness extends to family members, partly because they are often seen as the source of the patient's disorder. A recent editorial in the *Journal of the American Medical Association* tells the story of two sets of parents coping with the stress caused by other people's reactions to their children's mental illness, and the different responses they received when the children's disorders were thought to be a physical problem. The writer also tells of the problems encountered by the parents of an autistic child. The writer stated that family excursions were difficult, and continued, "My friend's wife was reprimanded by strangers for not being able to control her son. The boy was stared at and ridiculed. The inventive parent, fed up with the situation, bought a wheelchair to take the child out. The family was now asked about their child's disability. They were praised for their tolerance of his physical hardship and for their courage; the son was commended for his bravery. Same parents, same child, different view."

The results of stigma

The stigmatization of mental disorders has a number of consequences for the larger society. Patients' refusal to seek treatment, noncompliance with treatment, and inability to find work has a high price tag. Disability related to mental illness accounts for fully 15% of the economic burden caused by *all* diseases in developed countries.

Seeking treatment

Stigmatization of mental illness is an important factor in preventing persons with mental disorders from asking for help. This factor affects even mental health services on university campuses; interviews with Harvard students following a 1995 murder in which a depressed student killed a classmate, found that students hesitated to consult mental health professionals because many of their concerns were treated as disciplinary infractions, rather than illnesses. The tendency to stigmatize mental disorders as character faults is as prevalent among educators as among medical professionals. In addition, studies of large corporations indicate that employees frequently hesitate to seek treatment for depression and

other stress-related disorders for fear of receiving negative evaluations of job performance and possible termination. These fears are especially acute during economic downturns and periods of corporate downsizing.

Compliance with treatment

Another connection between mental disorders and stigma is the low rates of treatment **compliance** among patients. To a large extent, patient compliance is a direct reflection of the quality of the doctor-patient relationship. One British study found that patients with mental disorders were likely to prefer the form of treatment recommended by psychiatrists with whom they had good relationships, even if the treatment itself was painful or difficult. Some patients preferred **electroconvulsive therapy** (ECT) to tranquilizers for depression because they had built up trusting relationships with the doctors who used ECT, and perceived the doctors who recommended medications as bullying and condescending. Other reasons for low compliance with treatment regimens are related to stigmatized side effects. Many patients, particularly women, discontinue medications that cause weight gain because of the social stigma attached to obesity in females.

Social and economic consequences

As already mentioned, persons with a history of treatment for mental disorders frequently encounter prejudice in the job market and the likelihood of long periods of unemployment; this can result in lower socioeconomic status, as well as loss of self-esteem. These problems are not limited to North America. A recent study of mental health patients in Norway, which is generally considered a progressive nation, found that the patients had difficulty finding housing as well as jobs, and were frequently harassed on the street as well as being socially isolated. In 1990, the Congress of the U.S. included mental disorders (with a few exceptions for disorders related to substance abuse and compulsive sexual behaviors) in the anti-discriminatory provisions of the Americans with Disabilities Act (ADA). As of 2002, mental disorders constitute the third-largest category of discrimination claims against employers.

Stigmatization of mental disorders also affects funding for research into the causes and treatment of mental disorders. Records of recent Congressional debates indicate that money for mental health research is still grudgingly apportioned as of 2002.

Future prospects

The stigma of mental illness will not disappear overnight. Slow changes in attitudes toward other social issues have occurred in the past three decades, giving hope to the lessening of stigma toward people with mental illness. However, limitations on indefinite economic expansion are an reason for concern. As the economic "pie" has to be divided among a larger number of groups, causing competition for public funding, persons with mental disorders will need skilled and committed advocates if their many serious needs are to receive adequate attention and help.

See also Stress

Resources

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Stress

Definitions

Stress is a term that refers to the sum of the physical, mental, and emotional strains or tensions on a person. Feelings of stress in humans result from interactions between persons and their environment that are perceived as straining or exceeding their adaptive capacities and threatening their well-being. The element of perception indicates that human stress responses reflect differences in personality as well as differences in physical strength or health.

A stressor is defined as a stimulus or event that provokes a stress response in an organism. Stressors can be categorized as acute or chronic, and as external or internal to the organism. The ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*) defines a psychosocial stressor as "any life event or life change that may be associated temporally (and perhaps causally) with the onset, occurrence, or exacerbation [worsening] of a mental disorder."

Stress affects the lives of most adults in developed countries in many ways. It is a major factor in rising health care costs; one public health expert maintains that 90% of all diseases and disorders in the United States are stress-related. Stress plays a part in many social problems such as child and elder **abuse**, workplace violence, juvenile crime, **suicide**, substance **addiction**, "road rage," and the general decline of courtesy and good manners. Stress also affects the productivity of businesses and industries. One nationwide survey found that 53% of American workers name their job as the single greatest source of stress in their lives. Furthermore, the overall cost of medical care, time lost from work, and workplace accidents in the United States comes to over $150 million per year.

The neurobiology of stress

One way to understand stress as a contemporary health problem is to look at the human stress response as a biologically conditioned set of reactions that was a necessary adaptation at earlier points in human evolution, but is less adaptive under the circumstances of modern life. Hans Selye (1907-1982), a Canadian researcher, was a pioneer in studying stress. Selye defined stress, in essence, as the rate of wear and tear on the body. He observed that an increasing number of people, particularly in the developed countries, die of so-called diseases of civilization, or degenerative diseases, which are primarily caused by stress. Selye also observed that stress in humans depends partly on people's evaluation of a situation and their emotional reaction to it; thus, an experience that one person finds stimulating and exciting—for example, bungee jumping—would produce harmful stress in another.

The stress response

In humans, the biochemical response to acute stress is known as the "fight-or-flight" reaction. It begins with

KEY TERMS

Adjustment disorder—A disorder defined by the development of significant emotional or behavioral symptoms in response to a stressful event or series of events. Symptoms may include depressed mood, anxiety, and impairment of social and occupational functioning.

Adrenaline—Another name for epinephrine, the hormone released by the adrenal glands in response to stress. It is the principal blood-pressure raising hormone and a bronchial and intestinal smooth muscles relaxant.

Allostasis—The process of an organism's adaptation to acute stress.

Amygdala—An almond-shaped brain structure in the limbic system that is activated in acute stress situations to trigger the emotion of fear.

Burnout—An emotional condition that interferes with job performance, marked by fatigue, loss of interest, or frustration; usually regarded as the result of prolonged stress.

Catecholamine—A group of neurotransmitters synthesized from the amino acid tyrosine and released by the hypothalamic-pituitary-adrenal system in the brain in response to acute stress. The catecholamines include dopamine, serotonin, norepinephrine, and epinephrine.

Coping—In psychology, a term that refers to a person's patterns of response to stress.

Cortisol—A steroid hormone released by the cortex (outer portion) of the adrenal gland when a person is under stress.

Dissociation—A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

Eustress—A term that is sometimes used to refer to positive stress.

Flashback—The re-emergence of a traumatic memory as a vivid recollection of sounds, images, and sensations associated with the trauma. The person having the flashback typically feels as if he or she is reliving the event.

Hippocampus—A part of the brain that is involved in memory formation and learning. The hippocampus is shaped like a curved ridge and belongs to an organ system called the limbic system.

Homeostasis—The tendency of the physiological system in humans and other mammals to maintain its internal stability by means of a coordinated response to any stimulus that disturbs its normal condition.

Limbic system—A group of structures in the brain that includes the amygdala, hippocampus, olfactory bulbs, and hypothalamus. The limbic system is associated with homeostasis and the regulation and arousal of emotions.

Relaxation response—The body's inactivation of stress responses and return of stress hormone levels to normal after a threat has passed.

Stress management—A set of techniques and programs intended to help people deal more effectively with stress in their lives by analyzing the specific stressors and taking positive actions to minimize their effects. Most stress management programs deal with job stress and workplace issues.

Stressor—A stimulus or event that provokes a stress response in an organism. Stressors can be categorized as acute or chronic, and as external or internal to the organism.

the activation of a section of the **brain** called the hypothalamic-pituitary-adrenal system, or HPA. This system first activates the release of steroid hormones, which are also known as glucocorticoids. These hormones include cortisol, the primary stress hormone in humans.

The HPA system then releases a set of **neurotransmitters** known as catecholamines, which include dopamine, norepinephrine, and epinephrine (also known as adrenaline). Catecholamines have three important effects:

- They activate the amygdala, an almond-shaped structure in the limbic system that triggers an emotional response of fear.
- They signal the hippocampus, another part of the limbic system, to store the emotional experience in long-term memory.
- They suppress activity in parts of the brain associated with short-term memory, concentration, and rational thinking. This suppression allows a human to react

quickly to a stressful situation, but it also lowers ability to deal with complex social or intellectual tasks that may be part of the situation.

In reaction to stress, heart rate and blood pressure rise, and the person breathes more rapidly, which allows the lungs to take in more oxygen. Blood flow to the muscles, lungs, and brain may increase by 300–400%. The spleen releases more blood cells into the circulation, which increases the blood's ability to transport oxygen. The immune system redirects white blood cells to the skin, bone marrow, and lymph nodes; these are areas where injury or infection is most likely.

At the same time, nonessential body systems shut down. The skin becomes cool and sweaty as blood is drawn away from it toward the heart and muscles. The mouth becomes dry, and the digestive system slows down.

The relaxation response

After the crisis passes, the levels of stress hormones drop and the body's various organ systems return to normal. This return is called the relaxation response. Some people are more vulnerable to stress than others because their hormone levels do not return to normal after a stressful event. An absent or incomplete relaxation response is most likely to occur in professional athletes and in people with a history of depression.

Physical effects of chronic stress

In chronic stress, the organ systems of the body do not have the opportunity to return fully to normal levels. Different organs become under- or overactivated on a long-term basis. In time, these abnormal levels of activity can damage an organ or organ system.

Cardiovascular system

Stress has a number of negative effects on the heart and circulatory system. Sudden stress increases heart rate, but also causes the arteries to narrow, which may block the flow of blood to the heart. The emotional effects of stress can alter the rhythm of the heart. In addition, stress causes the release of extra clotting factors into the blood, which increases the risk of a clot forming and blocking an artery. Stress also triggers the release of fat into the bloodstream, which temporarily raises blood cholesterol levels. Lastly, it is thought that people who regularly have sudden increases in blood pressure due to mental stress may over time suffer injuries to the inner lining of their blood vessels.

Gastrointestinal system

The effects of chronic stress on the gastrointestinal system include diarrhea, constipation, bloating, and irritable bowel syndrome. Although stress is not the direct cause of either peptic ulcers or inflammatory bowel disease, it may predispose people to develop ulcers and worsen flareups of inflammatory bowel disease.

Stress is the cause of abnormal weight loss in some people and of weight gain in others, largely from stress-related eating. It is thought that stress related to the physical and emotional changes of puberty is a major factor in the development of eating disorders.

Reproductive system

Stress affects sexual desire in both men and women and can cause impotence in men. It appears to worsen the symptoms of premenstrual syndrome (PMS) in women. Stress affects fertility, in that high levels of cortisol in the blood can affect the hypothalamus, which produces hormones related to reproduction. Very high levels of cortisol can cause amenorrhea, or cessation of menstrual periods.

Stress during pregnancy is associated with a 50% higher risk of miscarriage. High stress levels on the mother during pregnancy are also related to higher rates of premature births and babies of lower than average birth weight; both are risk factors for infant mortality.

Musculoskeletal system

Stress intensifies the chronic pain of arthritis and other joint disorders. It also produces tension-type headaches, caused by the tightening of the muscles in the neck and scalp. Research indicates that people who have frequent tension headaches have a biological predisposition for converting emotional stress into muscle contraction.

Brain

The physical effects of stress hormones on the brain include interference with memory and learning. Acute stress interferes with short-term memory, although this effect goes away after the stress is resolved. People who are under severe stress become unable to concentrate; they may become physically inefficient, clumsy, and accident-prone. In children, however, the brain's biochemical responses to stress clearly hamper the ability to learn.

Chronic stress appears to be a more important factor than aging in the loss of memory in older adults. Older people with low levels of stress hormones perform as well as younger people in tests of cognitive (knowledge-related) skills, but those with high levels of stress hormones test between 20% and 50% lower than the younger test subjects.

Immune system

Chronic stress affects the human immune system and increases a person's risk of getting an infectious illness. Several research studies have shown that people under chronic stress have lower than normal white blood cell counts and are more vulnerable to colds and influenza. Men with HIV infection and high stress levels progress more rapidly to AIDS than infected men with lower stress levels.

Stress and mental disorders

DSM-IV-TR specifies two major categories of mental disorders directly related to stress—the post-traumatic syndromes and adjustment disorders. Stress is, however, also closely associated with depression, and can worsen the symptoms of most other disorders.

Post-traumatic disorders

Post-traumatic stress disorder (PTSD) and **acute stress disorder** (ASD) are defined by their temporal connection to a traumatic event in the individual's life. The post-traumatic disorders are characterized by a cluster of anxiety and dissociative symptoms, and by their interference with the patient's normal level of functioning. **Magnetic resonance imaging** (MRI) studies have shown that the high levels of sustained stress in some PTSD patients cause demonstrable damage to the hippocampus. Excessive amounts of stress hormones in brain tissue cause the nerve cells, or neurons, in parts of the hippocampus to wither and eventually die. One group of Vietnam veterans with PTSD had lost as much as 8% of the tissue in the hippocampus.

Substance abuse disorders

Stress is related to substance abuse disorders in that chronic stress frequently leads people to self-medicate with drugs of abuse or alcohol. Substance abuse disorders are associated with a specific type of strategy for dealing with stress called emotion-focused coping. Emotion-focused coping strategies concentrate on regulating painful emotions related to stress, as distinct from problem-focused coping strategies, which involve efforts to change or eliminate the impact of a stressful event. Persons who handle stress from a problem-oriented perspective are less likely to turn to mood-altering substances when they are under stress.

Adjustment disorders

DSM-IV-TR defines adjustment disorders as psychological responses to stressors that are excessive given the nature of the stressor; or result in impairment of the person's academic, occupational, or social functioning. The most important difference between the post-traumatic disorders and adjustment disorders is that most people would not necessarily regard those stressors involved in the latter disorder as traumatic. **Adjustment disorder** appear to be most common following natural disasters, divorce, becoming a parent, and retirement from work.

Causes of stress

The causes of stress may include any event or situation that a person considers a threat to his or her resources or coping strategies. A certain amount of stress is a normal part of life; it represents a person's response to inevitable changes in his or her physical or social environment. Moreover, positive events can generate stress as well as negative events. Graduating from college, for example, is accompanied by stress related to employment or possible geographical relocation and the stress of saying good-bye to friends and family, as well as feelings of positive accomplishment. Some researchers refer to stress associated with positive events as eustress.

Acute stress is defined as a reaction to something perceived as an immediate threat. Acute stress reactions can occur to a falsely perceived danger as well as to a genuine threat; they can also occur in response to memories. For example, a war veteran who hears a car backfire may drop to the ground because the noise triggers vivid memories, called flashbacks, of combat experience. Common acute stressors include loud, sudden noises being in a crowded space such as an elevator, being cut off in heavy traffic; and dangerous weather. Chronic stress is a reaction to a situation that is stressful but ongoing, such as financial worries or caring for an elderly parent. Modern life is stressful because changes in various areas of life have increased the number of acute and chronic stressors in most people's lives at the same time that they have weakened certain buffers or protections against stress.

Social changes

Social changes that have increased the stress level of modern life include increased population mobility and the sprawling size of modern cities. It is not unusual for adults to live hundreds of miles away from parents and siblings; and it is hard to make and keep friendships when people move every few years. In most large cities, many people live in apartment buildings where they do not know their neighbors. Social isolation and loneliness can produce chronic stress. A study done in Norway between 1987 and 1993 found that social support networks made a significant difference in lowering the impact of both acute and chronic stress on mental health.

Social scientists have observed that the increased isolation of married couples from extended families and friendship networks increases strains on the marriage. The rising divorce rate in the United States has been attributed in part to the loss of social supports that once helped to keep married couples together. The experience of divorce then adds to the stress level on the former spouses and the children, if any. A long-term study at the University of Pittsburgh has found that divorce is associated with a higher rate of premature death in men.

Economic changes

The rapid pace of change in manufacturing and other businesses means that few people will work at the same job for their entire career. In addition, corporate mergers and downsizing have weakened job security, thus producing chronic anxiety about unemployment in the minds of many employees. Many people work two jobs in order to make ends meet; and even those who work only one job often have to commute by car or train to their workplace. In many large American cities, traffic jams, cost of gasoline, and other problems related to commuting are a major factor in job-related stress. Another stress factor is sleep deprivation. Many people get only six or less hours of sleep each night even though the National Sleep Foundation estimates that most adults need 8–8-1/2 hours per night for good health. **Fatigue** due to sleep deprivation causes additional stress.

Lastly, economic trends have produced a "winner-take-all" economy in which the gap between the well-off and the average family is constantly widening. Socioeconomic status (SES) affects health in a number of ways. Persons of higher SES can afford better health care, are less likely to suffer from exposure to environmental toxins, and generally lead healthier lifestyles. In addition, chronic stress associated with low SES appears to increase morbidity and mortality among persons in these income groups.

Technological changes

Technology has proved to be a source of stress as well as a solution to some kinds of stress. Machines that help workers to be more productive also make their jobs more complicated and raise the level of demands on them. An office clerk in 2002 can produce many more letters per day than one in 1952, but is often expected to produce more elaborate, professional-looking documents as well as a higher number of them.

One specific technological development that has been singled out as a major stressor in modern life is the evolution of news reporting. For most of human history, people had to wait several days or even weeks to hear about the outcome of an election, a battle, or some other important event. Moreover, they usually heard only the news that affected their region or their country. Today, however, news is reported as soon as it happens, it is broadcast 24 hours a day, and it covers events around the world. This "communications overload," as it has been termed, is a source of genuine stress to many people, particularly when the newscast emphasizes upsetting or frightening events. It is not surprising that a common recommendation for lowering one's stress level is to cut down on watching television news programs. A team of physicians conducted telephone interviews following the events of September 11, 2001, in order to assess stress reactions in the general American population. The team found that the single most important factor was not geographical location relative to the attacks or educational level, but the amount of time spent watching televised reports of the attacks. The interviewers discovered that 49% of the adults had watched at least eight hours of television on September 11, and also that "extensive television viewing was associated with a substantial stress reaction."

Environmental changes

One significant source of stress in modern life is the cumulative effect of various toxic waste products on the environment. Studies of the aftermath of such environmental disasters as Three Mile Island and Chernobyl found that not only evacuees and people living in the contaminated area had high levels of emotional distress, but also cleanup workers and people living in nearby noncontaminated areas. In the case of Chernobyl, Russian physicians have reported a psychoneurological syndrome with several unexplained symptoms, including fatigue, impaired memory, muscle or joint pain, and sleep disturbances. The syndrome appears to be due to chronic emotional stress rather than radiation exposure.

Changes in beliefs and attitudes

Changes in beliefs that influence stress levels include the contemporary emphasis on individualism and a corresponding change in attitudes toward trauma. A number of observers have remarked that Western culture has moved away from its traditional high valuation of the family and community toward an increased focus on the individual. Some have called this trend the "Me First!" society—it emphasizes personal rights and entitlements rather than duties and responsibilities to others. It has, in the view of some physicians, encouraged people to dwell on trauma and its effects on them as individuals rather than to live up to more traditional ideals of composure and resilience in the face of distress.

Risk factors

Research indicates that some categories of people have a higher risk of stress-related illnesses and disorders:

- Children have very little control over their environments. In addition, they are often unable to communicate their feelings accurately.
- In elderly adults, aging appears to affect the body's response to stress, so that the relaxation response following a stressful event is slower and less complete. In addition, the elderly are often affected by such major stressors as health problems, the death of a spouse or close friends, and financial worries.
- Caregivers of mentally or physically disabled family members.
- Women in general.
- People with less education.
- People who belong to racial or ethnic groups that suffer discrimination.
- People who live in cities.
- People who are anger-prone. Chronic anger is associated with narrowing of the arteries, a factor in heart disease.
- People who lack family or friends.
- People who are biologically predisposed to an inadequate relaxation response.

Coping with stress

Coping is defined as a person's patterns of response to stress. Many clinicians think that differences in attitudes toward and approaches to stressful events are the single most important factor in assessing a person's vulnerability to stress-related illnesses. A person's ability to cope with stress depends in part on his or her interpretation of the event. One person may regard a stressful event as a challenge that can be surmounted while another views it as a problem with no solution. The person's resources, previous physical and psychological health, and previous life experience affect interpretation of the event. Someone who has had good experiences of overcoming hardships is more likely to develop a positive interpretation of stressful events than someone who has been repeatedly beaten down by abuse and later traumas.

Coping styles

The ways in which people cope with stress can be categorized according to two different sets of distinctions. One is the distinction between emotion-focused and problem-focused styles of coping, which was described earlier in connection with substance abuse. Problem-focused coping is believed to lower the impact of stress on health; people who use problem-focused coping have fewer illnesses, are less likely to become emotionally exhausted, and report higher levels of satisfaction in their work and feelings of personal accomplishment. Emotion-focused coping, on the other hand, is associated with higher levels of interpersonal problems, depression, and social isolation. Although some studies reported that men are more likely to use problem-focused coping and women to use emotion-focused coping, other research done in the last decade has found no significant gender differences in coping styles.

The second set of categories distinguishes between control-related and escape-related coping styles. Control-related coping styles include direct action, behavior that can be done alone; help-seeking, behavior that involves social support; and positive thinking, a cognitive style that involves giving oneself pep talks. Escape-related coping styles include avoidance/resignation, as in distancing oneself from the stressful event, and alcohol use. There appears to be no relationship between gender and a preference for control-related or escape-related coping.

Stress management

Stress management refers to a set of programs or techniques intended to help people deal more effectively with stress. Many of these programs are oriented toward job- or workplace-related stress in that burnout is a frequent result of long-term occupational stress. Most stress management programs ask participants to analyze or identify the specific aspects of their job that they find stressful, and then plan a course of positive action to minimize the stress. In general, the severity of job-related stress appears to be related to two factors: the magnitude of the demands being made on the worker, and the degree of control that she or he has in dealing with the demands. The workers who are most vulnerable to stress-related heart disease are those who are subjected to high demands but have little control over the way they do their job. In many cases, stress management recommendations include giving an employee more decision-making power.

Treatments for stress

There are a number of allopathic and alternative/complementary treatments that are effective in relieving the symptoms of stress-related disorders:

- Medications may include drugs to control anxiety and depression as well as drugs that treat such physical symptoms of stress as indigestion or high blood pressure.
- Psychotherapy, including insight-oriented and cognitive/behavioral approaches, is effective in helping people understand how they learned to overreact to stressors, and in helping them reframe their perceptions and

interpretations of stressful events. Anger management techniques are recommended for people who have stress-related symptoms due to chronic anger.

- Relaxation techniques, **anxiety reduction techniques**, breathing exercises, **yoga**, and other physical exercise programs that improve the body's relaxation response.
- Therapeutic massage, hydrotherapy, and **bodywork** are forms of treatment that are particularly helpful for people who tend to carry stress in their muscles and joints.
- **Aromatherapy**, pet therapy, humor therapy, music therapy, and other approaches that emphasize sensory pleasure are suggested for severely stressed people who lose their capacity to enjoy life; sensory-based therapies can counteract this tendency.
- Naturopathic recommendations regarding diet, exercise, and adequate sleep, and the holistic approach of naturopathic medicine can help persons with stress-related disorders to recognize and activate the body's own capacities for self-healing.

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See also Creative therapies; Diets; Nutrition and mental health

Rebecca J. Frey, Ph.D.

Stroke

Definition

A stroke, also called a cerebral vascular accident (CVA), is the sudden death of cells in a specific area of the **brain** due to inadequate blood flow.

KEY TERMS

Aneurysm—A symptomless bulging of a weak arterial wall that can rupture, leading to stroke.

Angiography—A procedure in which a contrast medium is injected into the bloodstream (through an artery in the neck) and its progress through the brain is tracked. This illustrates where a blockage or hemorrhage has occurred.

Anticoagulant—A medication (such as warfarin, Coumadin, or Heparin) that decreases the blood's clotting ability preventing the formation of new clots. Although anticoagulants will not dissolve existing clots, they can stop them from getting larger. These drugs are commonly called blood thinners.

Atrial fibrillation—A disorder in which the upper chambers (atria) of the heart do not completely empty with each contraction (heartbeat). This can allow blood clots to form and is associated with a higher risk of stroke.

Electrocardiogram—(EKG) A test that measures the electrical activity of the heart as it beats. An abnormal EKG can indicate possible cardiac disease.

Electroencephalogram—(EEG) A test that measures the electrical activity of the brain by means of electrodes placed on the scalp or on or in the brain itself. It may be used to determine whether or not a stroke victim has had a seizure.

Hypertension—High blood pressure, often brought on by smoking, obesity, or other causes; one of the major causes of strokes.

Pressure ulcers—Also known as pressure sores or bed sores, these can develop in stroke patients who are unable to move. If not treated properly, they can become infected.

Tissue plasminogen activator (tPA)—A drug that is sometimes given to patients within three hours of a stroke to dissolve blood clots within the brain; also used to treat heart attack victims.

Ultrasound—A noninvasive test in which high-frequency sound waves are reflected off a patient's internal organs allowing them to be viewed. In stroke victims, a cardiac ultrasound, or echocardiogram, allows the beating heart to be examined.

Description

A stroke occurs when blood flow is interrupted to a part of the brain, either when an artery bursts or becomes closed when a blood clot lodges in it. Blood circulation to the area of the brain served by that artery stops at the point of disturbance, and the brain tissue beyond that is damaged or dies. (Brain cells need blood to supply oxygen and nutrients and to remove waste products.) Depending on the region of the brain affected, a stroke can cause paralysis, loss of vision, speech impairment, memory loss and reasoning ability, coma, or death. The effects of a stroke are determined by how much damage occurs, and which portion of the brain is affected.

About a third of all strokes are preceded by transient ischemic attacks (TIAs), or mini-strokes, that temporarily interrupt blood flow to the brain. While TIAs cause similar symptoms (such as sudden vision loss or temporary weakness in a limb), they abate much more quickly than full-fledged strokes, usually within a few hours—sometimes as quickly as a few minutes.

Stroke is a medical emergency requiring immediate treatment. Prompt treatment improves the chances of survival and increases the degree of recovery that may be expected. A person who may have suffered a stroke should be seen in a hospital emergency room without delay. Treatment to break up a blood clot, the major cause of stroke, must begin within three hours of the stroke to be most effective. Improved medical treatment of all types of stroke has resulted in a dramatic decline in death rates in recent decades. In 1950 nine in ten stroke victims died, compared to slightly less than one in three today.

Causes and symptoms

Causes

There are four main types of stroke: cerebral thrombosis, cerebral embolism, subarachnoid hemorrhage, and intracerebral hemorrhage. *Cerebral thrombosis* and *cerebral embolism,* known as *ischemic strokes,* are caused by blood clots that block an artery supplying the brain, either in the brain itself or in the neck. They account for 70–80% of all strokes. *Subarachnoid hemorrhage* and *intracerebral hemorrhage* are *hemorrhagic strokes* that occur when a blood vessel bursts around or in the brain, either from trauma or excess internal pressure. Hypertension (high blood pressure) and atherosclerosis are usually contributing factors in these types of strokes.

CEREBRAL THROMBOSIS. Cerebral thrombosis, the most common type of stroke, occurs when a blood clot, or thrombus, forms within the brain itself, blocking blood

flow through the affected vessel. This is usually due to atherosclerosis (hardening) of brain arteries, caused by a buildup of fatty deposits inside the blood vessels. Cerebral thrombosis occurs most often at night or early in the morning, and is often preceded by a TIA. Recognizing the occurrence of a TIA, and seeking immediate treatment, is an important step in stroke prevention.

CEREBRAL EMBOLISM. Cerebral embolism occurs when a blood clot from elsewhere in the circulatory system breaks free. If it becomes lodged in an artery supplying the brain, either in the brain or in the neck, it can cause a stroke. The most common cause of cerebral embolism is atrial fibrillation, which occurs when the upper chambers (atria) of the heart beat weakly and rapidly, instead of slowly and steadily. Blood within the atria does not empty completely, and may form clots that can then break off and enter the circulation. Atrial fibrillation is a factor in about 15% of all strokes, but this risk can be dramatically reduced with daily use of anticoagulant medication (such as Heparin or Coumadin).

SUBARACHNOID HEMORRHAGE. In this type of stroke, blood spills into the subarachnoid space between the brain and cranium. As fluid builds up, pressure on the brain increases, impairing its function. Hypertension is a frequent cause of these types of stroke, but vessels with preexisting defects, such as an aneurysm, are also at risk for rupture. Aneurysms are most likely to burst when blood pressure is highest, and controlling blood pressure is an important preventive strategy. Subarachnoid hemorrhages account for about 7% of all strokes.

INTRACEREBRAL HEMORRHAGE. Representing about 10% of all strokes, intracerebral hemorrhage affects vessels and tissue within the brain itself. As with subarachnoid hemorrhage, bleeding deprives affected tissues of blood supply, and the accumulation of fluid within the inflexible skull creates pressure on the brain that can quickly become fatal. Despite this, recovery may be more complete for a person who survives hemorrhage than for one who survives a clot, because the effects of blood deprivation are usually not as severe.

Risk factors

Risk factors for stroke involve:

- *Age and sex*—the risk of stroke increases with age, doubling for each decade after age 55. Men are more likely to have a stroke than women.
- *Heredity*—People with a family history of stroke have an increased risk of stroke themselves. In addition, African-Americans, Asians, and Hispanics all have higher rates of stroke than whites, related partly to higher blood pressure.
- *Diseases*—People with diabetes, heart disease (especially atrial fibrillation), high blood pressure, or prior stroke are at greater risk for stroke. Patients with one or more TIAs have ten times the risk.
- *Other medical conditions*—Stroke risk increases with **obesity**, high blood cholesterol, or high red blood cell count.
- *Lifestyle choices*—Stroke risk increases with cigarette smoking (especially if combined with the use of oral contraceptives), a sedentary lifestyle, alcohol consumption above two drinks per day, and/or the use of cocaine or intravenous drugs.

Symptoms

Knowing the symptoms of stroke is as important as knowing those of a heart attack. Patients with stroke symptoms should seek emergency treatment without delay, which may mean dialing 911 rather than their family physician. Specific symptoms of a stroke depend on the type, but all types share some characteristics in common.

An embolic stroke usually comes on quite suddenly and is intense right from the start, while symptoms of a thrombotic stroke come on more gradually. Symptoms for these ischemic strokes may include:

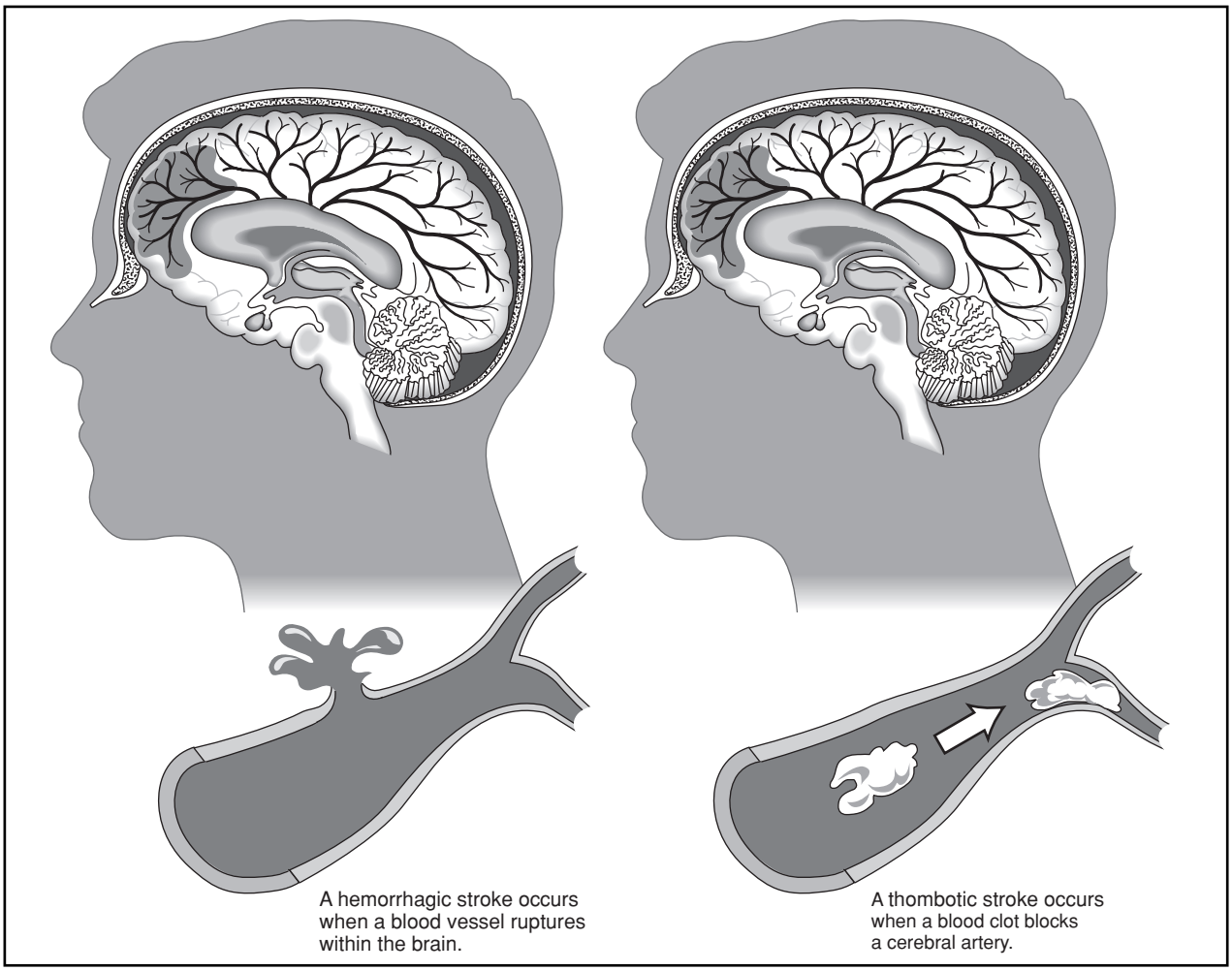
- blurring or decreased vision in one or both eyes
- severe headache, often described as "the worst headache of my life"
- weakness, numbness, or paralysis of the face, arm, or leg, usually confined to one side of the body
- dizziness, loss of balance or coordination, especially when combined with other symptoms

Hemorrhagic strokes are somewhat different. An intracranial hemorrhage exhibits any or all of the following symptoms:

- loss of consciousness
- altered mental state
- seizure
- vomiting or severe nausea
- extreme hypertension
- weakness, numbness, or paralysis, especially on one side of the body
- sudden, severe headache

Symptoms of a subarachnoid hemorrhage include:

- severe headache that begins suddenly



A hemorrhagic stroke (left) compared to a thrombotic stroke (right). *(Illustration by Hans & Cassady, Inc.)*

- nausea or vomiting
- stiff neck
- light intolerance
- loss of consciousness

Demographics

Each year, more than half a million people in the United States have a stroke. It is the third leading cause of death, killing about a third of its victims—approximately 150,000 Americans each year. For those that survive, stroke is the leading cause of disability. Two-thirds of all strokes occur in people over age 65, with men more affected than women, although women are more likely to die from a stroke. African-Americans suffer strokes more often than whites, and are more likely to be die from them as well. This may be because African-Americans tend to suffer from hypertension more frequently than other groups.

Diagnosis

Diagnosing a stroke begins with a careful medical history, especially concerning the onset and distribution of symptoms, presence of risk factors; in this way other possible causes are excluded. A brief neurological exam is performed to identify the degree and location of any deficits, such as weakness, lack of coordination, or vision loss.

Once stroke is suspected, imaging technology is used to determine what type the patient has suffered—a critical distinction that guides therapy. A noncontrast **computed tomography** scan (CT scan) can reliably identify hemorrhagic strokes, caused by uncontrolled bleeding in the brain. **Magnetic resonance imaging** (MRI), on the other hand, particularly diffusion-weighted imaging, can detect ischemic strokes, caused by blood clots, earlier and more reliably than CT scanning.

Blood and urine tests are also run to look for possible abnormalities. Other investigations that may be per-

formed to guide treatment include electrocardiogram, angiography, ultrasound, and electroencephalogram.

Treatment

When brain cells die during a stroke, they release toxic chemicals that can trigger a chain reaction that can injure or kill other nearby cells. Damage from stroke may be significantly reduced by emergency treatment, and is a significant factor in how fully a patient will recover.

Emergency treatment

Emergency treatment of an ischemic stroke attempts to dissolve the clot. This "thrombolytic therapy" is performed most often with tissue plasminogen activator (t-PA), which must be administered within three hours of the stroke event. (Patients who awaken with stroke symptoms are ineligible for this type of therapy, since the time of onset cannot be reliably determined.) t-PA therapy has been shown to improve recovery and decrease long-term disability in patients. It carries a 6.4% risk of inducing a cerebral hemorrhage, however, and is not appropriate for patients with bleeding disorders, very high blood pressure, known aneurysms, any evidence of intracranial hemorrhage, or incidence of stroke, head trauma, or intracranial surgery within the past three months. Patients with clot-related stroke who are ineligible for t-PA treatment may be treated with heparin or other blood thinners, or with aspirin or other anticlotting agents in some cases.

Emergency treatment of hemorrhagic stroke is aimed at controlling intracranial pressure that accompanies these types of strokes. New surgical techniques can effectively relieve the pressure, especially when begun soon after the stroke event occurs. Surgery for hemorrhage due to aneurysm may be performed if the aneurysm is close enough to the cranial surface to allow access. Ruptured vessels are closed off to prevent rebleeding. For aneurysms that are difficult to reach surgically, endovascular treatment, in which a catheter is guided from a larger artery up into the brain to reach the aneurysm, may be effective. Small coils of wire are discharged into the aneurysm, which plug it up and block off blood flow from the main artery.

Rehabilitation

Rehabilitation refers to a comprehensive program designed to regain as much function as possible and compensate for permanent losses. Approximately 10% of stroke survivors are without any significant disability and able to function independently. Another 10% are so severely affected that they must remain institutionalized for severe disability. The remaining 80% can return home with appropriate therapy, training, support, and care.

Rehabilitation is coordinated by a team of medical professionals and may include the services of a neurologist, a physician who specializes in rehabilitation medicine, a physical therapist, an occupational therapist, a speech-language pathologist, a nutritionist, a mental health professional, and a social worker. Rehabilitation services may be provided in an acute care hospital, rehabilitation hospital, long-term care facility, outpatient clinic, or at home.

The rehabilitation program is based on the patient's individual deficits and strengths. Strokes on the left side of the brain primarily affect the right half of the body, and vice versa. In addition, in left brain-dominant people, who constitute a significant majority of the population, left-brain strokes usually lead to speech and language deficits, while right-brain strokes may affect spatial perception. Patients with right-brain strokes may also deny their illness, neglect the affected side of their body, and behave impulsively.

Rehabilitation may be complicated by cognitive losses, including diminished ability to understand and follow directions. Poor results are more likely in patients whose strokes left them with significant or prolonged cognitive changes, sensory losses, language deficits, or incontinence.

PREVENTING COMPLICATIONS. Rehabilitation begins with prevention of medical complications, including stroke recurrence, using many of the same measures used to prevent stroke, such as smoking cessation and getting hypertension under control.

One of the most common medical complications following stroke is deep venous thrombosis, in which a clot forms within a limb immobilized by paralysis. Clots can also become lodged in an artery feeding the lungs, a condition called pulmonary embolism, that is a common cause of death in the weeks following a stroke. Resuming activity within a day or two after the stroke is an important preventive measure, along with use of elastic stockings on the lower limbs. Drugs that prevent clotting may also be given, including intravenous heparin and oral warfarin.

Weakness and loss of coordination of the swallowing muscles may impair swallowing (dysphagia), and allow food to enter the lower airway. This may lead to aspiration pneumonia, another common cause of death shortly after a stroke. Dysphagia may be treated with retraining exercises and temporary use of pureed foods.

Other medical complications include urinary tract infections, pressure ulcers, falls, and **seizures**. Not surprisingly, depression occurs in 30–60% of stroke patients; its severity is usually related to the level of per-

manent functional impairment It can be treated with antidepressants and **psychotherapy**.

TYPES OF REHABILITATIVE THERAPY. Brain tissue that dies in a stroke cannot regenerate. In some cases, however, rehabilitation training can help other brain regions perform the same functions of that tissue. In other cases, compensatory actions may be developed to replace lost abilities.

Physical therapy is used to maintain and restore range of motion and strength in affected limbs, and to maximize mobility in walking, wheelchair use, and transferring (from wheelchair to toilet or from standing to sitting, for instance). The physical therapist advises patients on mobility aids such as wheelchairs, braces, and canes. In the recovery period, a stroke patient may develop muscle spasticity and contractures (abnormal muscle contractions) that can be treated with a combination of stretching and splinting.

Occupational therapy improves self-care skills such as feeding, bathing, and dressing, and helps develop effective compensatory strategies and devices for activities of daily living. A speech-language pathologist focuses on communication and swallowing skills. When dysphagia is a problem, a nutritionist can advise alternative meals that provide adequate nutrition.

Psychological therapy can help treat depression or loss of thinking (cognitive) skills. A social worker may help coordinate services and ease the transition out of the hospital back into the home. Both **social workers** and mental health professionals help counsel the patient and family during the difficult rehabilitation period. Caring for a person affected with stroke requires a new set of skills and adaptation to new demands and limitations. Home caregivers may develop **stress**, anxiety, and depression—caring for the caregiver is an important part of the overall stroke treatment program. **Support groups** can provide an important source of information, advice, and comfort for stroke patients and caregivers; joining one can be an important step in the rehabilitation process.

Prognosis

Stroke is fatal for about 27% of white males, 52% of African-American males, 23% of white females, and 40% of African-American females. Stroke survivors may be left with significant deficits. Emergency treatment and comprehensive rehabilitation can significantly improve both survival and recovery.

Prevention

The risk of stroke can be reduced through lifestyle changes:

- stop smoking
- control blood pressure
- get regular exercise
- maintain a healthy weight
- avoid excessive alcohol consumption
- get regular checkups and follow the doctor's advice regarding diet and medicines

Use of high-estrogen dose oral contraceptives increase the chances for developing stroke, particularly in women who smoke and/or who are over 35. Currently, there are low-estrogen dose oral contraceptives, for which a clear relationship with stroke development is unclear.

Treatment of atrial fibrillation may also significantly reduce the risk of stroke. Preventive anticoagulant therapy may benefit those with untreated atrial fibrillation. Warfarin (Coumadin) has proven to be more effective than aspirin for those with higher risk.

Screening for aneurysms may be an effective preventive measure in those with a family history of aneurysms or autosomal polycystic kidney disease, which tends to be associated with aneurysms.

Resources

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Krishnan, K. Ranga Rama. "Depression as a contributing factor in cerebrovascular disease." *American Heart Journal* 140 (October 2000): 563.

ORGANIZATIONS

American Heart Association and American Stroke Association. 7272 Greenville Ave. Dallas, TX 75231. (214) 373-6300. <http://www.americanheart.org>.

National Stroke Association. 9707 E. Easter Lane, Englewood, Co. 80112. (800) 787-6537. <http://www.stroke.org>.

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Stuttering

Definition

There is no standard definition of stuttering, but most attempt to define stuttering as the blockages, discoordination, or fragmentations of the forward flow of speech (fluency). These stoppages, referred to as disfluencies, are often excessive and characterized by specific types of disfluency. These types of disfluencies include repetitions of sounds and syllables, prolongation of sounds, and blockages of airflow. Individuals who stutter are often aware of their stuttering and feel a loss of control when they are disfluent. Both children and adults stutterers expend an excessive amount of physical and mental energy when speaking. Older children and adults who stutter show myriad negative reactive behaviors, feelings, and attitudes. These behaviors, referred to as "secondary behaviors," make the disorder more severe and difficult.

Description

Stuttering is a confusing and often misunderstood developmental speech and language disorder. Before discussing stuttering, it is important to understand the concepts of speech fluency and disfluency. Fluency is generally described as the forward flow of speech. For most speakers, fluent speech is easy and effortless. Fluent speech is free of any interruptions, blockages, or fragmentations. Disfluency is defined as a breakdown or blockage in the forward flow of speech, or fluency. For all speakers, some occurrence of disfluency is normal. For example, people may insert short sounds or words, referred to as "interjections," when speaking; examples of such are "um," "like," or "uh." Also, speakers might repeat phrases, revise words or phrases, or sometimes repeat whole words for the purpose of clarification. For young children, disfluency is a part of the normal development of speech and language, especially during the preschool years (between the ages of two and five years).

The occurrence of disfluency is not the same as stuttering, though stuttered speech is characterized by an excessive amount of disfluency. The disfluencies produced by people who stutter will often be similar to those in the speech of individuals who do not stutter; however, certain types of disfluent behavior are likely to appear only in the speech of people who stutter. These disfluencies are sound and syllable repetitions (i.e., ca-ca-ca-cat), sound prolongations ("ssssss-salad," "ffffff-fish"), and complete blockages of airflow. These behaviors, often referred to as stuttering type disfluencies, distinguish stuttered speech from nonstuttered speech.

KEY TERMS

Disfluency—Disruptions, breakage, or blockages in the forward flow of speech.

Secondary behaviors—Negative behavioral, emotional, or cognitive reactions to stuttering.

Speech-language pathologist—Specialists trained in assessment and diagnosis of communication disorders.

Unlike speakers who do not stutter, most people who stutter react negatively to their disfluencies. A person may develop a number of physical reactions, including tension of the muscles involved in speech (tongue, jaw, lips, or chest, for example) and tension in muscles not related to speech (such as shoulders, limbs, and forehead). In addition to these physiological reactions, people who stutter will often have negative emotional reactions to the disorder. Among the emotions that people who stutter report are embarrassment, guilt, and frustration.

Finally, many people who stutter will develop a number of negative attitudes and beliefs regarding themselves and speaking—because of their stuttering. These may be negative attitudes and beliefs in certain speaking situations, with people with whom they interact, and in their own abilities. These physiological, emotional, and attitudinal (cognitive) reactions to stuttering, described as secondary stuttering behaviors, are often very disruptive to the communication process and the person's life.

Stuttering behaviors can develop and vary throughout the life span. Sometimes, children will experience periods when the stuttering appears to "go away," only to return in a more severe pattern. Many children, (estimates range between 50 and 80%) will develop normal fluency after periods of stuttering. For those who continue to stutter during late childhood, adolescence, and into adulthood, stuttering can become a chronic problem. Lifelong efforts will be needed to cope successfully with the behavior.

Due to the effect that stuttering has on communication, the person who stutters may experience certain difficulties in various parts of his/her life. These problems might be secondary to factors inside the person (symptoms of stuttering) and outside the person (society's attitudes toward stuttering and other barriers). For example, many people who stutter report difficulties in social settings. Children who stutter often experience teasing and other social penalties. Adolescents and adults also report a variety of social problems. Academic settings may be

difficult for children who stutter because of the emphasis schools place on verbal performance.

Finally, there appears to be some evidence that people who stutter might confront barriers in employment. These barriers might take the form of inability to do certain tasks easily (talking on the phone, for example), limitations in job choices, and discrimination in the hiring and promotion processes.

Causes and symptoms

Though research has not identified a single cause, there appears to be several factors that are viewed as being important to the onset and development of stuttering. Therefore, stuttering is often described as being related to multiple factors and having possibly multiple causes. First, there is a genetic predisposition to stutter, as evidenced by studies of families and twins. A second important factor in the onset of stuttering is the physiological makeup of people who stutter. Research suggests that the brains of people who stutter may function abnormally during speech production. These differences in functioning may lead to breakdowns in speech production and to the development of disfluent speech.

Third, there is some evidence that speech and language development is an important issue in understanding the development of stuttering. Studies have found some evidence that children who are showing stuttering type behaviors may also have other difficulties with speech-language. Additionally, children with speech-language delays will often show stuttering type behaviors. Finally, environmental issues have a significant impact on the development of stuttering behaviors. An environment that is overly stressful or demanding, may cause children to have difficulties developing fluent speech. Though the environment, in particular parental behaviors, does not cause stuttering, it is an important factor that might adversely affect a child who is operating at a reduced capacity for developing fluent speech.

There is no evidence that stuttering is secondary to a psychological disturbance. It is reasonable to assume that stuttering might have some effect on psychological adjustment and a person's ability to cope with speaking situations. People who stutter might experience a lower self-esteem and some might report feeling depressed. These feelings and difficulties with coping are most likely the result and not the cause of stuttering. In addition, several research studies have reported that many people who stutter report high levels of anxiety and **stress** when they are talking and stuttering. These feelings, psychological states, and difficulties with coping are most likely the result and not the cause of stuttering.

Generally, children begin to stutter between the ages of two and five years. Nevertheless, there are instances when individuals begin to show stuttering type behaviors in late childhood or as adults. These instances are often related to specific causes such as a **stroke** or a degenerative neurological disease. This type of stuttering, stuttering secondary to a specific neurological process, is referred to as neurogenic stuttering. In other cases, stuttering may be secondary to a psychological **conversion disorder** due to a psychologically traumatic event. When stuttering has abrupt onset secondary to a psychological trauma, it is described as psychogenic stuttering.

As stated earlier, the primary symptoms of stuttering include excessive disfluency, both stuttering and normal types (core behaviors), as well as physical, emotional, and cognitive reactions to the problem. These behaviors will vary in severity across people who stutter from very mild to very severe. Additionally, the behaviors will vary considerably across different speaking situations. There are specific situations when people tend to experience more stuttering (such as talking on the phone or with an authority figure) or less stuttering (speaking with a pet or to themselves, for example). It is likely that this variability might even extend to people having periods (days and even weeks) when they can maintain normally fluent or nonstuttered speech.

Demographics

Stuttering is a relatively low-prevalence disorder. Across all cultures, roughly 1% of people currently has a stuttering disorder. This differs from incidence, or number of individuals who have been diagnosed with stuttering at some point in their lives. Research suggests that roughly 5% of the population has ever been diagnosed with a stuttering disorder. This difference suggests that a significant number of individuals who stutter will someday develop through or ""grow out of" the problem. Research suggests that roughly 50-80% of all children who begin to stutter will stop stuttering. In addition, approximately three times as many men stutter as women. This ratio seems to be lower early in childhood, with a similar number of girls and boys stuttering. The ratio of boys to girls appears to get larger as children become older. This phenomenon suggests that males are more likely to continue to stutter than females.

Diagnosis

Speech-language pathologists are responsible for making the **diagnosis** and managing the treatment of adults and children who stutter. Preferably, a board-certified speech-language pathologist board should be sought for direct **intervention** or consulting. Diagnosis of stut-

tering, or identifying children at risk for stuttering, is difficult because most children will show excessive disfluencies in their speech. With children, diagnostic procedures include the collection and analysis of speech and disfluent behaviors in a variety of situations. In addition, the child's general speech-language abilities will be evaluated.

Finally, the speech-language pathologist will interview parents and teachers regarding the child's general developmental, speech-language development, and their perceptions of the child's stuttering behaviors. For adults and older children, the diagnostic procedures will also include gathering and analyzing speech samples from a variety of settings. In addition, the speech-language pathologist will conduct a lengthy interview with the person about their stuttering and history of their stuttering problem. Finally, the person who stutters might be asked to report his/her attitudes and feelings related to stuttering, either while being interviewed or by completing a series of questionnaires.

Treatments

General considerations

It is generally accepted that conducting interventions with children and families early in childhood (preschool) is the most effective means of total recovery from stuttering. The chances for a person to fully recover from stuttering by obtaining near-normal fluency are reduced as the person ages. This is why early intervention is critical. For older children and adults for which stuttering has become a chronic disorder, the focus of therapy is on developing positive coping mechanisms for dealing with the problem. This therapy varies in success based on the individual.

Treatment options for young children

Treatment of young children generally follows one of two basic approaches. These approaches may also be combined into a single treatment program. The first type of approach, often referred to as indirect therapy, focuses on altering the environment to allow the child opportunities to develop fluent speech. With this approach, counseling parents regarding the alteration of behaviors that affect fluency is the focus. For example, parents may be taught to reduce the amount of household stress or in the level of speech-language demands being placed on the child. In addition, parents may be advised to change characteristics of their speech, such as their speech rate and turn-taking style; this is done to help their children develop more fluent speech.

The other basic approach in treatment with young children targets the development of fluent speech. This type of approach, often referred to as direct therapy, teaches children to use skills that will help them improve fluency and they are sometimes given verbal rewards for producing fluent speech.

Treatment options for older children and adults

Treatment approaches for older children and adults usually take one of two forms. These approaches target either helping the person to modify his/her stuttering or modify his/her fluency. Approaches that focus on modifying stuttering will usually teach individuals to reduce the severity of their stuttering behaviors by identifying and eliminating all of the secondary or reactive behaviors. Individuals will also work to reduce the amount of emotional reaction toward stuttering.

Finally, the speech-language pathologist will help the individual to learn techniques that allow them to stutter in an easier manner. Therapy does not focus on helping the individual to speak fluently, though most individuals will attain higher levels of fluency if this approach is successful. The other groups of approaches will focus on assisting adults and children who stutter to speak more fluently. This type of therapy, which focuses less on changing secondary and emotional reactions, helps the person to modify their speech movements in a specific manner that allows for fluent sounding speech. These procedures require the individual to focus on developing new speech patterns. This often requires a significant amount of practice and skill. The successful outcome of these approaches is nonstuttered, fluent sounding speech. Many therapists will integrate stuttering modification and fluency shaping approaches into more complete treatment programs. In addition, psychological counseling may be used to supplement traditional speech therapy.

Prognosis

Complete alleviation of recovery from stuttering is most likely possible when children and their families receive treatment close to the time of onset. Thus, early identification and treatment of stuttering is critical. For older children and adults, stuttering becomes a chronic problem that requires a lifetime of formal and self-directed therapy. For individuals who show this more chronic form of the disorder, internal motivation for change and support from significant others is an important part of recovery.

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ORGANIZATIONS

National Stuttering Association. 5100 East La Palma, Suite #208, Anaheim Hills, CA 92807. <http://www.nsastutter.org>.

Stuttering Foundation of America. 3100 Walnut Grove Road, Suite 603, P.O. Box 11749, Memphis, TN 38111-0749. <http://www.stuttersfa.org>.

See also Speech-language pathology

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Substance abuse and related disorders

Definition

Substance-related disorders are disorders of intoxication, dependence, abuse, and substance withdrawal caused by various substances, both legal and illegal. These substances include: alcohol, **amphetamines**, caffeine, inhalants, nicotine, prescription medications that may be abused (such as sedatives), opioids (morphine, heroin), marijuana (cannabis), cocaine, hallucinogens, and phencyclidine (PCP).

Description

According to the mental health clinician's handbook, ***Diagnostic and Statistical Manual of Mental Disorders*** (the *DSM*), fourth edition text revised (*DSM-IV-TR*), all of the substances listed above, with the exceptions of nicotine and caffeine, have disorders of two types: substance use disorders and substance-induced disorders. Substance use disorders include abuse and dependence. Substance-induced disorders include intoxication, withdrawal, and various mental states (**dementia**, **psychosis**, anxiety, mood disorder, etc.) that the substance induces when it is used.

Substance dependence is characterized by continued use of a substance even after the user has experienced serious substance-related problems. The dependent user desires the substance ("craving") and needs more of the substance to achieve the effect that a lesser amount of the substance induced in the past. This phenomenon is known as tolerance. The dependent user also experiences withdrawal symptoms when the substance is not used. Withdrawal symptoms vary with the substance, but some symptoms may include increased heart rate, shaking, **insomnia**, **fatigue**, and irritability.

Substance abuse is continued use of a substance in spite of school- or work-related or interpersonal problems, but the user has not gotten dependent on the substance. The individual who abuses a substance may experience legal problems and may have problems fulfilling responsibilities, such as caring for a child.

Intoxication is the direct effect of the substance after an individual has used or has been exposed to the substance. Different substances affect individuals in various ways, but some of the effects seen in intoxication might include impaired judgment, emotional instability, increase or decrease in appetite, or changed sleep patterns.

The *DSM-IV-TR* does not recognize caffeine abuse or dependence, but does recognize the caffeine-induced disorders caffeine intoxication (restlessness, nervousness, excitement, etc. after caffeine consumption), caffeine-induced anxiety disorder (feelings of anxiety or panic attacks after caffeine consumption), and caffeine-induced sleep disorder (usually insomnia, but some may experience excessive sleepiness when caffeine is not consumed). As for nicotine, the *DSM-IV-TR* recognizes nicotine dependence and nicotine withdrawal.

The *DSM-IV-TR* lists disorders in the following categories:

- alcohol-related disorders
- amphetamine-related disorders
- caffeine-related disorders
- cannabis-related disorders
- cocaine-related disorders
- hallucinogen-related disorders
- inhalant-related disorders
- nicotine-related disorders
- opioid-related disorders
- phencyclidine-related disorders
- sedative-, hypnotic-, or anxiolytic-related disorders
- polysubstance dependence

See also Addiction; Alcohol and related disorders; Amnestic disorders; Amphetamines and related disorders; Antianxiety drugs and abuse-related disorders; Caffeine and related disorders; Cannabis and related disorders; Cocaine and related disorders; Denial; Disease concept of chemical dependency; Hallucinogens and related disorders; Inhalants and related disorders; Nicotine and related disorders; Opioids and related disorders; Phencyclidine and related disorders; Polysubstance dependence; Sedatives and related disorders; Substance Abuse Subtle

KEY TERMS

Amphetamines—A group of powerful and highly addictive substances that stimulate the central nervous system. May be prescribed for various medical conditions, but are often purchased illicitly and abused.

Anxiety—A feeling of apprehension and fear characterized by physical symptoms (heart palpitations, sweating, and feelings of stress, for example).

Anxiolytic—A preparation or substance given to relieve anxiety; a tranquilizer.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Hallucinogens—Substances that cause hallucinations.

Hypnotic—A type of medication that induces sleep.

Inhalants—A class of drugs that are inhaled in order for the user to experience a temporary "high." These chemicals include volatile solvents (liquids that vaporize at room temperature) and aerosols (sprays that contain solvents and propellants), and include glue, gasoline, paint thinner, hair spray, and others. They are dangerous because they can cause hallucinations, delusions, difficulty breathing, headache, nausea, vomiting, and even "sudden sniffing death." Inhalants can also cause permanent damage to the brain, lung, kidney, muscle, and heart.

Opioids—Substances that reduce pain and may induce sleep. Some opioids are endogenous, which means that they are produced within the human body. Other opioids are produced by plants or formulated synthetically in the laboratory.

Phencyclidine—The full name of the drug commonly called PCP that is often abused to induce hallucinations.

Psychosis—Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an over-arching disorder, not a disorder in itself. (Plural: psychoses)

Sedative—A medication that induces relaxation and sleep.

Screening Inventory; Substance-induced anxiety disorder; Substance-induced psychotic disorder; Urine drug screening; Wernicke-Korsakoff syndrome

Resources

BOOKS

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* Fourth edition, text revised. Washington DC: American Psychiatric Association, 2000.

Substance Abuse Subtle Screening Inventory

Definition

The Substance Abuse Subtle Screening Inventory is also referred to as the SASSI. Dr. Glenn A. Miller developed the SASSI as a screening questionnaire for identifying people with a high probability of having a substance dependence disorder.

Purpose

The SASSI is intended for gathering information, organizing it, and using it to help make decisions about the likelihood of an individual having a substance dependence disorder, even if the individual does not acknowledge symptoms of the disorder or misuse of substances. Guidelines are available for professionals to flag individuals with a potential substance abuse disorder for further evaluation. Interpreting the results of the SASSI helps professionals understand their clients better and plan their treatment.

Precautions

When used by trained professionals, the SASSI can be an important tool in the assessment of substance use disorders. The SASSI is not intended to prove or diagnose an individual as an alcoholic or addict; it is intended to screen for a person who has a "high probability of having a substance dependence disorder." It should be kept in mind that a thorough assessment integrates other

available information, such as self-report and family history, and is done by a skilled professional. This comprehensive assessment is required to determine if an individual meets the accepted standards in the mental health professional's handbook, ***Diagnostic and Statistical Manual of Mental Disorders***, for a clinical **diagnosis** of a substance-related disorder.

The accuracy rate of the SASSI is 94%. Although that is very high, this means that there is a 6% probability that an individual will be misclassified based on SASSI scores. While the SASSI is a popular and widely used screening questionnaire, independent research on it has been limited. Some researchers have questions about the SASSI regarding the extent to which subscales measure what they are intended to measure and the accuracy of classification based on direct versus indirect scales. In addition, the SASSI is not to be used to discriminate against individuals, including disqualifying job applicants. It would be a violation of the Americans With Disabilities Act to eliminate a job applicant based on SASSI scores.

Description

The SASSI is a simple, brief one-page paper-and-pencil questionnaire that can be answered in 10 to 15 minutes. The SASSI is easy to administer, to individuals or groups, and can be objectively scored by hand and interpreted, based on objective decision rules, in a minute or two. Optical scanning equipment is available for mass scoring and interpretation. The SASSI does not require a high level of reading ability. The SASSI may be used by a variety of programs and professionals, including school counselors, student assistance programs, employee assistance programs, vocational counselors, psychotherapists, medical personnel, criminal justice programs, and other human service providers.

The SASSI went through rigorous scientific development over a 16-year period before it was first published in 1988. Two new scales were added, and the SASSI-2 was published in 1994. In 1997 the SASSI-3 was published with a new scale and increased accuracy. Items on the SASSI were selected based on established research methods and statistical analysis. Items were included that identified individuals with substance dependence disorders. The selected items were consistently answered differently by individuals with a substance dependence disorder compared to individuals without a substance dependence disorder.

In 1996, a Spanish version was made available. In addition to the paper and pencil format, computer versions of the SASSI, in several formats, are available.

Some questions on the SASSI ask how frequently clients have had certain experiences directly related to alcohol and other drugs. These are answered on a four-point scale, ranging from never to repeatedly. Some items that may appear to be unrelated to substance use (indirect or subtle items) are in a true/false format. Overall, the items make up 10 subscales. The results are reported on a profile form that is discussed with the client. There are separate profile forms for males and females. The objective scoring system results in a yes or no answer about whether the client has a high probability of having a substance dependence disorder. The SASSI-3 has been empirically tested and can identify substance dependence disorder with an overall accuracy of 94%. More specifically, the SASSI identifies individuals with a substance dependence disorder with 94% accuracy, and it identifies those without a substance dependence disorder with 94% accuracy. The accuracy of the SASSI is not significantly affected by gender, age, socioeconomic status, ethnicity, occupational status, marital status, educational level, drug of choice, and general level of functioning. Research is ongoing to improve the accuracy and usefulness of the SASSI.

Since 1990 an adolescent version of the SASSI has been available. The second version of the Adolescent SASSI (SASSI-A2) has a 94% overall accuracy of identifying an adolescent with a substance dependence disorder, including both substance abuse and substance dependence. The SASSI-A2 is designed to screen individuals who are 12 to 18 years old. The accuracy of the SASSI-A2 is not affected by the respondent's gender, age, ethnicity, education, employment status, living situation, prior legal history, or general level of functioning.

Results

A profile of the SASSI results will be reviewed with the client. The actual scores are plotted on a profile graph in comparison to a sample of people who were not being evaluated or treated for addictions or other clinical problems (also called a normative sample). Feedback is then given in terms of whether the individual has a high or low probability of having a substance dependence disorder. Individual scale scores may be used to come up with ideas or hypotheses for further evaluation and treatment. This information is based on clinical experience with the SASSI. The results may indicate issues that are important for treatment (such as difficulty acknowledging personal shortcomings, or primarily focusing on others' needs while unaware of one's own needs). The results may suggest an approach to take with the client (such as increasing awareness, or acknowledging and validating their feelings). The results may suggest a

treatment plan that the client may respond to (such as addiction self-help groups or an education-focused program). Finally, the results may indicate appropriate treatment goals for the client (anger management and/ or social skills, for example). The goal of providing feedback about SASSI results is to have a two-way sharing and understanding of information that is descriptive and not judgmental.

See also Substance abuse and related disorders

Resources

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Joneis Thomas, Ph.D.

KEY TERMS

Anticholinergic agents—Medicines that include atropine, belladonna, hyoscyamine, scopolamine, and related products; used to relieve cramps or spasms of the stomach, intestines, and bladder.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Obsessive-compulsive—Characterized by obsessive and compulsive behaviors.

Phobia—Irrational fear of places, things, or situations that lead to avoidance.

Psychoactive substance—A drug that produces mood changes and distorted perceptions; mind-altering drug.

Sympathomimetics—Drugs that mimic the effects of impulses conveyed by adrenergic postganglionic fibres of the sympathetic nervous system.

Substance-induced anxiety disorder

Definition

Prominent anxiety symptoms (i.e., generalized anxiety, panic attacks, obsessive-compulsive symptoms, or phobia symptoms) determined to be caused by the effects of a psychoactive substance is the primary feature of a **substance-induced psychotic disorder**. A substance may induce psychotic symptoms during intoxication (i.e., while the individual is under the influence of the drug) or during withdrawal (i.e., after an individual stops using the drug).

Description

A substance-induced anxiety disorder is subtyped or categorized based on whether the prominent feature is generalized anxiety, panic attacks, obsessive-compulsive symptoms, or phobia symptoms. In addition, the disorder is subtyped based on whether it began during intoxication on a substance or during withdrawal from a substance. A substance-induced anxiety disorder that begins during substance use can last as long as the drug is used. A substance-induced anxiety disorder that begins during withdrawal may first manifest up to four weeks after an individual stops using the substance.

Causes and symptoms

Causes

A substance-induced anxiety disorder, by definition, is directly caused by the effects of drugs—including alcohol, medications, and toxins. Anxiety symptoms can result from intoxication on alcohol, **amphetamines** (and related substances), caffeine, cannabis (marijuana), cocaine, hallucinogens, inhalants, phencyclidine (PCP) and related substances, and other or unknown substances. Anxiety symptoms can also result from withdrawal from alcohol, sedatives, hypnotics, and anxiolytics, cocaine, and other or unknown substances. Some of the medications which may induce anxiety symptoms include anes-

thetics and analgesics, sympathomimetics (epinephrine or norepinephrine, for example) or other bronchodilators, anticholinergic agents, anticonvulsants, antihistamines, insulin, thyroid preparations, oral contraceptives, antihypertensive and cardiovascular medications, antiparkinsonian medications, corticosteroids, antidepressant medications, **lithium carbonate**, and antipsychotic medications. Heavy metals and toxins, such as volatile substances like fuel and paint, organophosphate insecticides, nerve gases, carbon monoxide, and carbon dioxide may also induce anxiety.

Symptoms

The ***Diagnostic and Statistical Manual of Mental Disorders***, (*DSM-IV-TR*)—produced by the American Psychiatric Association and used by most mental health professionals in North America and Europe to diagnose mental disorders—notes that a **diagnosis** is made only when the anxiety symptoms are above and beyond what would be expected during intoxication or withdrawal and when severe. The following list is the criteria necessary for the diagnosis of a substance-induced anxiety disorder as listed in the *DSM-IV-TR*:

- Prominent anxiety, panic attacks, or obsessions or compulsions.
- Symptoms develop during, or within one month, of intoxication or withdrawal from a substance or medication known to cause anxiety symptoms.
- Symptoms are not actually part of another anxiety disorder (such as **generalized anxiety disorder**, phobias, **panic disorder**, or **obsessive-compulsive personality disorder**) that is not substance induced. For instance, if the anxiety symptoms began prior to substance or medication use, then another anxiety disorder is likely.
- Symptoms do not occur only during **delirium**.
- Symptoms cause significant distress or impairment in functioning.

Demographics

Little is known regarding the demographics of substance-induced anxiety disorders. However, it is clear that they occur more commonly in individuals who abuse alcohol or other drugs.

Diagnosis

Diagnosis of a substance-induced anxiety disorder must be differentiated from an anxiety disorder due to a general medical condition. There are some medical conditions (such as hyperthyroidism, hypothyroidism, or hypoglycemia) that can produce anxiety symptoms, and since individuals are likely to be taking medications for these conditions, it can be difficult to determine the cause of the anxiety symptoms. If the symptoms are determined to be due to the medical condition, then a diagnosis of an anxiety disorder due to a general medical condition is warranted. Substance-induced anxiety disorders also need to be distinguished from delirium, **dementia**, primary psychotic disorders, and substance intoxication and withdrawal.

Clinical history and physical examination are the best methods to help diagnose anxiety disorders in general; however, appropriate laboratory testing will most likely be necessary to specifically identify substance-induced anxiety disorder. Lab tests may include:

- complete blood count (CBC)
- chemistry panels
- serum and/or urine screens for drugs

Treatments

The underlying cause of the anxiety symptoms, as well as the specific type of symptoms, determine course of treatment and is often similar to treatment for a primary anxiety disorder such as generalized anxiety disorder, phobias, panic disorder, or **obsessive-compulsive disorder**. Appropriate treatment usually includes medication (antianxiety or antidepressant medication, for example).

Prognosis

Anxiety symptoms induced by substance intoxication usually subside once the substance responsible is eliminated. Symptoms persist depending on the half-life of the substances (i.e., how long it takes the before the substance is no longer present in an individual's system). Symptoms, therefore, can persist for hours, days, or weeks after a substance is last used. Obsessive-compulsive symptoms induced by substances sometimes do not disappear, even although the substance inducing them has been eliminated. More intensive treatment for the obsessive-compulsive symptoms would be necessary and should include a combination of medication and behavioral therapy.

Prevention

Little is documented regarding the prevention of substance-induced anxiety disorder. However, abstaining from drugs and alcohol, or using these substances only in moderation, would clearly reduce the risk of developing this disorder. In addition, taking medication under the supervision of an appropriately trained physician should

reduce the likelihood of a medication-induced anxiety disorder. Finally, reducing one's exposure to toxins and heavy metals would reduce the risk of toxin-induced anxiety disorder.

See also Alcohol and related disorders; Amphetamines and related disorders; Anti-anxiety drugs and abuse-related disorders; Anxiety and anxiety disorders; Caffeine and related disorders; Cannabis and related disorders; Cocaine and related disorders; Hallucinogens and related disorders; Inhalants and related disorders; Phencyclidine and related disorders; Psychosis; Sedatives and related disorders; Substance abuse and related disorders; Substance-induced psychotic disorder

Resources

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American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 4th edition, text revised. Washington, DC: American Psychiatric Association, 2000.

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Jennifer Hahn, Ph.D.

Substance-induced persisting amnestic disorder *see* **Amnestic disorders; Wernicke-Korsakoff syndrome**

KEY TERMS

Anticholinergic agents—Medicines that include atropine, belladonna, hyoscyamine, scopolamine, and related products; used to relieve cramps or spasms of the stomach, intestines, and bladder.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness.

Delusion—A false belief that is resistant to reason or contrary to actual fact.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Hallucinations—False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present. Hallucinations can also affect the senses of smell, touch, and taste.

Persecutory delusions—Unrealistic conviction of being harassed, tormented, and persecuted.

Psychotic/psychosis—Episodes of inability to accurately perceive reality, think logically, and speak or behave normally. Hallucinations and delusions are symptoms of psychosis.

Substance-induced psychotic disorder

Definition

Prominent psychotic symptoms (i.e., **hallucinations** and/or **delusions**) determined to be caused by the effects of a psychoactive substance is the primary feature of a substance-induced psychotic disorder. A substance may induce psychotic symptoms during intoxication (while the individual is under the influence of the drug) or during withdrawal (after an individual stops using the drug).

Description

A substance-induced psychotic disorder is subtyped or categorized based on whether the prominent feature is delusions or hallucinations. Delusions are fixed, false beliefs. Hallucinations are seeing, hearing, feeling, tasting, or smelling things that are not there. In addition, the disorder is subtyped based on whether it began during intoxication on a substance or during withdrawal from a substance. A substance-induced psychotic disorder that begins during substance use can last as long as the drug is used. A substance-induced psychotic disorder that begins during withdrawal may first manifest up to four weeks after an individual stops using the substance.

Causes and symptoms

Causes

A substance-induced psychotic disorder, by definition, is directly caused by the effects of drugs including alcohol, medications, and toxins. Psychotic symptoms can result from intoxication on alcohol, **amphetamines** (and related substances), cannabis (marijuana), cocaine, hallucinogens, inhalants, opioids, phencyclidine (PCP)

and related substances, sedatives, hypnotics, anxiolytics, and other or unknown substances. Psychotic symptoms can also result from withdrawal from alcohol, sedatives, hypnotics, anxiolytics, and other or unknown substances.

Some medications that may induce psychotic symptoms include anesthetics and analgesics, anticholinergic agents, anticonvulsants, antihistamines, antihypertensive and cardiovascular medications, antimicrobial medications, antiparkinsonian medications, chemotherapeutic agents, corticosteroids, gastrointestinal medications, muscle relaxants, nonsteroidal anti-inflammatory medications, other over-the-counter medications, antidepressant medications, and **disulfiram**. Toxins that may induce psychotic symptoms include anticholinesterase, organophosphate insecticides, nerve gases, carbon monoxide, carbon dioxide, and volatile substances (such as fuel or paint).

The speed of onset of psychotic symptoms varies depending on the type of substance. For example, using a lot of cocaine can produce psychotic symptoms within minutes. On the other hand, psychotic symptoms may result from alcohol use only after days or weeks of intensive use.

The type of psychotic symptoms also tends to vary according to the type of substance. For instance, auditory hallucinations (specifically, hearing voices), visual hallucinations, and tactile hallucinations are most common in an alcohol-induced psychotic disorder, whereas persecutory delusions and tactile hallucinations (especially formication) are commonly seen in a cocaine- or amphetamine-induced psychotic disorder.

Symptoms

The ***Diagnostic and Statistical Manual of Mental Disorders*** (*DSM-IV-TR*) notes that a **diagnosis** is made only when the psychotic symptoms are above and beyond what would be expected during intoxication or withdrawal and when the psychotic symptoms are severe. Following are criteria necessary for diagnosis of a substance-induced psychotic disorder as listed in the *DSM-IV-TR*:

- Presence of prominent hallucinations or delusions.
- Hallucinations and/or delusions develop during, or within one month of, intoxication or withdrawal from a substance or medication known to cause psychotic symptoms.
- Psychotic symptoms are not actually part of another psychotic disorder (such as **schizophrenia**, **schizophreniform disorder**, **schizoaffective disorder**) that is not substance induced. For instance, if the psychotic symptoms began prior to substance or medication use, then another psychotic disorder is likely.
- Psychotic symptoms do not only occur during **delirium**.

Demographics

Little is known regarding the demographics of substance-induced **psychosis**. However, it is clear that substance-induced psychotic disorders occur more commonly in individuals who abuse alcohol or other drugs.

Diagnosis

Diagnosis of a substance-induced psychotic disorder must be differentiated from a psychotic disorder due to a general medical condition. Some medical conditions (such as temporal lobe epilepsy or Huntington's chorea) can produce psychotic symptoms, and, since individuals are likely to be taking medications for these conditions, it can be difficult to determine the cause of the psychotic symptoms. If the symptoms are determined to be due to the medical condition, then a diagnosis of a psychotic disorder due to a general medical condition is warranted.

Substance-induced psychotic disorder also needs to be distinguished from delirium, **dementia**, primary psychotic disorders, and substance intoxication and withdrawal. While there are no absolute means of determining substance use as a cause, a good patient history that includes careful assessment of onset and course of symptoms, along with that of substance use, is imperative. Often, the patient's testimony is unreliable, necessitating the gathering of information from family, friends, coworkers, employment records, medical records, and the like. Differentiating between substance-induced disorder and a psychiatric disorder may be aided by the following:

- Time of onset: If symptoms began prior to substance use, it is most likely a psychiatric disorder.
- Substance use patterns: If symptoms persist for three months or longer after substance is discontinued, a psychiatric disorder is probable.
- Consistency of symptoms: Symptoms more exaggerated than one would expect with a particular substance type and dose most likely amounts to a psychiatric disorder.
- Family history: A family history of mental illness may indicate a psychiatric disorder.
- Response to substance abuse treatment: Clients with both psychiatric and substance use disorders often have serious difficulty with traditional substance abuse treatment programs and relapse during or shortly after treatment cessation.
- Client's stated reason for substance use: Those with a primary psychiatric diagnosis and secondary substance use disorder will often indicate they "medicate symptoms," for example, drink to dispel auditory hallucina-

tions, use stimulants to combat depression, use depressants to reduce anxiety or soothe a manic phase. While such substance use most often exacerbates the psychotic condition, it does not necessarily mean it is a substance-induced psychotic disorder.

Unfortunately, psychological tests are not always helpful in determining if a psychotic disorder is caused by substance use or is being exacerbated by it. However, evaluations, such as the MMPI-2 MAC-R scale or the Wechsler Memory Scale—Revised, can be useful in making a differential diagnosis.

Treatments

Treatment is determined by the underlying cause and severity of psychotic symptoms. However, treatment of a substance-induced psychotic disorder is often similar to treatment for a primary psychotic disorder such as schizophrenia. Appropriate treatments may include psychiatric **hospitalization** and antipsychotic medication.

Prognosis

Psychotic symptoms induced by substance intoxication usually subside once the substance is eliminated. Symptoms persist depending on the half-life of the substances (i.e., how long it takes the before the substance is no longer present in an individual's system). Symptoms, therefore, can persist for hours, days, or weeks after a substance is last used.

Prevention

There is very little documented regarding prevention of substance-induced psychotic disorder. However, abstaining from drugs and alcohol or using these substances only in moderation would clearly reduce the risk of developing this disorder. In addition, taking medication under the supervision of an appropriately trained physician should reduce the likelihood of a medication-induced psychotic disorder. Finally, reducing one's exposure to toxins would reduce the risk of toxin-induced psychotic disorder.

See also Alcohol and related disorders; Amphetamines and related disorders; Antianxiety drugs and abuse-related disorders; Cannabis and related disorders; Cocaine and related disorders; Hallucinogens and related disorders; Inhalants and related disorders; Opioids and related disorders; Phencyclidine and related disorders; Psychosis; Sedatives and related disorders; Substance abuse and related disorders; Substance-induced anxiety disorders

Resources

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Jennifer Hahn, Ph.D.

Suicide

Definition

Suicide is defined as the intentional taking of one's own life. In some European languages, the word for suicide translates into English as "self-murder " Until the end of the twentieth century, approximately, suicide was considered a criminal act; legal terminology used the Latin phrase *felo-de-se*, which means "a crime against the self." Much of the social **stigma** that is still associated with suicide derives from its former connection with legal judgment, as well as with religious condemnation.

In the social climate of 2002, suicidal behavior is most commonly regarded— and responded to— as a psychiatric emergency.

Demographics of suicide

In the United States, the rate of suicide has continued to rise since the 1950s. More people die from suicide than from homicide in North America. Suicide is the eighth leading cause of death in the U.S., and the third leading cause of death for people aged 15 to 24. There are over 30,000 suicides per year in the U.S., or about 86 per day; each day about 1,500 people attempt suicide.

The demographics of suicide vary considerably from state to state. Some states, like Pennsylvania, have suicide rates that are very close to the national average; others, such as Connecticut, have significantly lower rates. However, other states have much higher rates than the national average. These variations are due in part to differences among age groups and racial groups, and between men and women. Males are three to five times more likely to succeed in their suicide attempts than females, but females are more likely to attempt suicide. Most suicides occur in persons below the age of 40; how-

KEY TERMS

Assisted suicide—A form of self-inflicted death in which a person voluntarily brings about his or her own death with the help of another, usually a physician, relative, or friend.

Cortisol—A steroid hormone released by the cortex (outer portion) of the adrenal gland when a person is under stress. Cortisol levels are now considered a biological marker of suicide risk.

Diathesis—The medical term for predisposition. The stress/diathesis model is a diagram that is used to explain why some people are at greater risk of suicidal behavior than others.

Euthanasia—The act of putting a person or animal to death painlessly or allowing them to die by withholding medical services, usually because of a painful and incurable disease. Mercy killing is another term for euthanasia.

Frontal cortex—The part of the human brain associated with aggressiveness and impulse control. Abnormalities in the frontal cortex are associated with an increased risk of suicide.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Slow suicide—A term used to refer to lifestyle behaviors known to shorten life expectancy, such as smoking, drinking heavily, having unsafe sex, etc.

Suicide gesture—Attempted suicide characterized by a low-lethality method, low level of intent or planning, and little physical damage. Pseudocide is another term for a suicide gesture.

ever, elderly Caucasians are the sector of the population with the highest suicide rate.

Race is also a factor in the demographics of suicide. Between 1979 and 1992, the suicide rate of Native Americans was 1.5 times the national average, with young males between the ages of 15 and 24 accounted for 64% of Native American deaths by suicide. Asian-American women have the highest suicide rate among all women over the age of 65. Further, between 1980 and 1996 the suicide rate more than doubled for African-American males between the ages of 15 and 19.

High-risk factors

Research indicates that the following factors increase a person's risk of suicide:

- Male sex.
- Age over 75.
- A family history of suicide.
- A history of suicide attempts.
- A history of **abuse** in childhood.
- Traumatic experiences after childhood
- Recent stressful events, such as separation or divorce, job loss, or death of spouse.
- Chronic medical illness. Patients with AIDS have a rate of suicide 20 times that of the general population.
- Access to a gun. Death by firearms is now the fastest-growing method of suicide among men and women. Nearly 57% of deaths caused by guns in the U.S. are suicides.
- Alcohol or substance abuse. While mood-altering substances do not cause a person to kill himself/herself, they weaken impulse control.
- High blood cholesterol levels.
- Presence of a psychiatric illness. Over 90% of Americans who commit suicide have a mental illness. Major depression accounts for 60% of suicides, followed by **schizophrenia**, alcoholism, substance abuse, **borderline personality disorder**, Huntington's disease, and epilepsy. The lifetime mortality due to suicide in psychiatric patients is 15% for major depression; 20% for **bipolar disorder**; 18% for alcoholism; 10% for schizophrenia; and 5–10% for borderline and certain other personality disorders.

Low-risk factors

Factors that lower a person's risk of suicide include:

- a significant friendship network outside the workplace
- religious faith and practice
- a stable marriage
- a close-knit extended family
- a strong interest in or commitment to a project or cause that brings people together, including community service, environmental concerns, neighborhood associations, animal rescue groups, etc.

Suicide in other countries

Suicide has become a major social and medical problem around the world. The World Health Organization (WHO) reported that one million people worldwide died from suicide in the year 2000. That is a global mortality rate of 16:100,000—or one death by suicide every 40 seconds. Since the mid-1950s, suicide rates around the world have risen by 60%. Rates among young people have risen even faster, to the point where they are now the age group at highest risk in 35% of the world's countries.

The specific demographics, however, vary from country to country. China's pattern, for example, is very different from that of most other countries. China has a suicide mortality rate of 23:100,000, with a total of 287,000 deaths by suicide each year. The rate for women is 25% higher than that for men, and rates in rural areas are three times higher than in cities. The means also vary; In China, Sri Lanka, and Turkey the primary means of suicide is ingestion of pesticides, rather than using guns.

Suicide in children and adolescents

The suicide rate among children and adolescents in the U.S. has risen faster than that of the world population as a whole. The suicide rate for Caucasian males aged 15 to 24 years has tripled since 1950; and it has more than doubled for Caucasian females in the same age bracket. In 1999, a survey of high school students found that 20% had seriously considered suicide or attempted it in the previous year. Of adolescents who do commit suicide, 90% have at least one diagnosable psychiatric disorder at the time of their death. Most frequently it is major depression, substance abuse disorder, or **conduct disorder**. Adolescents are particularly susceptible to dramatic or glamorized portrayals of suicide in the mass media.

Causes

Suicide is an act that represents the end result of a combination of factors in any individual. One model that has been used by clinicians to explain why people suffering under the same life stresses respond differently is known as the stress/diathesis model. Diathesis is a medical term for a predisposition that makes some people more vulnerable to thoughts of suicide. Components of a person's diathesis may include:

Neurobiological and genetic factors

Post-mortem studies of the brains of suicide victims indicate that the part of the brain associated with controlling agression and other impulsive behaviors (the frontal cortex) has a significantly lower level of serotonin, a neurotransmitter associated with mood disorders. Low serotonin levels are correlated with major depression. In addition, suicide victims have higher than normal levels of cortisol, a hormone produced in stressful situations, in the tissues of their central nervous system. Studies of the levels of other neurotransmitters in brain tissue are underway.

Other research has indicated that abuse in childhood may have permanent effects on the level of serotonin in the brain, possibly "resetting" the level abnormally low. In addition, twin studies have suggested that there may be genetic susceptibility in males to both suicidal ideation and suicide attempts which cannot be explained by inheritance of common psychiatric disorders. No twin studies of susceptibility to suicide in women have yet been reported.

History and lifestyle

Other components of a diathesis include:

- Chronic illness
- Traumatic experiences after childhood
- Alcohol or substance abuse
- High blood cholesterol levels.

Factors in the wider society

In addition to factors at the individual level, factors in the wider society have been identified as contributing to the rising rate of suicide in the United States:

- Stresses on the nuclear family, including divorce and economic hardship.
- The loss of a set of moral values held in common by the entire society.
- The weakening of churches, synagogues, and other mid-range social groups outside the family. In the past, these institutions often provided a sense of belonging for people from troubled or emotionally distant families.
- Frequent geographical moves, which makes it hard for people to make and keep long-term friendships outside their immediate family.
- Sensationalized treatment of suicide in the mass media. A number of research studies have shown that there is a definite risk of "contagion" suicides from irresponsible reporting, particularly among impressionable adolescents.
- The development over the past century of medications that allow relatively painless suicide. For most of human history, the available means of suicide were uncertain, painful, or both.
- The easy availability of firearms in the United States.

Treatment of attempted suicide

Researchers estimate that 8–25 people attempt suicide for every person who completes the act. Suicide attempts can be broadly categorized along a continuum that ranges from seriously planned attempts involving a highly lethal method that fail by good fortune, to impulsive or poorly planned attempts using a less lethal method. Suicide attempts at the lower end of the spectrum are sometimes referred to as suicide gestures or pseudocide.

A suicide attempt of any kind, however, is treated as a psychiatric emergency by rescue personnel. Treatment in a hospital emergency room includes a complete psychiatric evaluation, a mental status examination, and a detailed assessment of the circumstances surrounding the attempt. The physician will interview relatives or anyone else who accompanied the patient in order to obtain as much information as possible. As a rule, suicide attempts requiring advance planning, including precautions taken against discovery, and the use of violent or highly lethal methods are regarded as the most serious. The patient will be kept under observation while decisions are made about the need for **hospitalization**.

A person who has attempted suicide and who is considered a serious danger to him- or herself or to others can be hospitalized against their will. The doctor will base the decision on the severity of the patient's depression or agitation; availability of friends, relatives, or other social support; and the presence of other suicide risk factors, including a history of previous suicide attempts, substance abuse, recent stressful events, and symptoms of **psychosis**. If the attempt is judged to be a nonlethal suicide gesture, the patient may be released after the psychiatric assessment is completed.

Related issues

Survivors of suicide

One group of people that is often overlooked in discussions of suicide is the friends and family bereaved by the suicide. It is estimated that each person who kills him- or herself leaves six survivors to deal with the aftermath. On the basis of this figure, there are at least 4.5 million survivors of suicide in the United States. In addition to the **grief** that ordinarily accompanies death, survivors of suicide often struggle with feelings of guilt and shame as well. In spite of a general liberalization of social attitudes since World War II, suicide is still stigmatized in many parts of Europe and the United States. Survivors often benefit from group or individual **psychotherapy** in order to work through such issues as wondering whether they could have prevented the suicide or whether they are likely to commit suicide themselves. Increasing numbers of clergy as well as mental health professionals are taking advanced training in counseling survivors of suicide.

Assisted suicide

One question that has been raised in developed countries as the average life expectancy increases is the legalization of assisted suicide for persons suffering from a painful terminal illness. Physician-assisted suicide has become a topic of concern since it was legalized by recent legislation in the Netherlands (in April 2001) and in the state of Oregon. It is important to distinguish between physician-assisted suicide and euthanasia, or "mercy killing." Assisted suicide, which is often called "self-deliverance" in Britain, refers to a person's bringing about his or her own death with the help of another person. Because the other person is often a physician, the act is often called "doctor-assisted suicide." Euthanasia strictly speaking means that the physician or other person is the one who performs the last act that causes death. For example, if a physician injects a patient with a lethal overdose of a pain-killing medication, he or she is performing euthanasia. If the physician leaves the patient with a loaded syringe and the patient injects himself or herself with it, the act is an assisted suicide. As of 2002, assisted suicide is illegal everywhere in the United States except for Oregon, and euthanasia is illegal in all fifty states.

Media treatment of suicide

In 1989, the Centers for Disease Control (CDC) sponsored a national workshop to address the issue of the connection between sensationalized media treatments of suicide and the rising rate of suicide among American youth. The CDC and the American Association of Suicidology subsequently adopted a set of guidelines for media coverage of suicide intended to reduce the risk of suicide by contagion.

The CDC guidelines point out that the following types of reporting may increase the risk of "copycat" suicides:

- Presenting oversimplified explanations of suicide, when in fact many factors usually contribute to it. One example concerns the suicide of the widow of a man who was killed in the collapse of the World Trade Center on September 11, 2001. Most newspapers that covered the story described her death as due solely to the act of terrorism, even though she had a history of depressive illness.

- Excessive, ongoing, or repetitive coverage of the suicide.
- Sensationalizing the suicide by inclusion of morbid details or dramatic photographs. Some news accounts of the suicide of an Enron executive in January 2002 are examples of this problem.
- Giving "how-to" descriptions of the method of suicide.
- Referring to suicide as an effective coping strategy or as a way to achieve certain goals.
- Glorifying the act of suicide or the person who commits suicide.
- Focusing on the person's positive traits without mentioning his or her problems.

Prevention

Brain research is an important aspect of suicide prevention as of 2002. Since major depression is the single most common **diagnosis** in suicidal people, earlier and more effective recognition of depression is a necessary preventive measure. Known biological markers for an increased risk of suicide can now be correlated with personality profiles linked to suicidal behavior under **stress** to help identify individuals at risk. In addition, brain **imaging studies** using **positron emission tomography** (PET) are presently in use to detect abnormal patterns of serotonin uptake in specific regions of the brain. Genetic studies are also yielding new information about inherited predispositions to suicide.

A second major preventive measure is education of clinicians, media people, and the general public. Public health studies carried out in Sweden have shown that seminars for primary care physicians in the recognition and treatment of depression resulted in a rise in the number of prescriptions for antidepressants and a drop in suicide rates. Education of the general public includes a growing number of CDC, NIMH, and other web sites posting information about suicide, tips for identifying symptoms of depressed and suicidal thinking, and advice about helping friends or loved ones who may be at risk. Many of these web sites have direct connections to suicide hotlines.

An additional preventive strategy is restricting access to firearms in the developed countries and to pesticides and other poisons in countries where these are the preferred method of suicide.

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American Association of Suicidology. Suite 310, 4201 Connecticut Avenue, NW, Washington, DC 20008. (202) 237-2280. Fax: (202) 237-2282. <www.suicidology.org>.

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Support groups

Definition

Support groups are an informal resource that attempts to provide healing components to a variety of problems and challenges. An informal support outside of family, friends, or professionals often provides greater understanding, more similarity (from individuals experiencing similar life events), an opportunity for empathy and altruism, and a sense of identity for participants. Learning new ways to handle challenges, cope with changes, and maintain new behaviors are all important aspects of the support group experience.

A characteristic unique to support groups is the mutual support members are able to provide one another. This support and validation from other group members help facilitate personal growth and change in a way that individual therapy cannot. Although experts and professionals can provide support and positive direction, the mutual exchange of information between group members is a powerful experience that often induces lasting change.

Description

Most support groups are facilitated or led by lay persons, often in conjunction with existing organizations (such as NAMI, the National Alliance for the Mentally Ill, or AA, Alcoholics Anonymous). Support groups usually have a set meeting time (generally weekly or monthly), and an open format. Open format means that the groups are ongoing, and members have the option of attending when it is convenient for them. This is in contrast to other types of structured treatment or psycho-educational groups that may meet for a certain number of sessions, with the expectation that participants attend every meeting. The open format allows members to feel some degree of anonymity, and to participate as they are comfortable. For some people, simply attending meetings and listening to the experiences of others can be helpful.

The healing power of groups is well documented, and support groups offer many of the same therapeutic characteristics as more structured groups. These factors include: altruism (chance to help others), belongingness, universality (there are others who struggle with similar challenges), interpersonal learning, guidance, catharsis, identification, self-understanding, instillation of hope, and existential factors (such as the search for larger meaning in life). Each of these factors is directly related to the mutual support that members provide one another.

Support groups are generally less structured than psycho-educational groups or therapy groups; however, each group usually sets its own norms, rules, and schedules. Some groups, such as AA, traditionally reserve time for individual members to discuss their own challenges and progress in front of the group. Others bring in speakers periodically to provide information about disorders or specific coping skills. However, the strength of support groups lies in its members, and their willingness to share their own experiences, challenges, and solutions in the context of the group.

In addition to these traditional, face-to-face support groups, technology has had an impact on the functioning and availability of support groups. There are many listserves, e-mail groups, and chat groups that provide information about specific life problems (adoption of children outside the United States, for example), certain types of mental illness, and specific health problems. While there is always the risk of communicating with others who are not honest, many people benefit from these Internet interactions. Some individuals are actually more comfortable participating in Internet support groups due to the greater anonymity they offer.

There are a variety of problems and challenges that are addressed in support groups. Generally speaking, the severity of the symptom, as well as the phase of the illness or disorder, will determine whether participation in a support group is appropriate. For more severe types of mental illness, such as **schizophrenia**, or depression with psychotic episodes, a support group is probably not the optimal **intervention**, particularly at initial onset. After stabilization through therapy and medication (as appropriate), a support group may offer an important addition to more formal treatment. In these cases, the

socialization, interpersonal relationships, and social support that can be gained through the group may not be available elsewhere, and as such, it can be a very positive experience for the participant. In a group situation, a participant can learn how to express feelings in a healthy and positive way, practice assertive communication, receive feedback about appropriate and inappropriate content for conversation, receive feedback about nonverbal communication, learn new ways to ask for help from others, be able to help others, learn how to form friendships, and learn new coping skills and behaviors.

Types of support groups

Various types of support groups exist. Some groups provide support for very specific types of loss, illness, or life adjustment. A representative sample is listed below.

BEREAVEMENT/GRIEF COUNSELING GROUPS. Bereavement and **grief counseling** groups provide support to people who have experienced a loss. There are groups for people who have lost a spouse or partner, parents, children, or pets. There are specific groups for people who have lost a loved one due to homicide, **suicide**, SIDS, cancer, or miscarriage. These groups help individuals adjust to the death of a family member or friend, learn how to accept the loss, honor the memory of their loved one, and adjust to life after the loss.

MEDICAL SUPPORT GROUPS. Medical support groups may be more short-term than other types of support groups, depending on the specific disorder. Some groups are formed to help patients adjust to specific treatments, such as chemotherapy or radiation, while others focus on longer-term adjustment and recovery issues, such as a breast cancer support group. These groups may have a stronger educational component to help members understand physical changes they may be experiencing as a result of their medical procedures.

WEIGHT LOSS GROUPS. Although these groups are very specific in their focus, their individual structures can vary greatly. Some weight loss support groups are actively involved in the process of losing weight, and may include monitoring of diet and exercise, while others focus on maintaining weight loss, and, therefore, may focus more on social support.

MENTAL HEALTH/ILLNESS SUPPORT GROUPS. These groups usually focus on specific disorders, such as bipolar or eating disorders. Members of these support groups are often at different phases in dealing with their illnesses, and, therefore, the needs and contributions of individual members may vary greatly from meeting to meeting.

Most support groups are facilitated by lay persons, and usually have a set meeting time (generally weekly or monthly), and an open format. The open format allows members to feel some degree of anonymity, and to participate as they are comfortable. For some people, simply attending meetings and listening to the experiences of others can be helpful. *(Richard T. Nowitz/ CORBIS. Photo reproduced by permission.)*

FAMILY SUPPORT GROUPS. Family support groups, such as CHADD for parents of children with ADD, or NAMI for families with members who struggle with any type of mental illness, provide support from other parents and children who may be feeling the same level of frustration and exasperation. Meeting others who truly understand one's experience has a very powerful effect. For many parents, participation in a support group is the first opportunity to learn that there are other parents who are experiencing the same challenges and frustrations.

LIFE TRANSITIONS GROUPS. Life transitions groups include divorce and aging support groups. Support groups for children of divorce also exist in many communities and schools.

ADDICTIONS SUPPORT GROUPS. Traditional **addiction** support groups include Alcoholics Anonymous (AA), Narcotics Anonymous (NA), and Gambler's Anonymous (GA). Many of these groups follow the traditional "12-step" program of working through various aspects of the addiction, and, as such, are more structured than many other types of support groups.

Support group locations

Support groups meet in many different locations within a community. Hospitals and medical centers may provide meeting locations for medical support groups. **Community mental health** centers, inpatient psychiatric programs, and residential treatment centers are com-

mon locations for mental health and mental illness-related support groups. Life transition groups are often provided through schools, senior centers, and daycare centers. Bereavement groups and addiction support groups often meet in churches, community meeting rooms of local businesses, and mental health agencies.

Structure of support groups

Support groups are most successful when composed of persons close in age who are experiencing similar life challenges. Support groups are usually led by members of the group, such as the chapter president or another member of the organizing group. Some support groups may be led by paraprofessionals if they are offered as part of an aftercare program associated with a treatment facility.

Support groups usually have explicit norms and expectations for member participation, such as respecting members' feelings and opinions, and coming to meetings free from drugs or alcohol. Due to the open nature of most support groups, members typically feel free to miss a session here or there, which is usually not acceptable in a treatment or therapy group.

Conclusion

Group experiences can be very powerful in changing behavior and maintaining that change. The support group becomes part of the individual's daily life, and promotes healthy functioning by providing reminders about change and support when he or she is feeling down or is drawn toward old patterns. It also provides opportunities to own one's change by helping others. These factors contribute to the positive prognosis for most who participate in a group experience. However, a person could be harmed by a group experience as well. Much of this risk is dependent on the characteristics of individual members, particularly in support groups that operate without professional guidance. For example, if certain individuals dominate the group with their own agenda, perhaps at the expense of other group members, then the experience may have a negative impact on more vulnerable individuals.

See also Grief counseling and therapy

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Surmontil *see* **Trimipramine**

SVR-20 *see* **Sexual Violence Risk-20**

Symmetrel *see* **Amantadine**

Systematic desensitization

Definition

Systematic desensitization is a technique used to treat phobias and other extreme or erroneous fears based on principles of **behavior modification**.

Purpose

Systematic desensitization is used to help the client cope with phobias and other fears, and to induce relaxation. In progressive relaxation, one first tightens and then relaxes various muscle groups in the body. During the alternating clenching and relaxing, the client should be focusing on the contrast between the initial tension and the subsequent feelings of relaxation and softening that develop once the tightened muscles are released. After discovering how muscles feel when they are

deeply relaxed, repeated practice enables a person to recreate the relaxed sensation intentionally in a variety of situations.

After learning relaxation skills, the client and therapist create an "anxiety hierarchy." The hierarchy is a catalogue of anxiety-provoking situations or stimuli arranged in order from least to most distressing. For a person who is frightened by snakes, the anxiety hierarchy might start with seeing a picture of a snake, eventually move to viewing a caged snake from a distance, and culminate in actually handling a snake. With the therapist's support and assistance, the client proceeds through the anxiety hierarchy, responding to the presentation of each fearful image or act by producing the state of relaxation. The person undergoing treatment stays with each step until a relaxed state is reliably produced when faced with each item. As tolerance develops for each identified item in the series, the client moves on to the next. In facing more menacing situations progressively, and developing a consistent pairing of relaxation with the feared object, relaxation rather than anxiety becomes associated with the source of their anxiety. Thus, a gradual desensitization occurs, with relaxation replacing alarm. Several means of confronting the feared situations can be used. In the pre-computer era, the exposure occurred either through imagination and visualization (imagining a plane flight) or through actual real-life — or so-called *in vivo* — encounters with the feared situation (going on an actual plane flight). More recently, during the 1990s, virtual reality or computer simulated exposure has come to be utilized in lieu of *in vivo* exposure. Research findings indicate that mental imagery is the least effective means of exposure; *in vivo* and virtual reality exposure appear to be indistinguishable in terms of effectiveness.

KEY TERMS

Behavior modification—An approach to therapy based on the principles of operant conditioning. Behavior modification seeks to replace undesirable behaviors with preferable behaviors through the use of positive or negative reinforcement.

Classical conditioning—In psychology, a process in which a previously neutral stimulus eventually produces a specific response by being paired repeatedly with another stimulus that produces that response. The best-known example of classical conditioning is Pavlov's dogs, who were conditioned to salivate when they heard a bell ring (the previously neutral stimulus) because the sound had been paired repeatedly with their feeding time.

Description

Systematic desensitization is a therapeutic **intervention** that reduces the learned link between anxiety and objects or situations that are typically fear-producing. The aim of systematic desensitization is to reduce or eliminate fears or phobias that sufferers find are distressing or that impair their ability to manage daily life. By substituting a new response to a feared situation — a trained contradictory response of relaxation which is irreconcilable with an anxious response — phobic reactions are diminished or eradicated.

This behavior modification technique, which is founded on the principles of classical conditioning, was developed by Joseph Wolpe in the 1950s. Some of the most common fears treated with desensitization include fear of public speaking, fear of flying, stage fright, elevator phobias, driving phobias and animal phobias. Relaxation responses are trained to occur through *progressive relaxation training*, a technique initially perfected by Edmund Jacobson during the 1930s.

Precautions

Because of the potential for extreme panic reactions to occur, which can increase the phobia, this technique should only be conducted by a well-qualified, trained professional. Also, the relaxation response should be thoroughly learned before confronting the anxiety-provoking hierarchy.

Normal results

Desensitization is an effective form of therapy. Individuals who have a positive response are enabled to resume daily activities that were previously avoided. The majority of persons undergoing this treatment show symptom reduction.

See also Anxiety disorders

Resources

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T

Tacrine

Definition

Tacrine is a drug used to treat **dementia** associated with **Alzheimer's disease**. In the United States tacrine is sold under the brand name drug Cognex. It is also sometimes called tetrahydroaminoacridine or THA.

Purpose

Tacrine is used to treat symptoms of Alzheimer's disease in people with mild to moderate illness. The drug may result in mild improvements in thinking for a short period. Tacrine does not cure or stop the progression of Alzheimer's disease.

Description

The Food and Drug Administration approved tacrine in 1993 for treating Alzheimer's disease. In Alzheimer's disease, some cells in specific regions of the **brain** die. Because of this cell death, these brain cells lose their ability to transmit nerve impulses. Brain cells normally transmit nerve impulses another by secreting various chemicals known as **neurotransmitters**.

Brain cells that make and secrete a neurotransmitter called acetylcholine are affected early in the course of Alzheimer's disease. Tacrine helps prevent the breakdown of acetylcholine in the brain, thus temporarily increasing its concentration. In doing so, tacrine may improve the thinking process by facilitating nerve impulse transmission within the brain.

Tacrine is available as capsules in several different strengths. Tacrine is broken down by the liver.

Recommended dosage

The dose of tacrine will be different for different people. An initial dosage of tacrine is usually 10 mg taken four times per day. This dose should be continued for four weeks while liver function is monitored. If no adverse liver effects are detected, the dosage should be increased to 20 mg taken four times per day. Higher dosages such as 30-40 mg given four times per day may also be used. Liver function must be monitored every other week during the first 16 weeks of treatment. After 16 weeks of tacrine therapy, liver function can be assessed every three months. Dosage increases should not occur more often than every four weeks. Tacrine should be taken on an empty stomach between meals, but if stomach upset occurs, it may be taken with food.

If problems in liver function arise, tacrine may be stopped, or the dosage reduced, until liver function returns to normal. Very specific guidelines should be followed by physicians with regard to dosage adjustments based upon the severity of liver effects. Newer drugs that work in the same manner as tacrine are not as toxic to the liver and may be preferred for patients just beginning therapy for Alzheimer's-type dementia.

Precautions

Tacrine may cause liver damage. It may not be the best drug to treat symptoms of Alzeheimer's disease in people with known liver damage. If these individuals take tacrine, their liver function should be closely monitored. Tacrine may also slow heart rates, increase acid secretion in the stomach, make urination difficult, cause breathing difficulties, or contribute to **seizures**. As a result, it should be used carefully in people with certain heart conditions, those who are prone to stomach ulcers, people with bladder obstruction, individuals with asthma, and those with a history of seizure disorders.

People should not stop taking tacrine suddenly because this could cause behavioral disturbances. The drug may be stopped slowly if improvements are not noted by caregivers or physicians.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Dementia—A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Placebo—An inactive substance or preparation used as a control in experiments with human subjects to test the effectiveness of a drug or herbal preparation. Some patients may experience a medicinal response or experience side effects to a placebo simply because they have faith in its powers even though it contains no medicine.

Side effects

The most common side effect of tacrine is impaired liver function. This causes 8% of people to stop taking the drug. Other common side effects occurring in at least 5% of people and at twice the rate of placebo are stomach upset (nausea, vomiting, diarrhea, indigestion, or anorexia), muscle aches, and difficulty walking. Side effects affecting the stomach appear to be more severe at higher dosages.

Side effects that occur less often are behavioral disturbances, abnormal thinking, hostility, tremor, inability to sleep, slow heart rates, changes in blood pressure, urinary difficulties, rash, flushing, aggravation of asthma, or cold-like symptoms.

Health care providers should be informed immediately if nausea, vomiting, loose stools, or diarrhea occur soon after the dose of tacrine is increased or if rash, jaundice (yellow tinge to eyes or skin), or changes in stool color occur at any time.

Interactions

Many drugs can alter the effects of tacrine. Some drugs such as dicyclomine may lessen the effects of tacrine. Other drugs such as **propranolol**, cimetidine, ciprofloxacin, **fluoxetine**, **fluvoxamine**, neostigmine, or bethanechol may increase some of tacrine's side effects. **Rivastigmine** may interact with some of the drugs used to relax muscles during surgery. The interaction increases the effects of both drugs.

Tacrine may also diminish the effects of levodopa and increase the side effects of theophylline. Smoking cigarettes may reduce the effectiveness of tacrine.

Resources

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T'ai chi *see* **Bodywork therapies**

Talk therapy

Definition

Talk therapy is an alternate name for the various forms of **psychotherapy** that emphasize the importance of the client or patient speaking to the therapist as the main means of expressing and resolving issues.

Description

Psychoanalysis, the first modern form of psychotherapy, was called the "talking cure," and the many varieties of therapy practiced today are still characterized by their common dependence on a verbal exchange between the counselor or therapist and the person seeking help. Some of these therapies that are characterized

by the verbal exchange include: **cognitive-behavioral therapy**, behavior therapy, **couples therapy**, **family therapy**, **grief counseling** and therapy, **group therapy**, **interpersonal therapy**, **person-centered therapy**, **psychodynamic psychotherapy**, and **rational emotive therapy**. Both **self-help groups** and **support groups** also rely on the discussion of an issue as a main part of the cure.

KEY TERMS

Akathisia—An uncontrollable feeling of restlessness.

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

Tardive dyskinesia

Definition

Tardive dyskinesia is a neurological disorder consisting of abnormal, involuntary body movements caused by certain medicines. It is usually associated with long-term use of medicines for treating **schizophrenia** and other psychotic disorders.

Description

Tardive means "late" and dyskinesia means "abnormal movements." It refers to abnormal body movements that occur after a person has been taking a certain medicine for a long period of time. It sometimes starts after the medicine has been discontinued. In the early stages, the movements may be so subtle that neither the person nor the people around him or her notice them. For instance, the person may blink rapidly or lick their lips often. In later stages, the movements become noticeable and may affect the person's physical abilities.

Other types of tardive dyskinesia can occur. In tardive dystonia, there are abnormal contractions of the neck and shoulder muscles. In tardive akathisia, the person feels restless all the time.

Causes and symptoms

Causes

It is not altogether certain what causes tardive dyskinesia. The medicines that cause it affect how nerve impulses are transmitted across gaps between nerve cells (synapses). They do this in part by blocking a chemical made by the body called dopamine. After a while, the nerves seem to become hypersensitive to dopamine. Stimulation by even a little bit of dopamine may cause the abnormal movements.

The medicines most commonly associated with tardive dyskinesia include:

- Antipsychotic medicines used to treat schizophrenia and other psychoses. These are also known as neuroleptic medicines.
- Levodopa or L-dopa, which is used to treat Parkinson's disease (although high doses of L-dopa may actually help control tardive dyskinesia).
- Antiemetic medicines used to control nausea and vomiting.
- Tricyclic antidepressants used to treat depression and other mood disorders.
- Other medicines that block dopamine.

Symptoms

Symptoms of tardive dyskinesia include:

- involuntary movements of the face, including frowning, blinking, smiling, lip licking, mouth puckering, biting or chewing, clenching the jaw, sticking out the tongue, or rolling the tongue around in the mouth
- involuntary movements of the hands, arms, feet, or legs, such as twitching the hands or tapping the feet
- trunk movements, such as rocking, twisting, or squirming
- grunting or trouble speaking because of involuntary movements of the diaphragm

Movements may be rapid or slow and complicated. They are usually irregular and do not follow a pattern.

Demographics

Tardive dyskinesia develops in about a third of all people who take antipsychotic medicines for several years. The risk is higher in older patients. Approximately 5% of young adults taking antipsychotic medicines will develop tardive dyskinesia after a year of treatment, compared with a rate of 30% in elderly patients.

Treatments

Each case is treated differently. In some cases, the medicine causing the problem can be stopped. However, most people taking antipsychotic medicine cannot stop taking the medicine because of the high risk that their **psychosis** will return. Some newer antipsychotic medicines such as **clozapine** (Clozaril) do not seem to cause tardive dyskinesia. It may be possible to switch to a newer antipsychotic medication. If not, it may be possible to lower the dose to a level that does not cause the movements. There is controversy about whether or not "drug holidays" reduce the likelihood of developing tardive dyskinesia. "Drug holidays" are planned periods of time in which the person goes off the medicine, then resumes it.

Vitamin E has been shown to be helpful in patients, especially those who have had the problem for less than five years. L-dopa and some other medicines are sometimes helpful.

Prognosis

The earlier the problem is noticed and treatment begun, the better chance there is that the abnormal movements will go away. Most patients have a noticeable improvement in their symptoms within a year and a half. However, some abnormal movements may remain. People who are over 60 have a greater chance of having the problem go away on its own.

See also Medication-induced movement disorders

Resources

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National Alliance for the Mentally Ill. Colonial Place Three, 2107 Wilson Blvd., Suite 300, Arlington, VA 22201. Telephone: (703) 524-7600. NAMI HelpLine: (800) 950-NAMI. < www.nami.org>.

National Institute of Neurological Disorders and Stroke. Part of the National Institutes of Health (NIH), Bethesda, Maryland 20892.<www.ninds.nih.gov>.

Jody Bower, M.S.W

TAT *see* **Thematic Apperception Test**

Tegretol *see* **Carbamazepine**

Temazepam

Definition

Temazepam is a drug that belongs to a family of drugs known as benzodiazepines. Temazepam is sold under the brand name Restoril in the United States. Temazepam is also available as a generic.

Purpose

Temazepam is given to patients with sleeping problems (**insomnia**). It is often prescribed for insomnia characterized by frequent awakening during the night or by awakening early in the morning.

Description

Temazepam is one of several drugs in the class known as benzodiazepines. These drugs produce a variety of effects, but most cause some degree of drowsiness (sedation). Temazepam is used almost exclusively as a hypnotic, or drug given to help people fall asleep. It is nearly always taken just before bedtime. This drug produces its effects in the body by slowing down certain impulses in the **brain**, allowing the patient to fall asleep.

Recommended dosage

The typical dose starting for adults is 7.5–15 mg taken *just before* bedtime. The maximum recommended dose is 30 mg. Elderly patients and those in a weakened condition may need only 7.5 mg. The doctor should determine the dose in children 18 years of age and younger on an individual basis.

Precautions

The doctor should monitor any patient taking this drug to ensure that significant side effects do not develop. Insomnia that lasts longer than seven to 10 days may point to a significant medical problem that should be thoroughly evaluated. Temazepam should not be combined with alcohol or other drugs that lower the level of activity in the central nervous system. Examples of such drugs include prescription pain medications, antihistamines, **barbiturates**, and muscle relaxants. Some people may develop dizziness, lightheadedness, and clumsiness after taking temazepam. These side effects are especially common in the elderly.

Those with a history of anemia, liver disease, kidney disease, drug abuse, serious psychological disorders, and **suicide** attempts should be given temazepam only after being thoroughly evaluated by their physician. This cau-

tion also applies to persons with a history of lung disease, seizure disorders, and narrow-angle glaucoma.

People who are taking temazepam should not stop taking it abruptly. Instead, the dose should be reduced gradually. Withdrawal symptoms, including depressed mood, sweating, abdominal cramps, muscle cramps, vomiting, **seizures**, and shakiness can develop if the medication is stopped suddenly.

Although patients are instructed to take temazepam in the evening before bedtime, they often experience side effects the next day, particularly drowsiness and loss of coordination or clumsiness. Pregnant women should not use this drug because it increases the risk of birth defects in the baby. Nursing mothers should not be given temazepam because it can make their babies drowsy and unable to nurse properly. Patients should not operate heavy machinery or drive a car while they are taking temazepam or any other benzodiazepine.

Side effects

Temazepam is a relatively safe drug, safer than most of the benzodiazepines. Its less serious but more common side effects include clumsiness or unsteady behavior, dizziness, drowsiness, and slurred speech. Some patients taking temazepam experience abdominal cramps, dry mouth, constipation, diarrhea, headache, nausea, vomiting, a giddy sense of well-being, and changes in sexual drive.

A small number of patients taking temazepam have experienced anger outbursts, confusion, mental depression, unusually low blood pressure, memory difficulties, nervousness, irritability, and muscle weakness. As they can with many prescription drugs, people can overdose on temazepam. Symptoms of a temazepam overdose include extreme drowsiness, significant confusion, breathing difficulties, a very slow heartbeat, and staggering.

Rebound insomnia is one of the more common side effects of tapering a patient's dose of temazepam. Rebound insomnia is a reaction characterized by the re-emergence of the symptom that the drug was originally given to suppress, namely problems with falling or staying asleep. When a person takes a medication for sleep on a regular basis, the body adjusts to the presence of the drug. It tries to counteract the effects of the medication. As a result, when the person stops taking the sleeping medication, the body will take a few nights to return to its normal condition. During this period of readjustment, the person may experience a few sleepless hours each night. In addition, people often mistake the rebound insomnia for the ordinary variety, and consider it a good reason to continue taking the temazepam.

KEY TERMS

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Barbiturates—A class of medications (including Seconal and Nembutal) that causes sedation and drowsiness. They may be prescribed legally, but may also be used as drugs of abuse.

Generic—A term that refers to a medication that is not protected by a registered trademark.

Hypnotic—A type of medication that induces sleep.

Insomnia—A chronic inability to sleep or to remain asleep throughout the night.

Narrow-angle glaucoma—An eye disorder caused by a buildup of fluid pressure inside the eyeball due to an abnormally small angle between the iris (the colored portion of the eye) and the cornea (the transparent front part of the eye).

Rebound effect—A physical reaction to stopping a medication characterized by the reappearance of the symptom(s) that the medication was given to suppress. For example, people who stop taking temazepam may experience rebound excitability and sleeping problems.

Sedation—A state of emotional or physical relaxation. The term is usually used to refer to this condition when it is produced by a medication.

Withdrawal—Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

People can also develop withdrawal symptoms even when they are gradually decreasing their dose of temazepam, particularly if the original dose was high. The more common withdrawal symptoms include sleeping difficulties, irritability, and nervousness. Less common withdrawal side effects include abdominal cramps, confusion, sweating, nausea, trembling, increased heart rate, and mental depression.

Interactions

Patients should always inform any health care provider that they see—doctors, dentists, nurses, and others—about all the medications they are taking, including temazepam. This information is important because temazepam interacts with certain other drugs, including

cimetidine (an antihistamine); **disulfiram** (a drug given to help patients control cravings for alcohol); or **clozapine** (an antipsychotic medication). Rifampin, which is an antibiotic, may decrease the effectiveness of the temazepam if the two are taken together. The most important warning, however, is that the patient should avoid drinking alcohol or taking other medications that cause drowsiness (such as antihistamines) while taking temazepam, because these substances will intensify its effects. Heavy smoking, however, interferes with the effectiveness of temazepam.

See also Sleep disorders

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Thematic Apperception Test

Definition

The Thematic Apperception Test, or TAT, is a projective measure intended to evaluate a person's patterns of thought, attitudes, observational capacity, and emotional responses to ambiguous test materials. In the case of the TAT, the ambiguous materials consist of a set of cards that portray human figures in a variety of settings and situations. The subject is asked to tell the examiner a story about each card that includes the following elements: the event shown in the picture; what has led up to it; what the characters in the picture are feeling and thinking; and the outcome of the event.

Because the TAT is an example of a *projective* instrument— that is, it asks the subject to project his or her habitual patterns of thought and emotional responses onto the pictures on the cards— many psychologists prefer not to call it a "test," because it implies that there are "right" and "wrong" answers to the questions. They consider the term "technique" to be a more accurate description of the TAT and other projective assessments.

Purpose

Individual assessments

The TAT is often administered to individuals as part of a battery, or group, of tests intended to evaluate personality. It is considered to be effective in eliciting information about a person's view of the world and his or her attitudes toward the self and others. As people taking the TAT proceed through the various story cards and tell stories about the pictures, they reveal their expectations of relationships with peers, parents or other authority figures, subordinates, and possible romantic partners. In addition to assessing the content of the stories that the subject is telling, the examiner evaluates the subject's manner, vocal tone, posture, hesitations, and other signs of an emotional response to a particular story picture. For example, a person who is made anxious by a certain picture may make comments about the artistic style of the picture, or remark that he or she does not like the picture; this is a way of avoiding telling a story about it.

The TAT is often used in individual assessments of candidates for employment in fields requiring a high degree of skill in dealing with other people and/or ability to cope with high levels of psychological stress— such as law enforcement, military leadership positions, religious ministry, education, diplomatic service, etc. Although the TAT should not be used in the differential **diagnosis** of mental disorders, it is often administered to individuals who have already received a diagnosis in order to match them with the type of **psychotherapy** best suited to their personalities. Lastly, the TAT is sometimes used for forensic purposes in evaluating the motivations and general attitudes of persons accused of violent crimes. For example, the TAT was recently administered to a 24-year-old man in prison for a series of sexual murders. The results indicated that his attitudes toward other people are not only outside normal limits but are similar to those of other persons found guilty of the same type of crime.

The TAT can be given repeatedly to an individual as a way of measuring progress in psychotherapy or, in some cases, to help the therapist understand why the treatment seems to be stalled or blocked.

Research

In addition to its application in individual assessments, the TAT is frequently used for research into specific aspects of human personality, most often needs for achievement, fears of failure, hostility and aggression,

and interpersonal object relations. "Object relations" is a phrase used in psychiatry and psychology to refer to the ways people internalize their relationships with others and the emotional tone of their relationships. Research into object relations using the TAT investigates a variety of different topics, including the extent to which people are emotionally involved in relationships with others; their ability to understand the complexities of human relationships; their ability to distinguish between their viewpoint on a situation and the perspectives of others involved; their ability to control aggressive impulses; self-esteem issues; and issues of personal identity. For example, one recent study compared responses to the TAT from a group of psychiatric inpatients diagnosed with dissociative disorders with responses from a group of non-dissociative inpatients, in order to investigate some of the controversies about **dissociative identity disorder** (formerly called multiple personality disorder).

Precautions

Students in medicine, psychology, or other fields who are learning to administer and interpret the TAT receive detailed instructions about the number of factors that can influence a person's responses to the story cards. In general, they are advised to be conservative in their interpretations, and to err "on the side of health" rather than of psychopathology when evaluating a subject's responses. In addition, the 1992 Code of Ethics of the American Psychological Association requires examiners to be knowledgeable about cultural and social differences, and to be responsible in interpreting test results with regard to these differences.

Experts in the use of the TAT recommend obtaining a personal and medical history from the subject before giving the TAT, in order to have some context for evaluating what might otherwise appear to be abnormal or unusual responses. For example, frequent references to death or **grief** in the stories would not be particularly surprising from a subject who had recently been bereaved. In addition, the TAT should not be used as the sole examination in evaluating an individual; it should be combined with other interviews and tests.

Cultural, gender, and class issues

The large number of research studies that have used the TAT have indicated that cultural, gender, and class issues must be taken into account when determining whether a specific response to a story card is "abnormal" strictly speaking, or whether it may be a normal response from a person in a particular group. For example, the card labeled 6GF shows a younger woman who is seated turning toward a somewhat older man who is standing behind her and smoking a pipe. Most male subjects do not react to this picture as implying aggressiveness, but most female subjects regard it as a very aggressive picture, with unpleasant overtones of intrusiveness and danger. Many researchers consider the gender difference in responses to this card as a reflection of the general imbalance in power between men and women in the larger society.

KEY TERMS

Apperception—The process of understanding through linkage with previous experience. The term was coined by one of the authors of the TAT to underscore the fact that people don't "perceive" the story cards in a vacuum; rather, they construct their stories on the basis of past experiences as well as present personality traits.

Battery—A number of separate items (such as tests) used together. In psychology, a group or series of tests given with a common purpose, such as personality assessment or measurement of intelligence.

Forensic—Pertaining to courtroom procedure or evidence used in courts of law.

Idiographic—An approach to interpreting the results of a projective test within the context of the individual subject's record.

Nomothetic—An approach to interpreting the results of a projective test in which the subject's answers are measured against a normative comparison sample.

Object relations—In psychology, a phrase that refers to the way in which a subject describes relationships with other people in their environment, and the ways in which he or she has internalized interpersonal relationships.

Projective test or projective measure—A type of psychological evaluation that assesses a person's thinking patterns, observational ability, feelings, and attitudes on the basis of responses to ambiguous test materials. Projective measures are not intended to diagnose psychiatric disorders, although they are often used in outcome studies to compare the effectiveness of different forms of psychotherapy.

Rorschach test—A commonly administered projective measure in which subjects are asked to describe a series of black or colored inkblots.

In the TAT, the test subject (the boy shown here) examines a set of cards that portray human figures in a variety of settings and situations, and is asked to tell a story about each card. The story includes the event shown in the picture, preceding events, emotions and thoughts of those portrayed, and the outcome of the event shown. The story content and structure are thought to reveal the subject's attitudes, inner conflicts, and views. *(Lew Merrim/ Science Source. Photo Researchers, Inc. Reproduced by permission.)*

Race is another issue related to the TAT story cards. The original story cards, which were created in 1935, all involved Caucasian figures. As early as 1949, researchers who were administering the TAT to African Americans asked whether the race of the figures in the cards would influence the subjects' responses. Newer sets of TAT story cards have introduced figures representing a wider variety of races and ethnic groups. As of 2002, however, it is not clear whether a subject's ability to identify with the race of the figures in the story cards improves the results of a TAT assessment.

Multiplicity of scoring systems

One precaution required in general assessment of the TAT is the absence of a normative scoring system for responses. The original scoring system devised in 1943 by Henry Murray, one of the authors of the TAT, attempted to account for every variable that it measures. Murray's scoring system is time-consuming and unwieldy, and as a result has been little used by later interpreters. Other scoring systems have since been introduced that focus on one or two specific variables—for example, hostility or depression. While these systems are more practical for clinical use, they lack comprehensiveness. No single system presently used for scoring the TAT has achieved widespread acceptance. The basic drawback of any scoring system in evaluating responses to the TAT story cards is that information that is not relevant to that particular system is simply lost.

Computer scoring

A recent subject of controversy in TAT interpretation concerns the use of computers to evaluate responses. While computers were used initially only to score tests with simple yes/no answers, they were soon applied to interpretation of projective measures. A computerized system for interpreting the Rorschach was devised as early as 1964. As of 2002, there are no computerized systems for evaluating responses to the TAT; however, users of the TAT should be aware of the controversies in this field. Computers have two basic limitations for use with the TAT: the first is that they cannot observe and record

the subject's vocal tone, eye contact, and other aspects of behavior that a human examiner can note. Second, computers are not adequate for the interpretation of unusual subject profiles.

Description

The TAT is one of the oldest projective measures in continuous use. It has become the most popular projective technique among English-speaking psychiatrists and psychologists, and is better accepted among clinicians than the Rorschach.

History of the TAT

The TAT was first developed in 1935 by Henry Murray, Christiana Morgan, and their colleagues at the Harvard Psychological Clinic. The early versions of the TAT listed Morgan as the first author, but later versions dropped her name. One of the controversies surrounding the history of the TAT concerns the long and conflict-ridden extramarital relationship between Morgan and Murray, and its reinforcement of the prejudices that existed in the 1930s against women in academic psychology and psychiatry.

It is generally agreed, however, that the basic idea behind the TAT came from one of Murray's undergraduate students. The student mentioned that her son had spent his time recuperating from an illness by cutting pictures out of magazines and making up stories about them. The student wondered whether similar pictures could be used in therapy to tap into the nature of a patient's fantasies.

Administration

The TAT is usually administered to individuals in a quiet room free from interruptions or distractions. The subject sits at the edge of a table or desk next to the examiner. The examiner shows the subject a series of story cards taken from the full set of 31 TAT cards. The usual number of cards shown to the subject is between 10 and 14, although Murray recommended the use of 20 cards, administered in two separate one-hour sessions with the subject. The original 31 cards were divided into three categories, for use with men only, with women only, or for use with subjects of either sex. Recent practice has moved away from the use of separate sets of cards for men and women.

The subject is then instructed to tell a story about the picture on each card, with specific instructions to include a description of the event in the picture, the developments that led up to the event, the thoughts and feelings of the people in the picture, and the outcome of the story. The examiner keeps the cards in a pile face down in front of him or her, gives them to the subject one at a time, and asks the subject to place each card face down as its story is completed. Administration of the TAT usually takes about an hour.

Recording

Murray's original practice was to take notes by hand on the subject's responses, including his or her nonverbal behaviors. Research has indicated, however, that a great deal of significant material is lost when notes are recorded in this way. As a result, some examiners now use a tape recorder to record subjects' answers. Another option involves asking the subject to write down his or her answers.

Interpretation

There are two basic approaches to interpreting responses to the TAT, called *nomothetic* and *idiographic* respectively. Nomothetic interpretation refers to the practice of establishing norms for answers from subjects in specific age, gender, racial, or educational level groups and then measuring a given subject's responses against those norms. Idiographic interpretation refers to evaluating the unique features of the subject's view of the world and relationships. Most psychologists would classify the TAT as better suited to idiographic than nomothetic interpretation.

In interpreting responses to the TAT, examiners typically focus their attention on one of three areas: the content of the stories that the subject tells; the feeling or tone of the stories; or the subject's behaviors apart from responses. These behaviors may include verbal remarks (for example, comments about feeling stressed by the situation or not being a good storyteller) as well as nonverbal actions or signs (blushing, stammering, fidgeting in the chair, difficulties making eye contact with the examiner, etc.) The story content usually reveals the subject's attitudes, fantasies, wishes, inner conflicts, and view of the outside world. The story structure typically reflects the subject's feelings, assumptions about the world, and an underlying attitude of optimism or pessimism.

Results

The results of the TAT must be interpreted in the context of the subject's personal history, age, sex, level of education, occupation, racial or ethnic identification, first language, and other characteristics that may be important. "Normal" results are difficult to define in a complex multicultural society like the contemporary United States.

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ORGANIZATIONS

American Psychological Association. 750 First Street, NE, Washington, DC 20002. (800) 374-2721. Web site: <http://www.apa.org>.

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Thioridazine

Definition

Thioridazine is a potent antianxiety and antipsychotic agent. It is a member of the phenothiazine family of compounds. In the United States, thioridazine is sold as under the brand name of Mellaril and is also available under its generic name.

Purpose

Thioridazine is used to manage psychotic disorders. It reduces excitement, abnormal levels of energy, excessive movements (hypermotility), and agitation. The drug is also useful in the short-term treatment of depression that accompanies anxiety, sleep disturbances, agitation, and tension. Thioridazine is used in short-term treatment of children who display seriously inappropriate responses to exciting stimuli.

Description

Thioridazine is used in treating anxiety and **psychosis**. When used for the treatment of **schizophrenia**, thioridazine reduces symptoms of emotional withdrawal, anxiety, tension, **hallucinations**, and suspiciousness. Compared to other phenothiazine drugs, it is less likely to cause vomiting and Parkinson-like symptoms.

It is often successfully used to treat children who have impulsive conduct, difficulty in maintaining attention, or show high levels of aggression or have poor tolerance for frustration when other drugs have failed.

Recommended dosage

The dosage of thioridazine must be adjusted to each individual for whom it is prescribed to achieve maximum therapeutic effects and to minimize side effects. The usual initial dosage for adults is 50 to 100 mg three times a day. This may be gradually increased to a maximum of 800 mg per day. Once the desired therapeutic effect has been achieved, the dosage should be stabilized. A typical maintenance dosage is 200 to 800 mg per day, given in three to four doses.

The usual initial dosage for adults being treated for symptoms of anxiety is 25 mg three times per day. After reaching equilibrium and controlling undesired symptoms, the typical maintenance dosage is 20 to 200 mg per day divided into three or four doses.

For children between the ages of two and 12, the usual daily dosage of thioridazine is 0.5 to 3.0 mg per kg of body weight. Severely psychotic children who are hospitalized may receive 25 mg twice each day.

Precautions

It is dangerous to give thioridazine to persons in a comatose state. **Seizures** due to thioridazine therapy have been reported but are unusual. A sudden decrease in blood pressure due to a change in body position (orthostatic hypotension) with accompanying lightheadedness,

may occur in people who have taken the drug. This is more common among women than among men.

Thioridazine increases the level of prolactin, a hormone that stimulates the mammary glands in the breast, in the blood. This is a potential problem for persons with a personal or family history of breast cancer and may increase the risk of breast cancer. For this reason, the benefits and risks of the drug must be carefully evaluated before it is administered.

Long-term use of thioridazine increases the probability of developing **tardive dyskinesia** (See below). Because of potentially serious side effects, the risks and benefits of thioridazine must be carefully explained and understood before the drug is started.

Side effects

A common side effect of thioridazine is drowsiness and lack of physical and mental alertness. This side effect is especially noticeable early in therapy. Patients taking it should refrain from performing hazardous activities requiring mental alertness or coordination. Other common side effects include greater sensitivity to the sun and increased risk of serious sunburn, dry mouth, constipation, and urinary retention. Urinary retention (difficulty starting a urine flow or passing urine,) is a particular problem in men with enlarged prostates.

Thioridazine use may lead to the development of symptoms that resemble Parkinson's disease, but that are not caused by Parkinson's. These symptoms may include a taut or mask-like expression on the face, drooling, tremors, pill-rolling motions in the hands, cogwheel rigidity (abnormal rigidity in muscles, characterized by jerky movements when the muscle is passively stretched), and a shuffling gait. Taking anti-Parkinson drugs **benztropine** mesylate or **trihexyphenidyl** hydrochloride along with **trifluoperazine** usually readily controls these symptoms.

Thioridazine has the potential to produce a serious side effect called tardive dyskinesia. This syndrome consists of involuntary, uncoordinated movements that may not disappear or may only partially improve after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage of thioridazine. It may also appear after thioridazine use has been discontinued. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period.

KEY TERMS

Akathisia—Agitated or restless movement, usually affecting the legs. Movement is accompanied by a sense of discomfort and an inability to sit, stand still, or remain inactive for periods of time. Akathisia is a common side effect of some neuroleptic (antipsychotic) medications.

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

Orthostatic hypotension—A sudden decrease in blood pressure due to a change in body position, as when moving from a sitting to standing position.

Prolactin—A hormone that stimulates milk production and breast development.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

An occasionally reported side effect of thioridazine is neuroleptic malignant syndrome. This is a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia (fast heartbeat), and arrhythmias (irregular heartbeat). People who think they may be experiencing any side effects from this or any other medication should talk to their physician promptly.

Interactions

Thioridazine increases the effect of drugs and substances that depress the central nervous system. This class of drugs includes anesthetics, opiates, **barbiturates**, atropine, and alcohol. These substances should be avoided or used sparingly by people taking thioridazine.

Propranolol increases the concentration of thioridazine. Concurrent administration of pindolol also increases the concentration of thioridazine. The reverse effect also occurs: thioridazine increases the concentration of pindolol in the body. Thioridazine may interact with other drugs used to treat mental disorders. People planning to take this drug should review the other medications they are taking with their doctor and pharmacist before starting the drug.

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American Academy of Clinical Toxicology. 777 East Park Drive, PO Box 8820, Harrisburg, PA 17105-8820. Telephone: (717) 558-7750. Fax: (717) 558-7845. Web site: <http://www.clintox.org/index.html>.

American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax: (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

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Thiothixene

Definition

Thiothixene is in a class of drugs called antipsychotics. It is available with a prescription under the generic name of thiothixene or the brand name Navane.

Purpose

Thiothixene is a drug used to treat symptoms of **schizophrenia**. It is also sometimes used to calm severely agitated people.

Description

Thiothixene has been used in the United States for many years as a treatment for schizophrenia. It is believed to modify the balance of naturally occurring chemicals in the **brain** called **neurotransmitters** that regulate the transmission of nerve impulses from cell to cell. The proper balance between neurotransmitters is responsible, in part, for maintaining mental well-being. Thiothixene is thought to alter the balance among neurotransmitters in a way that improves symptoms of schizophrenia.

Thiothixene is available in several different strengths as capsules, as an injection, and as a concentrated liquid form taken by mouth. It is broken down by the liver and eliminated from the body by the kidneys.

Recommended dosage

The dosage of thiothixene varies widely from one individual to another. Initially, 2 mg of thiothixene taken by mouth three times daily is used in milder cases. This dosage may be increased slowly. Fifteen to 30 mg per day is often an effective range.

For more severe cases, 5 mg taken by mouth twice per day is a common starting dosage, with slow increases to 20–30 mg per day. Up to 60 mg of thiothixene may

be taken daily. Doses greater than 60 mg per day usually do not provide any additional benefit, but may increase side effects.

Precautions

Thiothixene may alter the rhythm of the heart. As a result, it should not be used by people with a history of irregular or prolonged heart rhythms (long QT syndrome), those with heart failure, or people who have recently had a heart attack. People with other heart conditions should discuss with their physician whether thiothixene is the right antipsychotic drug for them.

Thiothixene may increase the tendency to have **seizures**. People who have had seizures in the past, including alcohol or drug-induced seizures, should take thiothixene only after discussing the risks and benefits with their physician. People taking thiothixene should call their doctor immediately if they experience any abnormal, involuntary muscle movements, because this adverse effect may be permanent. The risk of abnormal, involuntary muscle movements is believed to increase with long-term use of thiothixene and high dosages.

Thiothixene may increase body temperatures to dangerously high levels. People who exercise strenuously, those exposed to extreme heat, individuals taking drugs with anticholinergic effects (this includes many common antidepressants), and those prone to dehydration, should be alert to increased body temperatures and dehydration-related side effects. Fevers, difficulty moving muscles, irregular heartbeats, rapid heartbeats, or excessive sweating are warning signs of possible overheating that should be addressed by a physician immediately.

People taking thiothixene should have regular eye examinations, since use of thiothixene has been associated with abnormalities of the retina, the light-sensitive layer of the eye. Thiothixene may also alter reproductive hormone levels causing irregular menstrual periods, difficulty getting pregnant, enlarged breasts, and breast milk production. Thiothixene can cause enlarged breasts and breast milk secretion in men as well as women. People who have had breast cancer should not take thiothixene unless the benefits of this drug substantially outweigh the risks.

Thiothixene may cause drowsiness. People should not perform hazardous tasks that require mental alertness until they see how the drug affects them. This side effect usually diminishes with continued use of the drug. Thiothixene may make it more difficult to make a patient vomit after a drug overdose or accidental poisoning. Because there is a high incidence of **suicide** in all patients with psychotic illnesses, people using thiothixene should be observed carefully for signs of suicidal behavior. Women who are pregnant or breast-feeding should not take thiothixene.

KEY TERMS

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Antipsychotic—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Side effects

Common side effects associated with the use of thiothixene are abnormal muscle movements and muscle stiffness, muscle tremors, weight gain, sleepiness, dry mouth, dry eyes, difficulty urinating, constipation, and sudden decreases in blood pressure that cause dizziness when standing up suddenly.

Other side effects that may occur when using thiothixene are headaches, seizures, high blood pressure, rapid heartbeats, blurred vision, liver changes, irregular menstrual periods, abnormal blood cell counts, difficulty breathing, and rash.

Uncommon and serious side effects include neuroleptic malignant syndrome and **tardive dyskinesia**. Neuroleptic malignant syndrome is an unusual but potentially life-threatening condition. The person with

this syndrome becomes extremely rigid, has a high fever, rapid heart rate, and abnormalities on blood tests. The affected person also may have a difficult time breathing and may sweat, and will be admitted to the hospital. Tardive dyskinesia (TD) is a condition that may occur after a long period of using antipsychotic medications. TD is characterized by involuntary movements of the facial muscles and tongue, and may also involve muscles in the trunk or hands or feet. TD may disappear as soon as the medication is stopped, but it may not; if it does not, it is difficult to treat. These potential side effects should be discussed with the patient's doctor.

Interactions

When thiothixene is used with drugs such as bethanechol, **propranolol**, levodopa, and some antidepressants, some of the side effects associated with thiothixene may increase. Use of narcotic drugs with thiothixene may cause blood pressure to fall to dangerously low levels. If thiothixene is used with levodopa, the actions of levodopa may be diminished.

When thiothixene is used with **barbiturates** or lithium, thiothixene may be less effective. Because thiothixene may cause sleepiness, it should not be used with other drugs that also cause drowsiness, such as antidepressants, antihistamines, some pain relievers, and alcohol.

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Thorazine *see* **Chlorpromazine**

Tic disorders

Definition

Tic disorders are characterized by the persistent presence of tics, which are abrupt, repetitive involuntary movements and sounds that have been described as caricatures of normal physical acts. The best known of these disorders is Tourette's disorder, or Tourette's syndrome.

Description

Tics are sudden, painless, nonrhythmic behaviors that are either motor (related to movement) or vocal and that appear out of context— for example, knee bends in science class. They are fairly common in childhood; in the vast majority of cases, they are temporary conditions that resolve on their own. In some children, however, the tics persist over time, becoming more complex and severe.

Tics may be simple (using only a few muscles or simple sounds) or complex (using many muscle groups or full words and sentences). Simple motor tics are brief, meaningless movements like eye blinking, facial grimacing, head jerks or shoulder shrugs. They usually last less than one second. Complex motor tics involve slower, longer, and more purposeful movements like sustained looks, facial gestures, biting, banging, whirling or twisting around, or copropraxia (obscene gestures).

Simple phonic tics are meaningless sounds or noises like throat clearing, coughing, sniffling, barking, or hissing. Complex phonic tics include syllables, words, phrases, and such statements as "Shut up!" or "Now you've done it!" The child's speech may be abnormal, with unusual rhythms, tones, accents or intensities. The echo phenomenon is a tic characterized by the immediate repetition of one's own or another's words. Coprolalia is a tic made up of obscene, inappropriate or aggressive words and statements. It occurs in fewer than 10% of people with tic disorders.

Children under the age of 10 with simple tics find them to be difficult to suppress, or control. Many older patients and children with complex tics describe feeling strong sensory urges in their joints, muscles and bones that are relieved by the performance of a motor tic in that particular body part. These patients also report inner conflict over whether and when to yield to these urges. A sensation of relief and reduction of anxiety frequently follows the performance of a tic. Unless the tic disorder is very severe, most people with tics can suppress them for varying periods of time.

Motor and vocal tics may be worsened by anxiety, **stress**, boredom, **fatigue**, or excitement. Some people have reported that tics are intensified by premenstrual syndrome, additives in food, and stimulants. The symptoms of tic disorders may be lessened while the patient is asleep. Cannabis (marijuana), alcohol, relaxation, playing a sport, or concentrating on an enjoyable task are also reported to reduce the severity and frequency of symptoms.

Tics are the core symptom shared by transient tic disorder, chronic motor or vocal tic disorder, and Tourette's disorder. It is the severity and course that distinguishes these disorders from one another. The age of onset for these disorders is between two and 15 years. In 75% of Tourette's disorder patients, the symptoms appear by age 11.

Causes and symptoms

Causes

Emotional factors were once viewed as the cause of tics, but this explanation has been largely discounted. The search for causes now focuses on biological, chemical and environmental factors. As of 2002, however, no definitive cause of tics has been discovered.

There appear to be both functional and structural abnormalities in the brains of people with tic disorders. While the exact neurochemical cause is unknown, it is believed that abnormal **neurotransmitters** (chemical messengers within the **brain**) contribute to the disorders. The affected neurotransmitters are dopamine, serotonin, and cyclic AMP. Researchers have also found changes within the brain itself, specifically in the basal ganglia (an area of the brain concerned with movement) and the anterior cingulate cortex. Functional imaging using **positron emission tomography** (PET) and **single photon emission computerized tomography** (SPECT) has highlighted abnormal patterns of blood flow and metabolism in the basal ganglia, thalamus, and frontal and temporal cortical areas of the brain. [The reader may wish to consult the "Brain" entry for a diagram of the brain's structures.]

Vulnerability to tic disorders appears to be genetic, or transmitted within families. Genetic factors are present in 75% of cases, although no single gene has been found to cause tic disorders. Researchers have not found a pattern suggesting that certain types of parenting or childhood experiences lead to the development of tic disorders, although some think that there is an interaction between genetic and environmental factors. Researchers are paying close attention to prenatal factors, which are thought to influence the development of the disorders.

In some cases, tic disorders appear to be caused or worsened by recreational drugs or prescription medications. The drugs most commonly involved are such psychomotor stimulants as **methylphenidate** (Ritalin); **pemoline** (Cylert); **amphetamines**; and cocaine. It is not clear whether tics would have developed anyway if stimulants had not been used. In a smaller percentage of cases, antihistamines, tricyclic antidepressants, antiseizure medications, and opioids have been shown to worsen tics.

Some forms of tic may be triggered by the environment. A cough that began during an upper respiratory infection may continue as an involuntary vocal tic. New tics may also begin as imitations of normally occurring events, such as mimicking a dog barking. How these particular triggers come to form enduring symptoms is a matter for further study.

In some cases, neuropsychiatric disorders, such as tic disorders and **obsessive-compulsive disorder**, have been shown to develop after streptococcal infection. No precise mechanism for this connection has been determined, although it appears to be related to the autoimmune system. There are other illness-related causes of tics, though they appear to be rare. These include the development of tics after head trauma, viral encephalitis or **stroke**.

Symptoms

The diagnostic criteria of all tic disorders specify that the symptoms must appear before the age of 18, and that they cannot result from ingestion of such substances as stimulants, or from such general medical conditions as Huntington's disease. Tic disorders can be seen as occurring along a continuum of least to most severe in terms of disruption and impairment, with transient tic disorder at one end and Tourette's disorder at the other.

Tics increase in frequency when a person is under any form of mental or physical stress, even if it is of a positive nature (excitement about an upcoming holiday, for example). Some people's tics are most obvious when the person is in a relaxed situation, such as quietly watching television. Tics tend to diminish when the person is placed in a new or highly structured situation, such as a doctor's office— a factor that can complicate **diagnosis**. When the symptoms of a tic are present over long time periods, they do not remain constant but will wax and wane in their severity.

Transient tic disorder occurs in approximately 4%–24% of schoolchildren. It is the mildest form of tic disorder, and may be underreported because of its temporary nature. In transient tic disorder, there may be single or multiple motor and/or vocal tics that occur many times a day nearly every day for at least four weeks, but not for longer than one year. If the criteria have been met at one time for Tourette's disorder or for chronic motor or vocal tic disorder, transient tic disorder may not be diagnosed.

Chronic motor or vocal tic disorder is characterized by either motor tics or vocal tics, but not both. The tics occur many times a day nearly every day, or intermittently for a period of more than one year. During that

KEY TERMS

Atypical antipsychotics—A group of newer medications for the treatment of psychotic symptoms that were introduced in the 1990s. The atypical antipsychotics include clozapine, risperidone, quetiapine, ziprasidone, and olanzapine. They are sometimes called serotonin dopamine antagonists, or SDAs.

Basal ganglia—A group of masses of gray matter located in the cerebral hemispheres of the brain that control movement as well as some aspects of emotion and cognition.

Behavioral therapy—An approach to treatment that focuses on extinguishing undesirable behavior and replacing it with desired behavior.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Cognitive-behavioral therapy—An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

Comorbidity—Association or presence of two or more mental disorders in the same patient. A disorder that is said to have a high degree of comorbidity is likely to occur in patients diagnosed with other disorders that may share or reinforce some of its symptoms.

Compulsion—A strong impulse to perform an act, particularly one that is irrational or contrary to one's will.

Coprolalia—A vocal tic characterized by uttering obscene, hostile, or inappropriate words. A motor tic characterized by obscene gestures is called copropraxia.

Cyclic AMP—A small molecule of adenosine monophosphate (AMP) that activates enzymes and increases the effects of hormones and other neurotransmitters.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Holistic—A treatment approach that is comprehensive and respectful of a person's emotional, social, cognitive, and interpersonal needs.

Neuroleptic—Another name for the older antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Obsession—A persistent image, idea, or desire that dominates a person's thoughts or feelings.

Onset—The point in time at which the symptoms of a disorder first became apparent.

Phenothiazines—A class of drugs widely used in the treatment of psychosis.

Remission—In the course of an illness or disorder, a period of time when symptoms are absent.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Stigma—A mark or characteristic trait of a disease or defect; by extension, a cause for reproach or a stain on one's reputation. Tic disorders are sometimes regarded as a stigma by the patient's family.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

Thalamus—The middle part of the diencephalon (a part of the human forebrain), responsible for transmitting and integrating information from the senses.

Tic—A sudden involuntary behavior that is difficult or impossible for the person to suppress. Tics may be either motor (related to movement) or vocal, and may become more pronounced under stress.

time, the patient is never without symptoms for more than three consecutive months. The severity of the symptoms and functional impairment is usually much less than for patients with Tourette's disorder.

For a diagnosis of Tourette's disorder, a patient must have experienced both multiple motor and one or more vocal tics at some time during the illness, though they do not have to occur at the same time. The tics occur many

times a day, usually in bouts, nearly every day or intermittently for a period of more than one year. The patient is never symptom-free for more than three months at a time.

Children and adolescents with Tourette's disorder frequently experience additional problems including aggressiveness, self-harming behaviors, emotional immaturity, social withdrawal, physical complaints, conduct disorders, affective disorders, anxiety, panic attacks, **stuttering**, **sleep disorders**, migraine headaches, and inappropriate sexual behaviors.

Tics seem to worsen during the patient's adolescence, although some clinicians think that the symptoms become more problematic rather than more severe, because the patient experiences them as more embarrassing than previously. The symptoms do become more unpredictable from day to day during adolescence. Many teenagers may refuse to go to school when their tics are severe. Coprolalia often appears first in adolescence; this symptom causes considerable distress for individuals and their families.

Behavioral problems also become more prominent in adolescence. There is some evidence that temper tantrums, aggressiveness, and explosive behavior appear in preadolescence, intensify in adolescence, and gradually diminish by early adulthood. Interestingly, aggression appears to increase at approximately the same time that the tics decrease in severity.

Demographics

Tourette's disorder is three to four times more common in males than females. Tic disorders have been reported in people of all races, ethnic groups, and socioeconomic classes. Tic disorders appear to occur more frequently in Caucasians than African Americans.

Diagnosis

There are no diagnostic laboratory tests to screen for tic disorders. Except for the tics, the results of the patient's physical and neurological examinations are normal. The doctor takes a complete medical history including a detailed account of prenatal events, birth history, head injuries, episodes of encephalitis or meningitis, poisonings, and medication or drug use. The patient's developmental, behavioral, and academic histories are also important.

There is an average delay of five to 12 years between the initial symptoms of a tic disorder and the correct diagnosis. This delay is largely related to the misperception that tics are caused by anxiety and should be treated by **psychotherapy**. This misperception in turn is fueled by the fact that tics tend to increase in severity when the affected person is angry, anxious, excited or fatigued. It is also common for the patient to manifest fewer tics in a doctor's office than at home, leaving parents feeling frustrated and undermined and physicians confused. In addition, children quickly learn to mask their symptoms by converting them to more socially acceptable movements and sounds. The diagnosis of a tic disorder can be aided in some cases by directly observing, videotaping or audiotaping the patient in a more natural setting.

Clinicians can also become confused by such additional symptoms of tic disorders as touching, hitting, jumping, smelling hands or objects, stomping, twirling and doing deep knee bends. They disagree, however, as to whether such symptoms should be classified as tics or compulsions. There appears to be a significant overlap between the symptoms of tic disorders and those of obsessive-compulsive disorder (OCD).

Abnormal obsessive-compulsive behavior has been found in 40% of patients with Tourette's disorder between the ages of six and 10 years. Obsessions are persistent ideas, thoughts, impulses, or images that are experienced as intrusive, inappropriate, senseless, and repetitive. Compulsions are defined as repetitive behaviors performed to reduce the anxiety or distress caused by the obsessions. For those diagnosed with OCD, common obsessions have to do with dirt, germs, and contamination. Patients with Tourette's disorder often have obsessions that involve violent scenes, sexual thoughts, and counting; their compulsions are often related to symmetry (lining things up and getting them "just right," for example). OCD symptoms occur considerably later than tics, and appear to worsen with age. Some theorists have suggested that obsessive thoughts are cognitive tics.

Tic disorders can be differentiated from **movement disorders** by the following characteristics: they are suppressible; they tend to persist during sleep; they are preceded by sensory symptoms; they have both phonic and motor components; and they wax and wane.

Dual diagnoses

Children and adults with tic disorders are at increased risk for depression and other mood disorders, as well as anxiety disorders. This comorbidity may be due to the burden of dealing with a chronic, disruptive, and often stigmatizing disorder. The energy and watchfulness required to suppress tic symptoms may contribute to social anxiety, social withdrawal, self-preoccupation, and fatigue. Low self-esteem and feelings of hopelessness are common in patients diagnosed with tic disorders.

While OCD behaviors have been noted in as many as 80% of individuals with tic disorders, only 30% meet the full criteria for OCD. Distinguishing complex tics

from simple compulsions can be difficult. Touching compulsions appear to be characteristic of the tic-related type of OCD. Compared to obsessive-compulsive disorder in persons without a history of tics, there will likely be an earlier age of onset, a greater proportion of males, a more frequent family history of chronic tics, and a poorer therapeutic response to selective serotonin reuptake inhibitors (SSRIs)— although the addition of a neuroleptic to the treatment regimen sometimes brings about improvement.

As many as 50%–80% of children with Tourette's disorder have some symptoms of **attention-deficit/hyperactivity disorder** (ADHD), including a short attention span, restlessness, poor concentration, and diminished impulse control. On average, ADHD will manifest two and a half years before the tics appear. A **dual diagnosis** of ADHD and tic disorder is associated with more severe tics and greater social impairment than for tic disorder by itself. Over time, the problems caused by the inattention, impulsivity, motor overactivity and the resultant underachievement in school associated with ADHD are often more disabling than the tics themselves.

Children with tic disorders are five times as likely as other children to require special education programs. The tics may be disruptive and mistakenly interpreted by teachers as intended to disturb the class. Often, children with tic disorders have underlying learning disabilities as well. While there does not appear to be any impairment in general intellectual functioning, researchers have identified patterns of specific learning problems in children with tic disorders. These problems include abnormal visual-perceptual performance, reduced visual-motor skills, and discrepancies between verbal and performance IQ. Many of these learning difficulties are also commonly found in children with ADHD.

Increasing numbers of children with tic disorders are also diagnosed with a **conduct disorder**. Children with conduct disorder show inappropriate and sometimes severe aggression toward people and animals. They may also act out other destructive impulses. Unfortunately, some of these children grow up to develop a personality disorder.

Treatments

A holistic approach is recommended for the treatment of tic disorders. A multidisciplinary team should work together with the affected child's parents and teachers to put together a comprehensive treatment plan. Treatment should include the following:

- Educating the patient and family about the course of the disorder in a reassuring manner.
- Completion of necessary diagnostic tests, including self-reports (by child and parents); clinician-administered ratings; and direct observational methods.
- Comprehensive assessment, including the child's cognitive abilities, perception, motor skills, behavior and adaptive functioning.
- Collaboration with school personnel to create a learning environment conducive to academic success.
- Therapy, most often behavioral or cognitive-behavioral, though other modalities may be appropriate.
- If necessary, evaluation for medication.

Behavioral and cognitive-behavioral therapy

Massed negative practice has been one of the most frequently used behavioral therapy techniques in the treatment of children with tic disorder. The patient is asked to deliberately perform the tic movement for specified periods of time interspersed with brief periods of rest. Patients have shown some decrease in tic frequency, but the long-term benefits of massed negative practice are unclear.

Contingency management is another behavioral treatment. It is based on positive **reinforcement**, usually administered by parents. Children are praised and rewarded for not performing tics and for replacing them with alternative behaviors. Contingency management, however, appears to be of limited use outside of such controlled settings as schools or institutions.

Self-monitoring consists of having the patient record tics by using a wrist counter or small notebook. It is fairly effective in reducing some tics by increasing the child's awareness.

Habit reversal is the most commonly used technique, combining relaxation exercises, awareness training, and contingency management for positive reinforcement. This method shows a 64%–100% success rate.

Adding a cognitive component to habit reversal involves the introduction of flexibility into rigid thinking, and confronting the child's irrational expectations and unrealistic anticipations. It has not been shown as of 2002 to increase treatment effectiveness. The specific cognitive technique of distraction, however, has been shown to help patients resist sensory urges and to restore the patient's sense of control over the tic.

Medications

Medication is the main treatment for motor and vocal tics. Patients and their families, however, should be

evaluated fully and use other treatment methods in conjunction with medication. Because the symptoms of tic disorders overlap those of OCD and ADHD, it is essential to determine which symptoms are causing the greatest concern and impairment, and treat the patient according to the single diagnostic category that best fits him or her, whether it is a tic disorder, OCD, or ADHD.

Medications prescribed for patients with tic disorders include:

- Typical neuroleptics (antipsychotic medications), including **haloperidol** (Haldol) and **pimozide** (Orap). Neuroleptics can have significant side effects, which include concentration problems, cognitive blunting, and rarely, **tardive dyskinesia** (a movement disorder that consists of lip, mouth, and tongue movements). Such side effects as stiffness, rigidity, tremor, sedation, and depression are common with haloperidol, but are less so with pimozide.
- Alpha-adrenergic receptor agonists, including **clonidine** (Catapres) and guanfacine (Tenex). Clonidine has fewer and milder side effects than the neuroleptics in general, with the most common being sedation. Sedation occurs in 10%–20% of cases and can often be controlled through adjusting the dosage.
- The phenothiazines may be used when haloperidol or pimozide has proven ineffective.
- Atypical antipsychotics and other agents that block dopamine receptors include **risperidone** (Risperdal) and **clozapine** (Clozaril).
- Tetrabenazine is a promising new medication with fewer side effects than other typical neuroleptics. It can be used in combination with the older antipsychotic medications, allowing for lower doses of both medications with substantial relief.
- Selective serotonin reuptake inhibitors (SSRIs), which include such medications as **fluoxetine** (Prozac) and **sertraline** (Zoloft), can be used to treat the obsessive-compulsive behaviors associated with Tourette's disorder. They can also be helpful with depression and impulse control difficulties, though they must be given at higher dosages for OCD than for depression. The SSRIs, however, can cause gastric upset and nausea.
- Benzodiazepines are used in some cases to lower the patient's anxiety level, but are often avoided because they can cause dependence and tolerance.
- Nicotine chewing gum appears to reduce tics when added to ongoing treatment with haloperidol, but is in need of further study.

Alternative therapies

There is growing interest in dietary changes and nutritional supplements to prevent and manage the symptoms of tic disorders, although formal studies have not yet been conducted in this area. Some theorists have suggested that hidden food and chemical allergies or nutritional deficiencies may influence the development and maintenance of tic disorders. Recommendations include eating organic food and avoiding pesticides; taking antioxidants; increasing intake of folic acid and the B vitamins; eating foods high in zinc and magnesium; eliminating caffeine from the diet; and avoiding artificial sweeteners, colors and dyes.

Prognosis

There is presently no cure for tic disorders, and there is no evidence that early treatment alters prognosis. When a child is first evaluated, it is not possible to determine whether the tics will be chronic or transient, mild or severe.

As recently as twenty years ago, tic disorders were considered to be lifelong conditions, with remissions believed to be rare. There is now a general consensus that if a tic disorder is the only diagnosis, the prognosis is favorable. Up to 73% of patients report that their tics decreased markedly or disappeared as they entered the later years of adolescence or early adulthood.

In a small number of patients, the most severe and debilitating forms of a tic disorder occur in adult life. In addition, stress in later life can cause tics to re-emerge. In rare cases, the tics may be new developments in adulthood, though this phenomenon may be more common than previously thought. Remission rates for tic disorders are difficult to pinpoint among this seldom-studied population, but appear to be extremely low.

While the tics themselves may decline, however, the associated problems often continue into adult life. Obsessive-compulsive symptoms and other behavioral problems, as well as learning disabilities, may grow worse. Obsessive-compulsive behaviors become most pronounced at age 15 and remain at that level. Panic attacks, depression, **agoraphobia** and alcoholism are most significant in the early adult years, while a tendency toward **obesity** increases steadily with age, particularly in women.

In adulthood, a patient's repertoire of tics is reduced and becomes predictable during periods of fatigue and heightened emotionality. Some studies suggest remission rates, with the complete cessation of symptoms, to be as high as 50%. Cases of total remission appear to be relat-

ed to the family's treatment of the patient when he or she was a child. Persons who were punished, misunderstood and stigmatized experience greater functional impairment as adults than those who were supported and understood as children.

Prevention

There are few preventive strategies for tic disorders. There is some evidence that maternal emotional stress during pregnancy and severe nausea and vomiting during the first trimester may affect tic severity. Attempting to minimize prenatal stress may possibly serve a limited preventive function.

Similarly, because people with tic disorders are sensitive to stress, attempting to maintain a low-stress environment can help minimize the number or severity of tics (reducing the number of social gatherings, which can be anxiety-provoking, for example). This approach cannot prevent tics altogether, and must be undertaken with an awareness that it is neither healthful nor advisable to attempt to eliminate all stressful events in life.

See also Abnormal Involuntary Movement Scale; Neuropsychological testing; Stereotypic movement disorder

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OTHER

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Tofranil *see* **Imipramine**

Token economy system

Definition

A token economy is a form of **behavior modification** designed to increase desirable behavior and decrease undesirable behavior with the use of tokens. Individuals receive tokens immediately after displaying desirable behavior. The tokens are collected and later exchanged for a meaningful object or privilege.

Purpose

The primary goal of a token economy is to increase desirable behavior and decrease undesirable behavior. Often token economies are used in institutional settings (such as psychiatric hospitals or correctional facilities) to manage the behavior of individuals who may be aggressive or unpredictable. However, the larger goal of token economies is to teach appropriate behavior and social skills that can be used in one's natural environment. Special education (for children with developmental or learning disabilities, hyperactivity, attention deficit, or behavioral disorders), regular education, colleges, various types of **group homes**, military divi-

sions, nursing homes, **addiction** treatment programs, occupational settings, family homes (for marital or parenting difficulties), and hospitals may also use token economies. Token economies can be used individually or in groups.

Description

Several elements are necessary in every token economy:

- Tokens: Anything that is visible and countable can be used as a token. Tokens should preferably be attractive, easy to carry and dispense, and difficult to counterfeit. Commonly used items include poker chips, stickers, point tallies, or play money. When an individual displays desirable behavior, he or she is immediately given a designated number of tokens. Tokens have no value of their own. They are collected and later exchanged for meaningful objects, privileges or activities. Individuals can also lose tokens (response cost) for displaying undesirable behavior.
- A clearly defined target behavior: Individuals participating in a token economy need to know exactly what they must do in order to receive tokens. Desirable and undesirable behavior is explained ahead of time in simple, specific terms. The number of tokens awarded or lost for each particular behavior is also specified.
- Back-up reinforcers: Back-up reinforcers are the meaningful objects, privileges, or activities that individuals receive in exchange for their tokens. Examples include food items, toys, extra free time, or outings. The success of a token economy depends on the appeal of the back-up reinforcers. Individuals will only be motivated to earn tokens if they anticipate the future reward represented by the tokens. A well-designed token economy will use back-up reinforcers chosen by individuals in treatment rather than by staff.
- A system for exchanging tokens: A time and place for purchasing back-up reinforcers is necessary. The token value of each back-up reinforcer is pre-determined based on monetary value, demand, or therapeutic value. For example, if the reinforcer is expensive or highly attractive, the token value should be higher. If possession of or participation in the reinforcer would aid in the individual's acquisition of skills, the token value should be lower. If the token value is set too low, individuals will be less motivated to earn tokens. Conversely, if the value is set too high, individuals may become easily discouraged. It is important that each individual can earn at least some tokens.

KEY TERMS

Back-up reinforcer—A desirable item, privilege, or activity that is purchased with tokens and serves as a delayed reward and subsequent motivation for target (desired) behavior.

Baseline data—Information regarding the frequency and severity of behavior, gathered before treatment begins.

Behavior modification—An approach to therapy based on the principles of operant conditioning. Behavior modification seeks to replace undesirable behaviors with preferable behaviors through the use of positive or negative reinforcement.

Fading—Gradually decreasing the amount or frequency of a reinforcer so that the target behavior will begin to occur independent of any rewards.

Reinforcement schedule—The frequency and amount of reinforcers administered.

Reinforcer—Anything that causes an increase of a particular behavior.

Response cost—A behavioral technique that involves removing a stimulus from an individual's environment so that the response that directly precedes the removal is weakened. In a token economy system, response cost is a form of punishment involving loss of tokens due to inappropriate behavior, which consequently results in decreased ability to purchase back-up reinforcers.

Target behavior—The specific behavior to be increased or decreased during treatment.

Therapeutic value—The potential benefit of an object or situation, in terms of its ability to enhance functioning (social, emotional, intellectual, occupational, etc.) in an individual.

Token—Any item that can be seen and collected (such as stickers or points in a point tally) that has no value of its own, but is used as an immediate reward for desirable behavior that is later exchanged for back-up reinforcers.

- A system for recording data: Before treatment begins, information (baseline data) is gathered about each individual's current behavior. Changes in behavior are then recorded on daily data sheets. This information is used to measure individual progress, as well as the effectiveness of the token economy. Information regarding the exchange of tokens also needs to be recorded.

- Consistent implementation of the token economy by staff: In order for a token economy to succeed, all involved staff members must reward the same behaviors, use the appropriate amount of tokens, avoid dispensing back-up reinforcers for free, and prevent tokens from being counterfeited, stolen, or otherwise unjustly obtained. Staff responsibilities and the rules of the token economy should be described in a written manual. Staff members should also be evaluated periodically and given the opportunity to raise questions or concerns.

Initially tokens are awarded frequently and in higher amounts, but as individuals learn the desirable behavior, opportunities to earn tokens decrease. (The amount and frequency of token dispensing is called a **reinforcement** schedule.) For example, in a classroom, each student may earn 25 to 75 tokens the first day, so that they quickly learn the value of the tokens. Later, students may earn 15 to 30 tokens per day. By gradually decreasing the availability of tokens (fading), students should learn to display the desirable behavior independently, without the unnatural use of tokens. Reinforcers that individuals would normally encounter in society, such as verbal praise, should accompany the awarding of tokens to aid in the fading process.

Advantages of token economies are that behaviors can be rewarded immediately, rewards are the same for all members of a group, use of punishment (response cost) is less restrictive than other forms of punishment, and individuals can learn skills related to planning for the future. Disadvantages include considerable cost, effort, and extensive staff training and management. Some professionals find token economies to be time-consuming and impractical.

Risks

Risks involved in token economies are similar to those in other forms of behavior modification. Staff members implementing the therapy may intentionally or unintentionally neglect the rights of individuals receiving treatment. Token economies should never deprive individuals of their basic needs, such as sufficient food, comfortable bedding, or reasonable opportunities for leisure. If staff members are inadequately trained or there is a shortage of staff, desirable behaviors may not be rewarded or undesirable behaviors may be inadvertently rewarded, resulting in an increase of negative behavior. Controversy exists regarding placing individuals in treatment against their will (such as in a psychiatric hospital), and deciding which behaviors should be considered desirable and which should be considered undesirable.

Normal results

Ideally, individuals will use the skills learned in a token economy in their everyday surroundings. They will display the undesirable behavior less frequently or not at all. They will also engage in positive, adaptive behaviors more often.

Abnormal results

If the token economy was ineffective, or time spent in the token economy was limited, individuals may show no changes or increases in the undesirable behavior.

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Cambridge Center for Behavioral Studies. 336 Baker Avenue, Concord, Massachusetts 01742-2107. (978) 369-2227. <www.behavior.org>.

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Tourette's disorder *see* **Tic disorders**

Tranquilizers *see* **Anti-anxiety drugs and abuse**

Transient tic disorder *see* **Tic disorders**

Transsexualism *see* **Gender identity disorder**

Transvestic fetishism

Definition

Transvestic **fetishism** is defined by the mental health professional's handbook, the ***Diagnostic and Statistical Manual of Mental Disorders***, fourth edition, text revision (2000), which is also called *DSM-IV-TR*, as one of the **paraphilias**. The paraphilias are a group of mental disorders characterized by **obsession** with unusual sexual practices or with sexual activity involving nonconsenting or inappropriate partners (such as children or animals). The essential feature of transvestic fetishism is recurrent intense sexual urges and sexually arousing fantasies involving dressing in clothing associated with members of the opposite sex. Another term for tranvestic fetishism is cross-dressing; people who frequently engage in cross-dressing are sometimes called transvestites. A **diagnosis** of transvestic fetishism is made only if an individual has acted on these urges or is markedly distressed by them. In other systems of psychiatric classification, transvestic fetishism is considered a sexual deviation.

For some people who are diagnosed with transvestic fetishism, fantasies or stimuli associated with cross-dressing may always be necessary for erotic arousal and are always included in sexual activity, if not actually acted out alone or with a partner. In other patients, cross-dressing may occur only episodically, for example, during periods of **stress**. At other times the person is able to function sexually without the transvestic fetish or related stimuli.

Description

A person with a transvestic fetish derives sexual gratification from dressing in clothing appropriate for a member of the opposite sex. Almost all patients diagnosed with transvestic fetishism, however, are men dressing as women. This lopsided gender ratio may be partly due to the fact that contemporary Western societies allow women to dress in a wide range of clothing styles influenced by menswear, whereas the reverse is not the case. While it is not at all unusual to see women wearing jeans, tailored trousers, Western-style boots, or even tuxedos in some circumstances, men wearing dresses or high-heeled shoes look distinctly out of place.

A person's participation in transvestism is usually gradual. Over time, a person with a transvestic fetish assumes the role and appearance of a member of the opposite gender. It is important to note that this activity is closely associated with achieving sexual gratification. Persons who have had extensive experience with a transvestic fetish may be difficult to distinguish from members of the opposite sex. A so-called mature transvestic fetish involves adopting all of the mannerisms, clothing, materials and other items associated with persons of the opposite sex.

KEY TERMS

Cross-dressing—Wearing clothing and other attire appropriate to the opposite sex.

Fetishism—A paraphilia in which a person requires a nonliving object (or occasionally a non-genital part of the body, such as the partner's feet) in order to achieve sexual arousal and satisfaction.

Gender dysphoria—A state of persistent discomfort or depression associated with one's gender role or biological sex.

Paraphilias—A group of mental disorders that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Transvestite—A person who derives sexual pleasure or gratification from dressing in clothing of the opposite sex.

Causes and symptoms

Causes

The basis for a transvestic fetish is obtaining sexual gratification by dressing in clothing appropriate for the opposite sex. The cause may be adolescent curiosity. A person with a transvestic fetish may not be aware of its roots. Transvestic fetishism sometimes begins when a young boy dresses up in the clothes of an older sister or his mother. The activity is continued because it is enjoyable but the reasons for the enjoyment remain unconscious. In other cases a boy's mother may initiate the cross-dressing by dressing him as if he were a girl. This behavior is sometimes related to the mother's anger at men or to a preference for having daughters rather than sons.

Persons with transvestic fetishes should not be assumed to be homosexual. According to *DSM-IV-TR*, most men who practice cross-dressing are basically heterosexual in their orientation. Some, however, have occasional sexual encounters with other men.

Symptoms

Early symptoms of transvestic fetishism involve touching or wearing items of clothing that are considered

typically feminine. This initial interest may progress to wearing undergarments or other items that can be hidden from the view of others while providing arousal to the wearer. Over time, the extent of dressing in women's clothing expands, sometimes to the point of dressing as a woman on a regular basis. A developed transvestic fetish often involves feminine hair styling and the use of women's cosmetics and accessories.

In some persons diagnosed with transvestic fetishism, the motivation for cross-dressing may change over time from a search for sexual excitement to simple relief from stress, depression, or anxiety.

In some cases, persons with a transvestic fetish discover that they are unhappy with their biological sex, a condition known as gender dysphoria. They may elect to have hormonal and surgical procedures to change their bodies. Some may choose to have gender reassignment surgery. The incidence of gender dysphoria and subsequent gender reassignment among persons diagnosed with transvestic fetishism is not known.

Demographics

Except for **sexual masochism**, in which the gender ratio is estimated to be 20 males for each female, paraphilias such as transvestic fetishism are practically never diagnosed in females, although a few cases have been reported. Virtually no information is available on family patterns of the disorder.

Diagnosis

Persons with transvestic fetishism may or may not seek **psychotherapy** on their own account. In some instances, the patient has agreed to consult a **psychiatrist** because his wife or girlfriend is distressed by the cross-dressing. The actual diagnosis of transvestic fetishism is most commonly made by taking a history or by direct observation. The diagnosis is made only if the patient has been markedly distressed by inability to dress in such a manner or if the disorder is interfering with his education, occupation, or social life. Dressing in women's clothing for such occasions as Halloween or a costume party is not sufficient for a diagnosis of transvestic fetishism.

Treatments

In the earliest period of behavior therapy, transvestic fetishes were narrowly viewed as inappropriate behavior that was confined to a limited range of situations, and were sometimes treated with **aversion therapy**, usually with electric shocks. This approach was largely unsuccessful. Persons with fetishes have also been treated by using a form of behavioral therapy known as orgasmic reorientation, which attempts to help people learn to respond sexually to culturally appropriate stimuli. This treatment also has had limited success.

Most persons who have a transvestic fetish never seek treatment from professionals. Most are capable of achieving sexual gratification in culturally appropriate situations. Their preoccupation with cross-dressing is viewed as essentially harmless to other persons, since transvestism is not associated with criminal activities or forcing one's sexual preferences on others. As of 2002, American society has developed tolerance for transvestites, thus further reducing the demand for professional treatment.

Prognosis

The prognosis for treatment of transvestic fetishism is poor, as most persons with this disorder do not desire to change. Most cases in which treatment was demanded by a spouse as a condition of continuing in a marriage have not been successful. The prognosis for personal adjustment is good, however, as a person with a transvestic fetish and his related activities do not usually disturb others.

Prevention

Most experts agree that providing gender-appropriate guidance in a culturally appropriate situation will prevent the formation of a transvestic fetish. The origin of some cases of transvestism may be a random association between clothing inappropriate for one's own gender and sexual gratification. There is no reliable way to predict the formation of such associations. Supervision during childhood and adolescence, combined with acceptance of a child's biological sex, may be the best deterrent that parents can provide.

See also Aversion therapy; Gender identity disorder; Gender issues in mental health

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org>.

American Academy of Pediatrics. 141 Northwest Point Boulevard, Elk Grove Village, IL 60007-1098. Telephone: (847) 434-4000. Fax: (847) 434-8000. Web site: <http://www.aap.org/default.htm>.

American College of Physicians. 190 N Independence Mall West, Philadelphia, PA 19106-1572. Telephone: (800) 523-1546, x 2600 or (215) 351-2600. Web site: <http://www.acponline.org>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850.

American Psychological Association. 750 First Street NW, Washington, DC, 20002-4242. Telephone: (800) 374-2721 or (202) 336-5500. Web site: <http://www.apa.org>.

American Public Health Association. 800 I Street, NW, Washington, DC 20001-3710. Telephone: (202) 777-2742. Fax: (202) 777-2534. Web site: <http://www.apha.org>.

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Tranxene *see* **Clorazepate**

Tranylcypromine

Definition

Tranylcypromine is classified as a monoamine oxidase (MAO) inhibitor. It is used to treat serious depression. In the United States, tranylcypromine is sold under the brand name Parnate. As of 2002, there were no generic forms of tranylcypromine available in the United States.

KEY TERMS

Agoraphobia—People with this condition worry that they will not be able to get help or flee a place if they have a panic attack; or refusal to go to places that might trigger a panic attack.

Bulimia—An eating disorder characterized by binges in which large amounts of food are consumed, followed by forced vomiting.

Congestive heart failure—Condition characterized by abdominal pain, swelling in the lower extremities, and weakness caused by a reduced output of blood from the left side of the heart.

Diabetes mellitus—A chronic disease affecting the metabolism of carbohydrates that is caused by insufficient production of insulin in the body.

Panic disorder—An anxiety disorder in which an individual experiences sudden, debilitating attacks of intense fear.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tyramine—Intermediate product between the chemicals tyrosine and epinephrine in the body and a substance normally found in many foods. Found especially in protein-rich foods that have been aged or fermented, pickled, or bacterially contaminated, such as cheese, beer, yeast, wine, and chicken liver.

Purpose

Tranylcypromine is used primarily to treat depression that does not respond to other types of drug therapy. It is also used occasionally to treat **panic disorder**, **agoraphobia**, and **bulimia nervosa**.

Description

Trancyclopromine is a member of a class of drugs called monoamine oxidase inhibitors. Monoamine oxidase, or MAO, is an enzyme found throughout the body. In the **brain**, MAO breaks down norepinephrine and serotonin, two naturally occurring chemicals that are important for maintaining mental well-being preventing depression. Monoamine oxidase inhibitors, such as

tranylcypromine, reduce the activity of MAO. Less norepinephrine and serotonin are broken down, so their levels rise. This helps to lift depression.

Tranylcypromine is effective for treating depression, especially complicated types of depression that have not responded to more traditional antidepressants. However, tranylcypromine also affects the MAO enzyme in many other areas of the body. This accounts for the large number of serious side effects and drug interactions it causes.

Recommended dosage

The typical starting dosage of tranylcypromine in adults is 10 mg taken twice per day. This dosage is sometimes increased to 30 mg per day after a two-week period. The maximum recommended amount is 60 mg per day. The elderly (over age 60) are usually started on a dose of 2.5 mg per day. After this, their doctor will make an individualized decision about increasing the dosage. Older adults typically take smaller doses and do not take more than 45 mg per day. A doctor must make an individual determination of whether to give tranylcypromine to youths under the age of 18 years, because guidelines for this age group have not been developed.

The benefits of this drug may not become apparent for several weeks. Patients should be aware of this and continue taking the drug as directed, even if they do not see an immediate improvement.

Precautions

People taking tranylcypromine should not eat foods rich in tyramine. These foods include yeast or meat extracts, fermented sausage, overripe fruit, sauerkraut, cheese, and fava beans. Alcohol should not be consumed, and the same holds true for alcohol-free beer and wine. Large amounts of caffeine-containing food and beverages, such as chocolate, tea, coffee, and cola should be avoided. The treating doctor needs to approve the use of any drug, including prescription, over-the-counter drugs, and herbal treatments, that the patient takes while taking tranylcypromine.

Tranylcypromine should be used with great caution in pregnant and nursing women only after the risks and benefits of treatment have been assessed. Likewise, this drug may not be appropriate for people with a history of **seizures**, children under age 18 years, people at risk for **suicide**, those with severe depression, a history of **schizophrenia**, or diabetes mellitus. People with these conditions should discuss the risks and benefits of this drug with their physician, and a decision to treat should be made on an individual basis. People should not stop taking tranylcypromine suddenly. Instead, the dose should be gradually reduced, then discontinued.

People with a history of high blood pressure, congestive heart failure, severe liver disease, severe kidney disease, severe heart disease, and blood vessel problems in the brain should not take tranylcypromine.

Side effects

The enzyme monoamine oxidase regulates functions throughout the body. Phenelzine decreases the activity of monoamine oxidase in all the areas of the body where it exists, not just in the brain. This is why tranylcypromine is capable of causing a wide variety of side effects in many different organ systems.

Tranylcypromine should be stopped if symptoms of unusually high blood pressure develop. These symptoms include severe chest pain, severe headache, nausea, vomiting, stiff or sore neck, enlarged pupils, and significant changes in heart rate. If these symptoms develop, it should be considered an emergency, and the affected person should get medical help immediately. Generally, these serious side effects are rare.

More common but less serious side effects include lightheadedness or dizziness when arising from a sitting position. These symptoms need to be reported to a doctor but are not considered an emergency. Less common symptoms that should be reported include pounding heartbeat, swelling of the lower extremities, nervousness, and diarrhea. Rare but reportable symptoms include fever, skin rash, dark urine, slurred speech, yellowing of the eyes or skin, and staggering when walking. Common but not serious side effects include decreased sexual performance, increased appetite, muscle twitching, trembling, blurred vision, and reduced urine output.

Overdose symptoms include confusion, seizures, severe dizziness, **hallucinations**, severe headache, severe drowsiness, significant changes in blood pressure, difficulty in sleeping, breathing difficulties, and increased irritability.

Interactions

Tranylcypromine interacts with a long list of drugs. Some of these interactions can cause death. This section is not a complete list of interactions, but it includes the most serious ones. Patients must make sure every health care professional who takes care of them (for example, doctors, dentists, podiatrists, optometrists, pharmacists, nurses) knows that they take tranylcypromine, as well as

all of the other prescription, nonprescription, and herbal drugs that they take.

The combination of tranylcypromine with any type of stimulant can increase the risk of developing serious increases in blood pressure. Tranylcypromine when taken with antidiabetic drugs can reduce blood sugar levels to far below normal. The combination of tranylcypromine with **barbiturates** can prolong the effects of the barbiturate drug.

Tranylcypromine should never be combined with other antidepressant drugs, especially the selective serotonin reuptake inhibitors (SSRIs), because of potentially severe or fatal reactions, including increased risk of dangerously high blood pressure. Patients taking tranylcypromine should stop the drug, then wait at least 14 days before starting any other antidepressant. The same holds true when discontinuing another antidepressant and starting tranylcypromine.

Alcohol combined with tranylcypromine can lead to significantly increased blood pressure. Tranylcypromine combined with the blood pressure drug guanethidine (Ismelin) can reduce the beneficial effects of the guanethidine. When tranylcypromine is combined with levodopa (Dopar, Larodopa), a drug used to treat Parkinson disease, severely increased blood pressure can develop. Tranylcypromine combined with lithium can cause fever. Meperidine (Demerol), when combined with tranylcypromine, can cause fever, seizures, increased blood pressure, and agitation. Tranylcypromine combined with norepinephrine can cause increased response to norepinephrine. Tranylcypromine combined with reserpine (Serpalan, Serpasil) can produce greatly increased blood pressure. When tranylcypromine is combined with the migraine drug sumatriptan (Imitrex), significantly increased concentrations of the latter drug develop that can produce potentially toxic effects.

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Trazodone

Definition

Trazodone is an oral antidepressant. It is sold in the United States under the brand name Desyrel and is also available under its generic name.

Purpose

Trazodone is used to treat depression and to treat the combination of symptoms of anxiety and depression. Like most antidepressants, trazodone has also been used in limited numbers of patients to treat **panic disorder**, **obsessive-compulsive disorder**, **attention-deficit/hyperactivity disorder**, **enuresis** (bed-wetting), eating disorders such as **bulimia nervosa**, cocaine dependency, and the depressive phase of bipolar (manic-depressive) disorder. It should be noted, however, that trazodone has not received official approval from the United States Food and Drug Administration (FDA) for these secondary uses.

Description

Trazodone acts to change the balance of naturally occurring chemicals in the **brain** that regulate the transmission of nerve impulses between cells. Its action primarily increases the concentration of norepinephrine and serotonin (both chemicals that stimulate nerve cells) and, to a lesser extent, blocks the action of another brain chemical, acetylcholine. Trazodone is classified as an atypical antidepressant, but it shares many of the properties of tricyclic antidepressants (**amitriptyline**, **clomipramine**, **desipramine**, **doxepin**, **imipramine**, **nortriptyline**, **protriptyline**, and **trimipramine**). It also shares some of the properties of selective serotonin reuptake inhibitor antidepressants (**fluoxetine**, **paroxetine**, and **sertraline**). Trazodone is the most sedating, and least anticholinergic, of all the currently marketed antidepressants.

The therapeutic effects of trazodone, like other antidepressants, appear slowly. Maximum benefit is often not evident for at least two weeks after starting the drug. People taking trazodone should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Recommended dosage

As with any antidepressant, trazodone must be carefully adjusted by the physician to produce the desired therapeutic effect. Trazodone is available as 50-mg, 100-mg, and 150-mg film-coated tablets that cannot be divid-

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that generally produces effects that are the opposite of those produced by dopamine and norepinephrine. Central nervous system well-being is dependent on a balance between acetylcholine, serotonin, dopamine and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical, acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Benign prostate hypertrophy—Enlargement of the prostate gland.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

ed, and 150-mg and 300-mg oral tablets that can be split. Therapy is usually started at a total of 150 mg per day divided into two or three doses. This dose is increased by 50 mg every three or four days until the desired effects are seen. Daily doses may be increased to a maximum of 400 mg per day in outpatients and up to 600 mg per day in hospitalized patients. In cases of extreme depression, daily doses of up to 800 mg have been used in hospitalized patients. To minimize daytime drowsiness, a major portion of the daily dose can be given at bedtime.

Precautions

The most common problem with trazodone is sedation (drowsiness, lack of mental and physical alertness). This side effect is especially noticeable early in therapy. In most patients, sedation decreases or disappears entirely with time, but until then patients taking trazodone should not perform hazardous activities requiring mental alertness or coordination, including driving and similar activities. The sedative effect is increased when trazodone is taken with other central nervous system depressants, such as alcoholic beverages, sleeping medications, other sedatives, or antihistamines. It may be dangerous to take trazodone in combination with these substances.

Although lower in anticholinergic side effects than tricyclic antidepressants, trazodone should be used cautiously and with close physician supervision in people, especially the elderly, who have benign prostatic hypertrophy, urinary retention, and glaucoma, especially angle-closure glaucoma (the most severe form). Before starting treatment, people with these conditions should discuss the relative risks and benefits of treatment with their doctors to help determine if protriptyline is the right antidepressant for them.

Trazodone may increase heart rate and stress on the heart. It may be dangerous for people with cardiovascular disease, especially those who have recently had a heart attack, to take this drug. In rare cases where patients with cardiovascular disease must take trazodone, they should be monitored closely for cardiac rhythm disturbances and signs of cardiac stress or damage.

Side effects

Trazodone shares side effects common to many antidepressants. The most frequent of these are dry mouth, constipation, and urinary retention, though these are less common than with tricyclic antidepressants. Increased heart rate, sedation, irritability, dizziness, and decreased coordination can also occur. As with most side effects associated with antidepressants, the intensity is highest at the beginning of therapy and tends to decrease with continued use.

Dry mouth, if severe to the point of causing difficulty in speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Patients may also chew sugarless gum or suck on sugarless candy in order to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement who take trazodone may be especially likely to have problems with urinary retention. Symptoms include having difficulty starting a urine flow and more difficulty than usual passing urine. In most cases, urinary retention is managed with dose reduction or by switching to another type of antidepressant. In extreme cases, patients may require treatment with bethanechol, a drug that reverses this particular side effect. In rare cases, trazodone has also been known to cause priapism, a prolonged and painful penile erection. People who think they may be experiencing any side effects from this or any other medication should tell their physicians.

Interactions

Because both trazodone and members of the class of antidepressants known as monoamine oxidase (MAO) inhibitors may increase serotonin levels in the brain, the combination of these drugs can lead to a condition known as serotonin syndrome. Symptoms of serotonin syndrome include a prolonged rapid heart rate, hypertension (high blood pressure), flushing of the skin, **hallucinations**, tremors, and hyperthermia (increased body temperature). Because of this, it can be dangerous to take trazodone in combination with MAO inhibitors such as Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate). The same holds true when combining trazodone with a selective serotonin uptake inhibitor (SSRI) antidepressant such as Prozac (fluoxetine), paroxetine, or sertraline.

Trazodone may increase the blood pressure–lowering effects in patients who are taking antihypertensive medications. Patients who take these drugs together should have their blood pressure monitored regularly so that their antihypertensive medications can be adjusted if their blood pressure becomes too low.

The sedative effects of trazodone are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or medications used for other mental disorders such as **schizophrenia**. The anticholinergic effects of trazodone may be additive with other anticholinergic drugs such as **benztropine**, **biperiden**, **trihexyphenidyl**, and antihistamines.

See also Neurotransmitters

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Triazolam

Definition

Triazolam is a hypnotic drug. It is a member of the benzodiazepine family of drugs. In the United States, it is sold under the brand name Halcion as well as under its generic name.

KEY TERMS

Amnesia—A general medical term for loss of memory that is not due to ordinary forgetfulness. Amnesia can be caused by head injuries, brain disease, or epilepsy as well as by dissociation.

Euphoria—A feeling or state of well-being or elation.

Hypnotic—A type of medication that induces sleep.

Insomnia—A chronic inability to sleep or to remain asleep throughout the night.

Tachycardia—A pulse rate above 100 beats per minute.

Purpose

Triazolam is used for the short-term (generally seven to 10 days) treatment of **insomnia**. Continued usage for more than two to three weeks requires a complete re-evaluation of the person receiving the drug.

Description

Triazolam increases the speed with which people achieve sleep, it increases the duration of sleep, and decreases the likelihood of being awakened during sleep. The effect of triazolam decreases after 14 days of continuous use. Often, sleep patterns return to those experienced prior to beginning use of triazolam or worse. This is called rebound insomnia.

Recommended dosage

The recommended dose of triazolam is 0.25 mg before going to bed. Persons with smaller body masses and older individuals can receive a comparable effect with 0.125 mg of triazolam. The lowest effective dosage of drug should be used to minimize adverse reactions.

Precautions

Because of problems with rebound insomnia, patients should not receive triazolam for more than seven consecutive days. Accompanying rebound insomnia may be daytime anxiety.

Triazolam can cause serious birth defects. Women should not take this medicine if they are pregnant, think they may be pregnant, or are trying to get pregnant.

The drug may cause daytime anxiety after as few as 10 days of continuous usage. If this occurs, triazolam use should be discontinued.

Persons using triazolam should exercise caution when driving or using power tools or machinery.

People who use **temazepam** to reduce jet lag on long flights should be aware of a condition sometimes called "traveler's amnesia." This is a condition where the traveler completes the flight and carries on with normal activities but has no memory of these activities. The period of **amnesia** may last for a few minutes to a few hours. Traveler's amnesia is most common when the traveler has had too little sleep or has been drinking alcohol.

Side effects

Triazolam has relatively few side effects. Those that have been reported include drowsiness, headache, dizziness, nervousness, a feeling of being light-headed, problems with coordination, nausea and vomiting.

Less frequent side effects include euphoria, tachycardia, **fatigue**, confusion, impaired memory, muscle cramping and pain and depression.

Interactions

Triazolam increases the effect of drugs and substances that depress the central nervous system. This class of drugs includes anesthetics, narcotics, sedatives and other sleeping pills, atropine and alcohol.

Some drugs and foods increase the effects of triazolam. They may also increase the chances of having side effects. These include cimetidine, isoniazid, oral contraceptives, and grapefruit juice.

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American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

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Trichotillomania

Definition

Individuals with trichotillomania repetitively pull out their own hair. Trichotillomania as an impulse-control disorder. Some researchers view it as a type of affective or **obsessive-compulsive disorder**. Nail-biting, skin-picking, and thumb-sucking are considered to be related conditions.

Description

Trichotillomania involves hair-pulling episodes that result in noticeable hair loss. Although any area of the body can be a target, the most common areas are the scalp, followed by the eyelashes, eyebrows, and pubic region. Hair-pulling can occur without the individual's awareness, but is frequently preceded by a sense of increasing tension and followed by a sense of relief or

gratification. The resulting hair loss can be a source of embarrassment or shame. Because of a tendency to hide symptoms, and because professionals are relatively unfamiliar with the disorder, individuals either may not seek, or are offered treatment. Untreated trichotillomania can result in impaired social functioning and medical complications.

Causes and symptoms

Causes

Scientific research regarding trichotillomania has been conducted primarily in the past 10 years and causes are only theoretical. Psychoanalytic theories suggest that the behavior is a way of dealing with unconscious conflicts or childhood trauma (such as sexual **abuse**). Biological theories look for a genetic basis. For instance, people with trichotillomania often have a first-degree relative with an obsessive-compulsive spectrum disorder. Researchers are also evaluating similarities between trichotillomania and Tourette's disorder. Behavioral theories assume that symptoms are learned, that a child may imitate a parent who engages in hair-pulling. The behavior may also be learned independently if it serves a purpose. For example, hair-pulling may begin as a response to **stress** and then develop into a habit.

Symptoms

According to the ***Diagnostic and Statistical Manual of Mental Disorders***, (*DSM-IV-TR*), produced by the American Psychiatric Association and used by most mental health professionals in North America and Europe to diagnose mental disorders, the following conditions must be present for a **diagnosis** of trichotillomania:

- noticeable hair loss (alopecia) due to recurrent hair-pulling
- tension immediately before hair-pulling, or when attempting to resist hair-pulling
- reduction of tension, or a feeling of pleasure or gratification, immediately following hair-pulling
- significant distress or impairment in social, occupational, or other important areas of functioning

In addition, the *DSM-IV-TR* requires that hair-pulling not be due to another medical or mental disorder. The tension-release requirement is controversial because 17% of people who otherwise qualify for this diagnosis do not experience this.

Symptoms usually emerge in early adolescence. Episodes may last a few minutes or a few hours during periods of stress or relaxation. Hairs with unique textures or qualities may be preferred. The pulling may include rituals, such as twirling hair off or examining the root. Half of those individuals with trichotillomania engage in oral behaviors—running hair across the lips or through the teeth, biting off the root (trichophagy), or eating hair (trichophagia). The usually try to control their behavior in the presence of others and may hide the affected areas. Symptoms may come and go for weeks, months, or years at a time.

KEY TERMS

Alopecia—Hair loss (also, loss of feathers or wool in animals).

Selective serotonin reuptake inhibitors—Commonly prescribed drugs for treating depression. SSRIs affect the chemicals that nerves in the brain use to send messages to one another. These chemical messengers (neurotransmitters) are released by one nerve cell and taken up by others. Neurotransmitters not taken up by other nerve cells are taken up by the same cells that released them. This process is termed "reuptake". SSRIs work by inhibiting the reuptake of serotonin, an action which allows more serotonin to be taken up by other nerve cells.

Serotonergic—Containing, activating, or otherwise involving serotonin, which is a chemical that aids in the transmission of nerve impulses.

Trichobezoar—A hairball that results from a buildup of swallowed hairs becoming lodged in the digestive system.

Trichophagia—Eating hair.

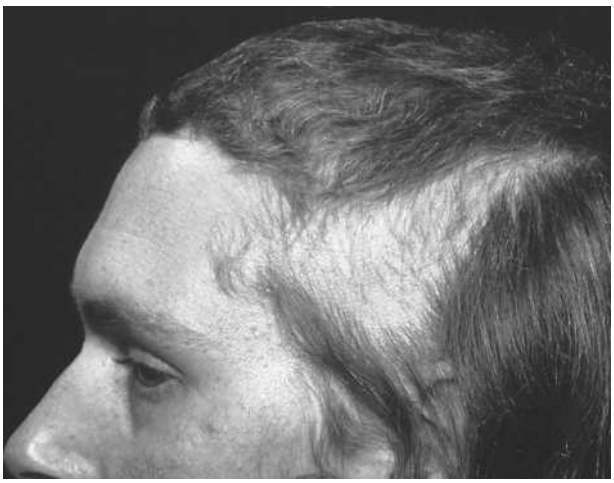
Trichophagy—Biting hair.

Demographics

Once regarded as rare, trichotillomania is now considered more common, affecting 1–4% of people in the general population. When the tension-release requirement is excluded, trichotillomania occurs in adult females (3.4%) more often than adult males (1.5%). Among children, both genders are affected equally.

Diagnosis

Other possible causes of symptoms must first be ruled out. Hair loss may have a medical cause, such as a dermatological condition. Hair-pulling may have another



Left side of a man's scalp showing the effects of trichotillomania. *(Custom Medical Stock Photo. Reproduced by permission.)*

psychological cause, such as a delusion or hallucination in **schizophrenia**.

Severity of symptoms is also important. Twisting or playing with hair when nervous does not qualify as trichotillomania. If symptoms are minor or undetectable, a diagnosis should be given only if the individual expresses significant distress. Children should be given the diagnosis only if symptoms persist because hair-pulling may be a temporary phase, much like thumb-sucking.

If individuals deny symptoms, hair-pulling behavior can be assessed by objective measures such as the presence of short, broken hairs or damaged follicles. Some psychological assessment instruments are also available.

Treatments

Treatment usually starts by determining the current frequency and severity of symptoms. This information, which serves as a measure of progress, is gathered by (a) self-report; (b) reports from significant others; (c) objective measures, such as saving pulled hairs, videotapes, or measuring areas of hair loss; or (d) a combination of these methods.

Primarily, three categories of therapy have been used in the treatment of trichotillomania:

- **Psychoanalysis** focuses on childhood experiences and unresolved conflicts during early development.
- Medications. Those typically used are antidepressants with serotonergic properties (also used with obsessive-compulsive disorders). **Clomipramine** (Anafranil) has proven most effective. The selective serotonin reuptake inhibitors (SSRIs) have had mixed results. Some researchers recommend low doses of antipsychotic drugs (neuroleptics) in conjunction with SSRIs. Medications are usually combined with behavior therapy.
- Behavior therapy. This includes a number of different approaches: Punishment procedures such as electric shock, topical cream to enhance pain, or mittens placed on the person's hands, are effective but controversial. They are intrusive and are often used with individuals who may be unable to consent, such as children or people with developmental disabilities. Habit-reversal training is the most accepted approach. It teaches individuals to monitor their hair-pulling and substitute it for more healthy behaviors. Alternative forms of behavior therapy include **biofeedback** and hypnosis.

Prognosis

The effects of trichotillomania can be very serious: Associated feelings of shame may result in avoidance of social situations; chewing hair can result in dental erosion; eating hair may result in hairballs (trichobezoars) becoming lodged in the stomach or large intestine, which can lead to anemia, abdominal pain, nausea and vomiting, hematemesis (vomiting blood), or bowel obstruction or perforation.

Studies show low success rates with medications and traditional psychoanalysis. Behavioral therapy has reported long-term success rates of 90% or better. Follow-up sessions are encouraged to prevent relapse. A major issue in prognosis is whether an individual receives treatment. Professionals may not recognize or know how to treat trichotillomania effectively. Conversely, individuals with the disorder may be too embarrassed to address their symptoms.

Prevention

Because scientific research is lacking, no specific information is available regarding prevention.

See also Anxiety and anxiety disorders; Cognitive-behavioral therapy; Tic disorders

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Trichotillomania Learning Center, Inc. 303 Potrero #51, Santa Cruz, CA 95060. (831) 457-1004. <http://www.trich.org>.

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Trifluoperazine

Definition

Trifluoperazine is a phenothiazine antipsychotic agent. In the United States, this drug is sold under the brand name Stelazine.

Purpose

Trifluoperazine is a drug used to treat psychotic disorders, agitation, and **dementia**.

Description

Trifluoperazine is an effective agent in treating symptoms of psychotic behavior. When used for the treatment of **schizophrenia**, trifluoperazine reduces symptoms of emotional withdrawal, anxiety, tension, **hallucinations**, and suspiciousness.

Recommended dosage

The dosage of trifluoperazine should be adjusted to the lowest level needed to control symptoms. The drug may be given orally or by intramuscular injection (a shot).

A useful initial dosage of trifluoperazine for psychotic adults is 2 to 5 mg two times each day. A common total dosage is 15 to 20 mg per day. Some persons may require up to 40 or more mg per day. When using deep intramuscular injection, 1 to 2 mg every four to six hours is usually sufficient to control symptoms within 24 hours. Total intramuscular trifluoperazine should not exceed 10 mg per day.

Control of psychotic symptoms in children between the ages of six and 12 can usually be achieved with 1 to 2 mg per day, given in 1-mg increments. Trifluoperazine is not recommended for use in children younger than six.

Precautions

Trifluoperazine increases the level of prolactin, a hormone that stimulates the mammary glands in the breast, in the blood. This is a potential problem for per-

KEY TERMS

Akathisia—Agitated or restless movement, usually affecting the legs. Movement is accompanied by a sense of discomfort and an inability to sit, stand still, or remain inactive for periods of time. Akathisia is a common side effect of some neuroleptic (antipsychotic) medications.

Amenorrhea—Absence of menstrual periods.

Anorexia—Loss of appetite or unwillingness to eat. Can be caused by medications, depression, or many other factors.

Cogwheel rigidity—An abnormal rigidity in muscles, characterized by jerky movements when the muscle is passively stretched.

Dystonia—A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving.

Orthostatic hypotension—A sudden decrease in blood pressure due to a change in body position, as when moving from a sitting to standing position.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

Tardive dyskinesia—A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs either late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

sons with a personal or family history of breast cancer and may increase the risk of breast cancer. For this reason, the benefits and risks of the drug must be carefully evaluated before it is administered.

Side effects

Relatively common side effects that accompany trifluoperazine include drowsiness, dizziness, rash, dry mouth, **insomnia**, **fatigue**, muscular weakness, anorexia, blurred vision, some loss of muscular control, and amenorrhea (lack of menstruation)in women.

Dystonia (difficulty walking or moving) may occur with trifluoperazine use. This condition may subside in 24 to 48 hours even when the person continues taking the drug and usually disappears when trifluoperazine is discontinued.

Trifluoperazine use may lead to the development of symptoms that resemble Parkinson's disease. These symptoms may include a tight or mask-like expression on the face, drooling, tremors, pill-rolling motions in the hands, cogwheel rigidity (abnormal rigidity in muscles characterized by jerky movements when the muscle is passively stretched), and a shuffling gait. Taking anti-Parkinson drugs **benztropine** mesylate or **trihexyphenidyl** hydrochloride along with the trifluoperazine usually controls these symptoms.

Trifluoperazine has the potential to produce a serious side effect called **tardive dyskinesia**. This syndrome consists of involuntary, uncoordinated movements that may appear late in therapy and may not disappear even after the drug is stopped. Tardive dyskinesia involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles. The incidence of tardive dyskinesia increases with increasing age and with increasing dosage of trifluoperazine. Women are at greater risk than men for developing tardive dyskinesia. There is no known effective treatment for tardive dyskinesia, although gradual (but rarely complete) improvement may occur over a long period.

An occasionally reported side effect of trifluoperazine is neuroleptic malignant syndrome. This is a complicated and potentially fatal condition characterized by muscle rigidity, high fever, alterations in mental status, and cardiac symptoms such as irregular pulse or blood pressure, sweating, tachycardia (fast heartbeat), and arrhythmias (irregular heartbeat). People who think they may be experiencing any side effects from this or any other medication should talk to their physician promptly.

Interactions

Trifluoperazine may reduce the effectiveness of oral anticoagulant (blood thinning) drugs.

Trifluoperazine increases the effect of drugs and substances that depress the central nervous system and. These drugs include anesthetics, opiates, **barbiturates**, atropine, and alcohol. These substances should be avoided or used sparingly by people taking trifluoperazine.

Propranolol increases the concentration of trifluoperazine. The blood pressure-lowering effects of guanethidine may be diminished by trifluoperazine. The use of diuretics with trifluoperazine may cause a sudden decrease in blood pressure often accompanied by dizziness due to a change in body position (known as orthostatic hypotension).

The blood concentration of phenytoin is increased by trifluoperazine. This may lead to phenytoin toxicity.

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ORGANIZATIONS

American Academy of Clinical Toxicology. 777 East Park Drive, PO Box 8820, Harrisburg, PA 17105-8820. Telephone: (717) 558-7750. Fax: (717) 558-7845. Web site: <http://www.clintox.org/index.html>.

American Academy of Family Physicians, 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. Telephone: (913) 906-6000. Web site: <http://www.aafp.org/>.

American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax: (202) 682-6850. Web site: <http://www.psych.org/>.

American Society for Clinical Pharmacology and Therapeutics. 528 North Washington Street, Alexandria, VA 22314. Telephone: (703) 836-6981. Fax: (703) 836-5223.

American Society for Pharmacology and Experimental Therapeutics. 9650 Rockville Pike, Bethesda, MD 20814-3995. Telephone: (301) 530-7060. Fax: (301) 530-7061. Web site: <http://www.aspet.org/>.

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Trihexyphenidyl

Definition

Trihexyphenidyl is classified as an antiparkinsonian agent. It is sold in the United States under the brand name Artane and is also available under its generic name.

Purpose

Trihexyphenidyl is used to treat a group of side effects (called parkinsonian side effects) that include tremors, difficulty walking, and slack muscle tone. These side effects may occur in patients who are taking antipsychotic medications used to treat mental disorders such as **schizophrenia**.

Description

Some medicines, called antipsychotic drugs, that are used to treat schizophrenia and other mental disorders can cause side effects that are similar to the symptoms of Parkinson's disease. The patient does not have Parkinson's disease, but he or she may experience shaking in muscles while at rest, difficulty with voluntary movements, and poor muscle tone. These symptoms are similar to the symptoms of Parkinson's disease.

One way to eliminate these undesirable side effects is to stop taking the antipsychotic medicine. Unfortunately, the symptoms of the original mental disorder usually come back, so in most cases simply stopping the antipsychotic medication is not a reasonable option. Some drugs such as trihexyphenidyl that control the symptoms of Parkinson's disease also control the parkinsonian side effects of antipsychotic medicines.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Catheterization—Placing a tube in the bladder so that it can be emptied of urine.

Dopamine—A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

Neurotransmitter—A chemical involved in the transmission of nervous impulses from cell to cell.

Parkinsonian—Related to symptoms associated with Parkinson's disease, a nervous system disorder characterized by abnormal muscle movement of the tongue, face, and neck, inability to walk or move quickly, walking in a shuffling manner, restlessness, and/or tremors.

Trihexyphenidyl works by restoring the chemical balance between dopamine and acetylcholine, two neurotransmitter chemicals in the **brain**. Taking trihexyphenidyl along with the antipsychotic medicine helps to control symptoms of the mental disorder, while reducing parkinsonian side effects. Trihexyphenidyl is in the same family of drugs (commonly known as anticholinergic drugs) as **biperiden** and **benztropine**.

Recommended dosage

Trihexyphenidyl is available in 2-mg and 5-mg tablets and an elixir containing 2 mg per teaspoonful. For the treatment of tremor, poor muscle tone, and similar side effects, trihexyphenidyl should be started at a dose of 1 to 2 mg orally two to three times daily or as needed, to a maximum daily dose of 15 mg per day. Parkinson-like side effects caused by antipsychotic drugs may come

and go, so trihexyphenidyl may not be needed on a regular basis. Trihexyphenidyl may also be prescribed to prevent these side effects before they actually occur. This is called prophylactic (preventative) therapy.

Precautions

Trihexyphenidyl should never be used in children under age three. It should be used cautiously and with close physician supervision in older children and in people over age 60. Trihexyphenidyl, like all anticholinergic drugs, decreases sweating and the body's ability to cool itself. People who are unaccustomed to being outside in hot weather should take care to stay as cool as possible and drink extra fluids. People who are chronically ill, have a central nervous system disease, or who work outside during hot weather may need to avoid taking trihexyphenidyl.

People who have the following medical problems may experience increased negative side effects when taking trihexyphenidyl. Anyone with these problems should discuss their condition with their physician before starting the drug:

- glaucoma, especially closed-angle glaucoma
- intestinal obstruction
- prostate enlargement
- urinary bladder obstruction

Although rare, some patients experience euphoria while taking trihexyphenidyl and may abuse it for this reason. Euphoria can occur at doses only two to four times the normal daily dose. Patients with a history of drug abuse should be observed carefully for trihexyphenidyl abuse.

Side effects

Although trihexyphenidyl helps to control the side effects of antipsychotic drugs, it can produce side effects of its own. A person taking trihexyphenidyl may have some of the following reactions, which may vary in intensity:

- dry mouth
- dry skin
- blurred vision
- nausea or vomiting
- constipation
- disorientation
- drowsiness
- irritability
- increased heart rate
- urinary retention

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by reducing or temporarily discontinuing trihexyphenidyl. Chewing sugarless gum or sucking on sugarless candy may also help to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement may be especially prone to urinary retention. Symptoms of this problem include having difficulty starting a urine flow and more difficulty passing urine than usual. This side effect may be severe and require discontinuation of the drug. Urinary retention may require catheterization. People who think they may be experiencing any side effects from this or any other medication should tell their physicians.

Patients who take an overdose of trihexyphenidyl are treated with forced vomiting, removal of stomach contents and stomach washing, activated charcoal, and respiratory support if needed. They are also given physostigmine, an antidote for anticholinergic drug poisoning.

Interactions

When drugs such as trihexyphenidyl are taken with antidepressants such as **amitriptyline**, **imipramine**, **trimipramine**, **desipramine**, **nortriptyline**, **protriptyline**, **amoxapine**, and **doxepin** or with many antihistamines that also have anticholinergic properties, the effects and side effects of trihexyphenidyl are usually intensified.

Drugs such as trihexyphenidyl decrease the speed with which food moves through the stomach and intestines. Because of this, the absorption of other drugs being taken may be enhanced by trihexyphenidyl. Patients receiving trihexyphenidyl should be alert to unusual responses to other drugs they might be taking and report any changes to their physician.

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Jack Raber, Pharm.D.

Trilafon *see* **Perphenazine**

Trimipramine

Definition

Trimipramine is an oral tricyclic antidepressant. It is sold in the United States under the brand name Surmontil.

Purpose

Trimipramine is used primarily to treat depression and to treat the combination of symptoms of anxiety and depression. Like most antidepressants of this chemical and pharmacological class, trimipramine has also been used in limited numbers of patients to treat **panic disorder**, **obsessive-compulsive disorder**, **attention-deficit/hyperactivity disorder**, **enuresis** (bed-wetting), eating disorders such as **bulimia nervosa**, cocaine dependency, and the depressive phase of bipolar (manic-depressive) disorder.

Description

Tricyclic antidepressants act to change the balance of naturally occurring chemicals in the **brain** that regulate the transmission of nerve impulses between cells. Trimipramine acts primarily to increase the concentration of norepinephrine and serotonin (both chemicals that stimulate nerve cells) and, to a lesser extent, to block the action of another brain chemical, acetylcholine. Trimipramine shares most of the properties of other tricyclic antidepressants, such as **amitriptyline**, **amoxapine**, **clomipramine**, **desipramine**, **imipramine**, **nortriptyline**, and **protriptyline**. Studies comparing trimipramine with these other drugs have shown that trimipramine is no more or less effective than other antidepressants of its type. Its choice for treatment is as much a function of physician preference as any other factor.

The therapeutic effects of trimipramine, like other antidepressants, appear slowly. Maximum benefit is often not evident for at least two weeks after starting the drug. People taking trimipramine should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Recommended dosage

As with any antidepressant, trimipramine must be carefully adjusted by a physician to produce the desired therapeutic effect. Trimipramine is available as 25-mg, 50-mg, and 100-mg oral capsules. Therapy is usually started at 75 to 100 mg per day and gradually increased up to 200 mg daily as needed. Hospitalized patients with more severe depression may require 300 mg per day. Amounts up to 200 mg may be given as a single dose. In people over age 60 and in adolescents, the therapeutic dose should start at 50 mg per day and is rarely increased beyond 100 mg per day.

KEY TERMS

Acetylcholine—A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Benign prostate hypertrophy—Enlargement of the prostate gland.

Norepinephrine—A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Precautions

Like all tricyclic antidepressants, trimipramine should be used cautiously and with close physician supervision in people, especially the elderly, who have benign prostatic hypertrophy, urinary retention, and glaucoma, especially angle-closure glaucoma (the most severe form). Before starting treatment, people with these conditions should discuss the relative risks and benefits of treatment with their doctors to help determine if trimipramine is the right antidepressant for them.

A common problem with tricyclic antidepressants is sedation (drowsiness, lack of physical and mental alert-

ness). This side effect is especially noticeable early in therapy. In most patients, sedation decreases or disappears entirely with time, but until then patients taking trimipramine should not perform hazardous activities requiring mental alertness or coordination. The sedative effect is increased when trimipramine is taken with other central nervous system depressants, such as alcoholic beverages, sleeping medications, other sedatives, or antihistamines. It may be dangerous to take trimipramine in combination with these substances. Trimipramine may increase the possibility of having **seizures**. Patients should tell their physician if they have a history of seizures, including seizures brought on by the abuse of drugs or alcohol. These people should use trimipramine only with caution and be closely monitored by their physician.

Trimipramine may increase heart rate and **stress** on the heart. It may be dangerous for people with cardiovascular disease, especially those who have recently had a heart attack, to take this drug or other antidepressants in the same pharmacological class. In rare cases where patients with cardiovascular disease must receive trimipramine, they should be monitored closely for cardiac rhythm disturbances and signs of cardiac stress or damage.

Side effects

Trimipramine shares side effects common to all tricyclic antidepressants. The most frequent of these are dry mouth, constipation, urinary retention, increased heart rate, sedation, irritability, dizziness, and decreased coordination. As with most side effects associated with tricyclic antidepressants, the intensity is highest at the beginning of therapy and tends to decrease with continued use.

Dry mouth, if severe to the point of causing difficulty speaking or swallowing, may be managed by dosage reduction or temporary discontinuation of the drug. Patients may also chew sugarless gum or suck on sugarless candy in order to increase the flow of saliva. Some artificial saliva products may give temporary relief.

Men with prostate enlargement who take trimipramine may be especially likely to have problems with urinary retention. Symptoms include having difficulty starting a urine flow and more difficulty than usual passing urine. In most cases, urinary retention is managed with dose reduction or by switching to another type of antidepressant. In extreme cases, patients may require treatment with bethanechol, a drug that reverses this particular side effect. People who think they may be experiencing any side effects from this or any other medication should tell their physicians.

Interactions

Dangerously high blood pressure has resulted from the combination of tricyclic antidepressants, such as trimipramine, and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Because of this, trimipramine should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting trimipramine or any other tricyclic antidepressant. The same holds true when discontinuing trimipramine and starting an MAO inhibitor.

Trimipramine may decrease the blood pressure–lowering effects of **clonidine**. Patients who take both drugs should be monitored for loss of blood-pressure control and the dose of clonidine increased as needed.

The sedative effects of trimipramine are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or medications used for other mental disorders such as **schizophrenia**. The anticholinergic effects of trimipramine are additive with other anticholinergic drugs such as **benztropine**, **biperiden**, **trihexyphenidyl**, and antihistamines.

See also Neurotransmitters

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U

Undifferentiated somatoform disorder

Definition

Undifferentiated somatoform disorder occurs when a person has physical complaints for more than six months that cannot be attributed to a medical condition. If there is a medical condition present, the complaints must be far more severe than can be accounted for by the presence of the medical problem.

Description

The physical complaints that are expressed by people with undifferentiated somatoform disorder are many and varied. The similarity between all physical complaints associated with undifferentiated somatoform disorder is an absence of medical evidence for the symptoms or for their severity.

The physical complaints usually begin or worsen when the patient is under **stress**. People with undifferentiated somatoform disorder experience problems functioning in their daily lives due to the physical symptoms that they experience. Seeing multiple doctors in an effort to find a physical cause for the reported symptoms is typical of people with this disorder. Undifferentiated somatoform disorder is also sometimes referred to as somatization syndrome.

Causes and symptoms

The symptoms of undifferentiated somatoform disorder vary widely from person to person. Some of the most common physical complaints are pain, **fatigue**, appetite loss, and various gastrointestinal problems. The physical complaints generally last for long periods. Patients with undifferentiated somatoform disorder tend to complain of many different physical problems over time.

No matter what symptoms a person complains about, the overarching characteristic of the complaints is that no physical reason can be found for them. Laboratory tests and thorough examinations by doctors will reveal no medical reason for the pains or problems the person is having. The physical problems, however, persist after the person has been told no explanation can be found.

The causes of undifferentiated somatoform disorder are not clear. Some experts believe that problems in the family when the affected person was a child may be related to the development of this disorder. Depression and stress are thought to be other possible causes. Other possible causes, especially in people who overreact to even minor medical conditions, include paying obsessive attention to any minor changes or sensations that their bodies experience. They give the feelings undue weight and worry unnecessarily about them.

Demographics

Undifferentiated somatoform disorder is relatively common. It is estimated that between 4% and 11% of the population experience the disorder at some time in their lives. Women are more likely than men to have undifferentiated somatoform disorder, as are the elderly and people of lower socioeconomic backgrounds. Young women who have low socioeconomic status are the most likely group to have undifferentiated somatoform disorder. Fifty percent of the people with this disorder have other psychological or psychiatric disorders as well, such as anxiety or depression.

Diagnosis

A person with undifferentiated somatoform disorder usually begins by visiting physicians looking for treatments for physical complaints. Later, he or she may be referred to a mental health professional. Referring physicians may continue to see the patient, however, so that a trusting relationship can be established, and the patient does not continue to bounce from doctor to doctor.

Mental health professionals use the handbook called the ***Diagnostic and Statistical Manual of Mental Disorders*** to diagnose mental disorders. The book lists diagnostic criteria, and requires that the following conditions be met in order for the clinician to diagnose this disorder:

- There must be no underlying medical cause evident that could explain the patient's physical complaints. If there is a medical condition that could be related to the complaints, the symptoms reported must be far worse than any that could be explained by the existing medical problems.
- The unexplained physical symptoms must persist for at least six months.
- The symptoms must cause problems in the patient's daily life or relationships or interfere with the patient achieving his or her goals.
- There cannot be another mental disorder that accounts for the complaints.
- The patient cannot knowingly make false complaints of physical distress.

Somatization disorder

Somatization disorder is very similar to undifferentiated somatoform disorder and the two can be easily confused. The symptoms are the same, but the diagnostic criteria are much more specific for somatization disorder. To be diagnosed with somatization disorder, the patient must have four different pain symptoms, two gastrointestinal symptoms, one sexual symptom, and one pseudoneurological symptom. These symptoms can occur at different times. The symptoms must be present for several years and must have begun before the patient was thirty years old. Just as with undifferentiated somatoform disorder, the complaints must not be traceable to any medical cause.

Hypochondriasis

Hypochondriasis is also similar in many ways to undifferentiated somatoform disorder. Patients with hypochondriasis are convinced that the physical symptoms they are experiencing are the signs of a major illness. Alternately, they may simply have an obsessive fear of contracting or developing a major illness. These patients often have a specific **diagnosis** in mind when they visit a doctor, unlike most patients with undifferentiated somatoform disorder who have complaints but do not have a cause in mind.

Treatments

Most treatments of undifferentiated somatoform disorder focus on treating any underlying psychological problems or stresses that may be causing the disorder. When the disorder occurs in conjunction with another mental health problem such as depression, treating that problem often helps to resolve or lessen the symptoms of undifferentiated somatoform disorder. Some studies indicate that antidepressants are effective in treating this disorder. Patients also may benefit from programs intended to teach them how to manage stress and to understand the correlation between psychological stressors and physiological symptoms. These programs also teach people how to cope with criticism and how to stop negative behavior patterns.

Prognosis

For many people, undifferentiated somatoform disorder is a life-long disorder. Often, the physical complaints increase or decrease in relation to stressors in the affected person's life. Many people with this disorder are eventually diagnosed with another mental disorder or with a legitimate medical problem. For some people, treatment can be successful at lessening or completely resolving symptoms.

Prevention

There are no known ways to prevent undifferentiated somatoform disorder; it is possible, however, for people who appear to be developing the disorder to enroll in programs designed to teach them coping strategies and about the relationship between psychological factors and physical symptoms.

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Urine drug screening

Definition

Urine drug screening, or toxicological screening, is a process of chemical analysis designed to test patients for drug abuse, or to insure that a patient is substance-free before undergoing a medical procedure.

Description

Urine drug screening can be used to evaluate possible accidental or intentional overdose or poisoning, to assess the type and amount of prescribed and/or illicit drugs used by a person, or to determine the cause of acute drug toxicity. It is also used to monitor drug dependency or to determine the presence of drugs in the body for medical and legal purposes.

In many occupations, urine drug screening has become a required condition of employment. Nearly all workers in certain occupations, such as law enforcement and transportation, must submit to periodic, random, and post-incident drug screening. Federal laws mandate the administration of drug screens to workers in the transportation industry, including bus drivers, truckers, airline employees, and railroad workers. Federally required testing must be conducted by a laboratory certified by the Substance Abuse and Mental Health Services Administration. Other industries must follow state regulations, which vary considerably.

Urine screening tests are able to detect general classes of compounds, such as **amphetamines**, **barbiturates**, benzodiazepines, and opiates. Drug screening can also detect cocaine, marijuana, and phencyclidine (PCP). The screening tests themselves are unable to distinguish between illicit and prescription drugs within the same class. A patient taking prescribed codeine pills and an individual using heroin would both show positive urine screening tests for opiates. It is also possible for some over-the-counter medications to cause a positive drug screen in someone who has taken neither illegal nor prescription drugs. These incorrect reactions are known as "false-positives."

Urine drug screens can detect the use of several drugs. Some of these drugs are as follows:

- cocaine
- amphetamines
- heroin
- morphine
- phencyclidine (PCP)
- benzodiazepines
- alcohol
- hydromorphone

KEY TERMS

Amphetamines—A group of powerful and highly addictive substances that stimulate the central nervous system. May be prescribed for various medical conditions, but are often purchased illicitly and abused.

Barbiturate—A class of medications (including Seconal and Nembutal) that causes sedation and drowsiness. They may be prescribed legally, but may also be used as drugs of abuse.

Benzodiazepines—A group of central nervous system depressants used to relieve anxiety or to induce sleep.

Cocaine—An illegal drug that increases energy and induces euphoria. It is addictive and is often abused.

Codeine—A medication that may be prescribed but also may be purchased illegally and is used to reduce pain.

False-positive—A test result that is positive for a specific condition or disorder, but this result is inaccurate.

Gas chromatography/mass spectrometry (GC/MS)—A definitive method of testing for specific drugs, used to confirm immunoassay results indicating drug use. GC/MS separates the substances present in the urine sample, then breaks them into unique molecular fragments, which are matched against a database of known substances.

Hydromorphone—A prescribed opiate (Dilaudid) used to treat severe pain; also abused illegally.

Immunoassay—The method used in routine or preliminary urine drug screening.

Methadone—A drug often prescribed legally as a replacement for heroin. It induces a slight high but blocks heroin from producing a more powerful euphoric effect. It may be used in heroin detoxification to ease the process, or it may be used daily after detoxification as maintenance therapy. Methadone maintenance therapy is controversial.

Tetrahydracannabinol (THC)—The active substance in marijuana.

- tetrahydrocannabinol (THC)
- propoxyphene
- methadone
- codeine
- barbituates

Certain foods, such as poppy seeds, may result in a positive urine screen for opiates, since poppy seeds are derived from opium poppies. Preliminary urine screening results, when positive, should be confirmed by a more accurate method that can distinguish between poppy seed ingestion and use of heroin or other opiates. Poppy seeds and opiates produce different chemicals, known as metabolic breakdown products or metabolites, as they travel through the body, allowing them to be distinguished from one another.

Sample collection

The method of collecting a urine sample for drug screening can be important. Some illicit drug users may attempt to substitute another person's urine, or chemically alter their own specimen. If the urine drug screen is being used for an important decision, such as employment or legal action, procedures to minimize chances of an adulterated or substituted sample may be necessary. These include measuring the temperature or pH of the sample immediately after it is procured, and using tamper-proof containers. Supervised specimen collection may be conducted to ensure that the urine indeed comes from the person being screened.

The most commonly used method for urine drug screening is immunoassay, a rapid and accurate test that uses antibodies embedded on test strips to reveal drug use. Antibodies react only in the presence of very specific substances—in this case, drugs present in urine. When a sufficient concentration of a drug (or drugs) are present, the test strip will indicate which substances have been detected. A control band on each strip confirms that the test was done correctly.

Positive screening results should always be confirmed by a more sensitive method. The most widely accepted corroborative test for all drugs is gas chromatography/mass spectrometry (GC/MS), which can determine the specific substances in the body by recognizing not only the molecular structure of the original compound, but also its metabolite, a chemical created when the drug is metabolized.

See also Addiction; Amphetamines and related disorders; Anti-anxiety drugs and abuse-related disorders; Barbiturates; Cannabis and related disorders; Cocaine and related disorders; Disease concept of chemical dependency; Methadone; Opioids and related disorders; Sedatives and related disorders

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V

Vaginismus

Definition

Vaginismus occurs when the muscles around the outer third of the vagina contract involuntarily when vaginal penetration is attempted during sexual intercourse.

Description

Vaginismus is a sexual disorder that is characterized by the outer third of the vaginal muscles tightening, often painfully. A woman with vaginismus does not willfully or intentionally contract her vaginal muscles. However, when the vagina is going to be penetrated, the muscles tighten spontaneously due to psychological or other reasons.

Vaginismus can occur under different circumstances. It can begin the first time vaginal penetration is attempted. This is known as "lifelong vaginismus." Alternately, vaginismus can begin after a period of normal sexual functioning. This is known as "acquired-type vaginismus." For some women, vaginal tightening occurs in all situations where vaginal penetration is attempted (generalized type). For other women, it occurs in only one or a few situations, such as during a gynecological examination at the doctor's office, or with a specific sex partner (situational type). According to the professional's handbook, the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*, in order for a condition to be diagnosed as vaginismus, the response must be due to psychological factors or a combination of psychological and medical factors, but not to medical factors alone. Because of this *DSM-IV-TR* criterion, this entry focuses on the psychological causes and treatments of vaginismus.

Causes and symptoms

Causes

There are many possible causes of vaginismus. One example is an upbringing in which sex was considered wrong or sinful—as in the case of some strict religious backgrounds. This is common among women with this disorder. Concern that penetration is going to be painful, such as during a first sexual experience, is another possible cause. It is also thought that women who feel threatened or powerless in their relationship may subconsciously use this tightening of the vaginal muscles as a defense or silent objection to the relationship. A traumatic childhood experience, such as sexual molestation, is thought to be a possible cause of vaginismus. Acquired-type vaginismus is often the result of sexual assault or rape.

Symptoms

Vaginismus can occur when any kind of penetration of the vagina is attempted. This includes attempted penetration by a penis, speculum, tampon, or other objects. The outer third of the vaginal muscles contract severely. This either prevents penetration completely, or makes it difficult and painful. The woman may truly believe that she wants to have sexual intercourse or allow the penetration. She may find that her subconscious desires or decisions do not allow her to relax the vaginal muscles.

Diagnosis

Diagnosing sexual disorders, including vaginismus, can often be very difficult. This is mainly due to lack of comfort many people feel in discussing sexual relations, even with their physicians. Often, cultural norms and taboos deter women from seeking assistance when they are experiencing such problems. When a physician or gynecologist is consulted, involuntary spasm during pelvic examination can confirm the **diagnosis** of vaginismus, and the physician will rule out any physiological causes for the condition. When psychological causes are suspected, referral should be made to a **psychologist** or **psychiatrist**.

According to the *DSM-IV-TR*, the first criterion for the diagnosis of vaginismus is the spasm of the muscles in the outer third of the vagina that are involuntary and recur-

KEY TERMS

Coitus—Sexual intercourse.

ring or persistent. The symptoms must cause physical or emotional distress, or, in particular, problems with relationships. The symptoms cannot occur during the course of another mental disorder that can account for them—they must exist on their own. As mentioned, the muscle spasm cannot be the direct result of any sort of physical or medical condition for vaginismus to be diagnosed.

Demographics

Although many women experience sexual disorders, it is hard to gather accurate data regarding the frequency of specific problems. Many cases go unreported. Vaginismus is thought to occur most often in women who are highly educated and of high socioeconomic status.

Treatment

There are many different treatments of vaginismus, as there is a multitude of ways to treat most sexual disorders. Therapists can use behavioral, hypnotic, psychological, educational, or **group therapy** techniques. Multiple techniques are often used simultaneously for the same patient. Much treatment is aimed at reducing the anxiety associated with penetration.

Psychotherapy

There are three settings in which psychological treatment can occur. These are in individual, couple, or group settings. During individual therapy, the treatment focuses on identifying and resolving any underlying psychological problems that could be causing the disorder. Problems stemming from issues such as childhood trauma or rape are often resolved this way. Revealing insecurities or fears about sex resulting from such things as parents' attitudes about it, or a religious upbringing, can often be discussed successfully if the affected woman can trust her therapist.

Couples therapy has been referred to as "dual-sex therapy." The idea behind couples therapy is that any sexual problem should be treated as a problem for the couple as a whole, and not just addressed as a problem for one person. Because this view is taken, the therapist interacts with the patients both separately and as a couple. The therapist addresses both the couple's sexual history and any other problems that may be occurring in the relationship. Confronting these problems may help to resolve the cause of the vaginismus. Working with a therapist on relationship problems can be very effective—perhaps especially so if the vaginismus is caused by a subconscious use of vaginal muscle spasms as a nonverbal form of protest about one or more aspects of the relationship. The couple is educated about vaginismus disorder and given advice on the kind of activities that can be engaged in at home that may be helpful in overcoming the disorder.

Group therapy, which can be very effective, is another form of therapy for vaginismus. In this form of therapy, couples or individuals who have the same or similar sexual disorders are brought together. For people who are embarrassed or ashamed of their disorder, this setting can provide comfort and strength. It is often very beneficial to witness another person discussing sex and sexual problems in an open and honest forum. It can also help to inspire patients to become more open and honest themselves.

Another positive feature of group therapy is that it provides a certain amount of pressure. Pressure to open up can help to provide a needed "push." Also the group's expectations for each other can provide positive pressure and encouragement for the group members. For example, the therapist may recommend "homework" outside the therapy sessions, including masturbation or certain kinds of foreplay. The group members will expect each other to complete the homework, and that expectation may help individual couples overcome their aversions to completing the activities.

Hypnotherapy

Hypnotherapy is also effective for some patients. In general, hypnotherapy tends to focus on overcoming the vaginismus itself, as opposed to resolving any causes or conflicts behind it. The therapist will determine if hypnotherapy is appropriate for a particular patient. There are often a number of sessions, during which the patient and therapist work to define the goals of the hypnotherapy. When the actual hypnosis occurs, the suggestions made are intended to resolve underlying fears or concerns, and to alleviate symptoms. For example, the patient may be told that she can have coitus without it being a painful experience, and that she will be able to overcome the muscle spasm.

During hypnosis, the problems causing the vaginismus may be explored, or an attempt may even be made to reverse feelings or fears that could be causing the disorder. Exploring causal relationships, as well as suggesting to the woman she can overcome her vaginal muscle spasms, can be very effective for certain patients.

Other treatments

Behavioral therapy is also used to treat vaginismus. When behavioral therapy is chosen, it is assumed that the vaginismus is a learned behavior that can be unlearned. Behavioral therapy generally involves desensitization. Patients are exposed to situations that they find create a mild sense of psychological discomfort or anxiety. Once these situations are conquered, the patient is exposed to sexual situations that they find more threatening, until coitus is eventually achieved without difficulty.

Another type of treatment for vaginismus involves desensitization over a period of time using systematic vaginal dilation. In the beginning of the treatment, the woman inserts a small object into her vagina. Over time, she inserts larger and larger vaginal dilators. Eventually, a dilator the size of a penis can be inserted comfortably and sexual intercourse can be achieved. There is some debate about this procedure, as it treats the symptoms and not the underlying causes of the vaginismus disorder.

Prognosis

Vaginismus is generally considered to be the most treatable sexual disorder. Successful treatment has been reported to be 63% or higher. For different people, the possibility of success using different treatments varies, because different cases of vaginismus disorder have varying causes. Generally, a treatment plan combining two or more therapeutic techniques is recommended.

Prevention

There is no known way to successfully prevent vaginismus; however, maintaining open marital communication may help to prevent the disorder, or to encourage seeking help if it does arise.

See also Cognitive-behavioral therapy; Systematic desensitization

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American Psychological Association. 750 First Street NE, Washington, D.C. 20002-4242, 800-374-2721;<www.apa.org>.

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Valerian

Definition

Valerian is an herbal remedy derived from the dried roots of the valerian plant, *Valeriana officinalis*. The plant belongs to the Valerianaceae family. It has been used for over a thousand years as a mild sedative and hypnotic (a preparation that brings on sleep). Valerian is native to Europe and parts of Asia; it has since been introduced in the United States, placed under cultivation and now growing in the wild, as well. It is often cultivated for its pinkish white or lavender flowers as well as for its medicinal uses. The name "valerian" is thought to derive from the Latin verb *valere*, which means "to be well." It is also sometimes said to derive from Valeria, the province of the Roman Empire where the plant may have originated.

According to one marketing research firm, valerian is the fastest-growing herbal remedy in the United States; its sales more than doubled between 2000 and 2001.

Purpose

Valerian is most commonly used to relieve mild cases of anxiety and **insomnia**. It was given during World War I to soldiers suffering from battle shock. It has also been recommended for the relief of menstrual cramps and as a carminative, or preparation that relieves gas in the stomach and intestines. Lotions made with valerian extract are said to soothe skin rashes and swollen joints.

Description

The valerian plant prefers the damp lime-rich soil near streams or rivers, where it may grow as tall as 5 ft (1.5 m). It can, however, be grown in drier soil at higher elevations, where it may grow only 2 ft (.67 m) tall. Some herbalists consider the drier-climate variety of valerian to have greater medicinal potency.

KEY TERMS

Carminative—A substance or preparation that relieves digestive gas.

Hypnotic—A type of medication that induces sleep.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Placebo—An inactive substance or preparation used as a control in experiments with human subjects to test the effectiveness of a drug or herbal preparation. Some patients may experience a medicinal response or experience side effects to a placebo simply because they have faith in its powers even though it contains no medicine.

Rhizome—The fleshy underground horizontal root of certain plants. Valerian preparations are made from dried rhizomes as well as from roots of the valerian plant.

Tincture—An alcohol-based herbal extract prepared by soaking parts of the plant in a mixture of alcohol and water. Established ratios and dilutions are followed.

Valerenic acid—The primary medicinal component in valerian preparations.

The parts of the plant that are used for medicinal purposes are the roots and rhizomes (horizontal underground stems), which are typically yellowish-brown in color. The roots and rhizomes are harvested in the autumn of the plant's second year. They can be freeze-dried and used to prepare tablets or capsules containing the ground herb. Juice can be pressed from the fresh root, or the root may be mixed with alcohol to become a fluid extract or tincture of valerian. When valerian is used to relieve tension or induce sleep, it is frequently combined with either **passionflower** (*Passiflora incarnata*), lemon balm (*Melissa officinalis*) or skullcap (*Scutellaria lateriflora*). Because valerian tea has a somewhat bitter taste, flavorings are often added, including peppermint or fruit flavor, to make a more pleasant-tasting drink.

Although not all the compounds in valerian that have medicinal value have been identified, two compounds in its essential oil—valerenic acid and bornyl— appear to be the most important. Like most prescription tranquilizers, valerian appears to affect a neurotransmitter (GABA) in the central nervous system.

There is some disagreement among researchers about the efficacy of valerian as a tranquilizer and aid to sleep. While a team of Swiss researchers found a valerian/lemon balm combination to be significantly more effective than a placebo in inducing sleep, another group in the United States concluded that valerian is overrated as a sedative. Further research may help to settle the question, but multiple studies that are currently available are inconclusive. It appears to have mild sedative properties.

Recommended dosage

Experts in herbal preparations recommend that valerian products should be standardized to contain 0.8% valerenic or valeric acid.

Adults may use the following amounts of valerian to reduce nervousness or relieve menstrual cramps:

- 2–3 g dried root in tea, up to several times daily
- 1/4–1/2 tsp (1–3 mL) valerian tincture, up to several times daily
- 1/4 tsp (1–2 mL) fluid extract
- 150–300 mg valerian extract, standardized to contain 0.8% valerenic acid

To relieve insomnia, one of the above dosages may be taken 30–45 min before bedtime. It may take one to two weeks of regular use before the herbal preparation takes effect.

When giving valerian to children, recommended adult dosages should be adjusted in proportion to the child's weight. Most dosages of herbal products are calculated for an adult weighing 150 lb (70 kg). A child weighing 75 lb (35 kg) should therefore receive 1/2 the adult dose.

Precautions

Persons who take valerian should consult an experienced herbalist about dosage and about reliable sources of the herb. Because herbal preparations are not regulated by the U. S. Food and Drug Administration, consumers cannot be certain of the freshness and potency of commercial herbal products. In July 2001, an independent laboratory published the results of its tests of 17 valerian products; only nine contained the amount of valerian that their labels claimed. Of the remaining eight products, four contained only half the amount of valerian that they should have, and the other four contained none at all.

Although valerian has a good reputation for safety when used as directed, it should not be used in high doses or taken continuously for longer than two to three weeks.

Side effects

Some people taking valerian may experience a paradoxical effect; that is, they may feel agitated or jittery instead of relaxed or sleepy. This side effect is not dangerous, but it should be reported to the patient's health care provider. If the dosage is too high, an individual could experience longer sleep than usual, and wake up not feeling well-rested.

Prolonged use of valerian results in tolerance, and increasing the dose may have serious side effects. According to some researchers, long-term use of valerian may cause psychological depression, damage to the liver, or damage to the central nervous system.

High short-term doses of valerian have been reported to cause headaches, muscle spasms, dizziness, digestive upsets, insomnia, and confusion.

Interactions

Although valerian has been regarded as a relatively safe herb because few interactions with prescription medications have been reported, newer research indicates that it should be used cautiously following surgery. Like **St. John's wort**, valerian can interact with anesthetics and other medications given to patients after surgery. Because valerian has a mild sedative effect, it should not be taken together with alcoholic beverages, benzodiazepines, **barbiturates**, or antihistamines. Some components of valerian are metabolized in the liver. This herb has the potential to interact with liver metabolizaed prescription medicines.

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Rebecca J. Frey, Ph.D.

Valium *see* **Diazepam**

Valproic acid

Definition

Valproic acid is an anticonvulsant (anti-seizure) drug. In the United States, valproic acid is also known as valproate, and is sold under the brand name Depakene.

Purpose

The United States Food and Drug Administration (FDA) recognizes valproic acid for the treatment of epilepsy and for mania that occurs with **bipolar disorder** (previously called manic-depressive disorder). Valproic acid is also approved for the prevention of migraine headaches.

Description

Valproic acid's properties in preventing **seizures** were first discovered in Europe in 1963. The medication was first used clinically in the United Stated in 1978.

Valproic acid is effective in treating a variety of seizure types, which include simple and complex absence seizures, partial seizures, and clonic-tonic seizures (grand mal seizures). Valproic acid is effective in treating the manic episodes of patients with bipolar disorder. Patients who have bipolar disorder resulting from a head injury and patients who do not respond to or who cannot tolerate conventional lithium therapy (normally the therapy of choice for bipolar disorder) can be treated with valproic acid. In addition, valproic acid provides a 50% or greater reduction in the frequency of migraine headaches. Valproic acid is also safe and effective in preventing headaches that arise as a side effect of taking a class of drugs known as selective serotonin reuptake inhibitors (SSRI). These drugs include **sertraline** (Zoloft), **paroxetine** (Paxil), **fluoxetine** (Prozac), **fluvoxamine** (Luvox), and **citalopram** (Celexa).

Valproic acid comes in 250-mg gelatin capsule and in 250 mg/5ml-syrup.

Recommended dosage

The dosage of valproic acid used to treat epilepsy depends on the type of seizures the patient has. The doses are determined based on the patient's weight and never based on the patient's age.

KEY TERMS

Absence seizure—Absence (petit mal) seizures usually begin with a brief loss of consciousness and last between one and 10 seconds. People having a petit mal seizure become very quiet and may blink, stare blankly, roll their eyes, or move their lips. A petit mal seizure lasts 15–20 seconds. When it ends, the individual resumes whatever he or she was doing before the seizure began, and may not realize that anything unusual happened.

Clonic-tonic seizure—This is the most common type of seizure among all age groups and is categorized into several phases beginning with vague symptoms hours or days before an attack. These seizures are sometimes called grand mal seizures.

The initial dose of valproic acid used to treat mania is 750 mg daily. This dose is then reduced to the lowest dose that will achieve the desired effects. Another dosage strategy is based on patient weight. The starting dose is 30 mg per kilogram of body weight on days one and two followed by 20 mg per kg of body weight taken daily on days three through ten.

For prevention of migraine headaches, a dose of 250 mg twice daily is beneficial. It may take up to 1,000 mg of valproic acid to control migraine attacks.

Precautions

Patients who have liver disease should not take valproic acid. Pregnant women should not take valproic acid, because it can harm the developing fetus. Patients who are allergic to valproic acid should not take it.

When it is necessary for children under age two and patients who have pancreatitis to take valproic acid, the drug should be used cautiously and with close physician monitoring.

Side effects

Valproic acid can cause liver damage. Before starting valproic acid therapy, every patient should have a blood test to assess his or her liver function. The risk of valproic acid causing liver damage is greatest during the first six months of treatment. Liver function tests should be done once a month during the first three months, then every three to six months for as long as the patient continues to take the drug. Vomiting, lethargy, anorexia, and jaundice (yellowing of the skin) may precede signs of liver damage. If a patient develops severe or unusual abdominal pain, this may be a sign of pancreatitis (inflammation of the pancreas). Pancreatitis can occur in both children and adults. It can develop shortly after valproic acid is started or after several years of use.

Other side effects of valproic acid may include nausea, vomiting, indigestion, and either diarrhea or constipation. Headaches, dizziness, lack of coordination, confusion, **fatigue**, tremor, drowsiness, and seizures have also been associated with the use of valproic acid. Behavioral changes associated with the drug including irritability, longer and deeper sleep, hyperactivity, increased sociability, increased sadness, happiness or aggression, are seen more often in children than in adults taking valproic acid.

Fewer than 1% of patients experience appetite changes. These changes may include either diminished or increased appetite. Skin rash, photosensitivity (acute sensitivity to the sun), hair loss, and other hair changes have also been reported in people using valproic acid.

Interactions

Using valproic acid with other anticonvulsant drugs, such as phenobarbital, **clonazepam**, and **lamotrigine** may cause excessive sedation (drowsiness and lack of physical and mental alertness). Valproic acid may diminish the benefits of phenytoin which is another commonly used anticonvulsant.

Taking aspirin during valproic acid therapy may cause valproic acid levels to increase to toxic (poisonous) levels. Other medications that may cause valproic acid toxicity are erythromycin, an antibiotic, and the antidepressant **amitriptyline**. Drugs that can decrease the effectiveness of valproic acid include **carbamazepine** and cholestyramine. **Ginkgo biloba**, an herbal supplement commonly available in the United States, may be prepared with a chemical called 4'-O-methylpyridoxine. If this chemical remains in the herbal preparation, it can cause seizures, and reduce the effectiveness of valproic acid. Severe central nervous depression has been reported with the use of valproic acid and another anticonvulsant called primidone.

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KEY TERMS

Cerebrovascular—Blood flow in the brain.

Delirium—A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness.

Vascular—Pertaining to the bloodstream (arteries, veins, and blood vessels).

Vascular dementia

Definition

Dementia is a decline in a person's mental capacities and intellectual abilities that is great enough to affect the person's normal daily functioning. Vascular dementia is dementia that is caused by disease of the blood vessels of the **brain** (cerebrovascular disease).

Description

Vascular dementia is caused by cerebrovascular disease that occurs almost entirely in the elderly. People with vascular dementia generally experience a decline in thought processes (cognitive function) that follows specific steps. This decline is often punctuated by small strokes—ruptures of tiny blood vessels in the brain. People experiencing vascular dementia often have problems with memory, abstract thinking, object identification or recognition, speech creation, speech comprehension, and motor activities.

Causes and symptoms

The signs of dementia often begin with impaired memory function. Sometimes a person has difficulty learning new things or remembering new events, and sometimes the person has difficulty recalling events or things that he or she used to know. Other signs of dementia include impairment in other areas of thought processing. Sometimes a person with vascular dementia may have difficulty producing coherent speech, or may have other language impairments, such as problems understanding spoken or written language. The signs of vascular dementia are similar to those of Alzheimer's disease (AD).

Difficulty with motor activities is a problem for some people with vascular dementia. Things that require hand-eye coordination, such as tying shoes or undoing buttons, are examples of motor activities that may be impaired. People with vascular dementia may also have difficulty recognizing familiar objects, or may be unable to name them. Problems organizing things, putting events in sequence, or problems performing other types of abstract thinking may be present.

Some people with vascular dementia exhibit neurological signs that indicate the presence of cerebrovascular disease. They may have weakness of the arms or legs, abnormal reflexes, or abnormalities in the way they walk. Some people also exhibit behavioral disturbances related to the dementia. A person can be violent or aggressive towards others—often his or her caretaker. The person may act impulsively and irritably, and sometimes scream.

Vascular dementia is thought to be caused by small strokes that interfere with blood flow to the brain. Usually, vascular dementia is caused by many small strokes over time, rather than one large **stroke**. Sometimes this is referred to as multi-infarct dementia (MID). If the vascular dementia is caused by one large stroke, or develops in less than three months, then it is called "acute onset vascular dementia." Acute onset vascular dementia is rare.

Demographics

In most countries, vascular dementia is a much less common form of dementia than AD. This is true in North America and Europe, but is not so in Japan, where it is more common than AD. Overall, vascular dementia is the second most common form of dementia, after AD. About 10–20% of patients who experience dementia have the vascular form of the disorder. The difference in prevalence in different countries may result from different lifestyle factors rooted in the culture.

Vascular dementia is more common in men than in women, which may be because men are more likely than women to suffer from strokes. Vascular dementia becomes increasingly prevalent as people grow older. The number of people affected by vascular dementia rises dramatically during and after the sixth decade. Vascular dementia usually occurs at a younger age than AD.

Diagnosis

The first step in the **diagnosis** of vascular dementia is to verify that dementia is present. The *DSM* indicates that impairments to memory must be present for a diagnosis of vascular dementia. Memory problems can include difficulties in learning and retaining new information, problems remembering past events, or things that were learned before dementia took root.

In addition to memory impairment, the *DSM* also specifies that one or more other impairments must be present. These impairments can include language problems that encompass not being able to form speech and/or not being able to understand language, either spoken or written. The patient may have problems performing activities that require hand-eye coordination such as tying shoes, even though motor function is normal. Another possible impairment is a problem recognizing or identifying objects, although the person is able to use his or her sense organs fully. Also, problems doing tasks such as organizing things, planning events, putting things into sequence, or problems thinking abstractly may exist.

If the patient has memory problems and one or more other impairments, For a diagnosis of vascular dementia to be made, these impairments must cause problems for the patient's functioning in important parts of his or her daily life. Also, the patient must be significantly less able to function than during a previous time. In addition, the problems cannot occur during the course of an event that is categorized as a **delirium**. There must be evidence that the problem is a result of cerebrovascular disease.

If the dementia occurs without any other significant signs or symptoms, then it is classified as uncomplicated. There are three other possible classifications as given by the *DSM*. These are based on the predominant feature of the dementia. They are: vascular dementia with delirium, vascular dementia with **delusions**, and vascular dementia with depressed mood. If there are significant behavioral disturbances occurring as a result of the dementia, then that is specified.

Vascular dementia and AD are similar in many ways, and can be confused. The most significant difference between the two is that vascular dementia can be diagnosed using physiological evidence of cerebrovascular disease. Also, AD generally occurs first as a slow loss of memory function, and then as a gradual decline into eventual dementia. Vascular dementia, however, generally occurs suddenly. The patient often declines in a stepwise fashion, with each step occurring after a stroke.

Treatments

The treatments for vascular dementia focus on attempts to slow or halt the progression of the disorder and alleviate some of the symptoms. The disorder cannot be cured or reversed. The most common way to treat vascular dementia is to try to prevent further strokes. Treatments include diet and drug treatment for hypertension (high blood pressure), aspirin therapy, smoking cessation, avoidance of heavy alcohol use, and **stress** reduction. Some drugs that are used to treat mild AD are being studied for their effectiveness in treating vascular dementia.

Prognosis

Vascular dementia is a disorder that cannot be reversed. The progression of the disorder can, however, be slowed. Using drugs, along with lifestyle changes to prevent more strokes from occurring, can be effective at slowing the progression of vascular dementia.

Prevention

Vascular dementia is generally associated with a series of strokes causing increasing mental impairment. Measures generally recommended by physicians may prevent strokes and may be effective in helping to prevent vascular dementia. These measures include such things as quitting smoking, decreasing cholesterol levels, treating hypertension by reducing sodium (salt) intake, decreasing alcohol consumption, quitting smoking, and other lifestyle changes. One study illustrated that consuming a small amount of red wine regularly reduces the risk of all forms of dementia.

See also Alzheimer's disease

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Venlafaxine

Definition

Venlafaxine is an antidepressant available in the United States under the trade name of Effexor or Effexor XR.

Purpose

Venlafaxine is used to treat depression and **generalized anxiety disorder**. It has also been used to treat **obsessive-compulsive disorder** and irritable bowel syndrome.

Description

Venlafaxine is an antidepressant. It has actions common to both the cyclic antidepressants such as **imipramine** (Tofranil) and **amitriptyline** (Elavil,) and the selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine (Prozac), **sertraline** (Zoloft), and **paroxetine** (Paxil). It is believed to derive its actions by increasing levels of both norepinephrine and serotonin in the **brain**.

The therapeutic effects of venlafaxine, like other antidepressants, appear slowly. Maximum benefit is often not evident for at least two weeks after starting the drug. People taking venlafaxine should be aware of this and continue taking the drug as directed even if they do not see immediate improvement.

Venlafaxine is broken down by the liver and eliminated from the body by the kidneys. As a result, the dose of venlafaxine must be lowered in people with liver or kidney disease.

KEY TERMS

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Antipsychotic—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Depression—A mental state characterized by excessive sadness. Other symptoms include altered sleep patterns, thoughts of suicide, difficulty concentrating, agitation, lack of energy, and loss of enjoyment in activities that are usually pleasurable.

Generalized anxiety disorder—A general form of fear that can dominate a person's life.

Mania—An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Obsessive-compulsive disorder—Disorder in which the affected individual has an obsession (such as a fear of contamination, or thoughts he or she doesn't like to have and can't control) and feels compelled to perform a certain act to neutralize the obsession (such as repeated handwashing).

Serotonin syndrome—A condition characterized by at least three of the following symptoms: diarrhea, fever, extreme perspiration, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking. It is a result of too much serotonin in the body.

Venlafaxine is available in 25-mg, 37.5-mg, 50-mg, 75-mg, and 100-mg rapid-release tablets and 75-mg and 150-mg extended-action capsules.

Recommended dosage

The recommended initial dose of venlafaxine is 75 mg daily taken as two or three equal doses. The dose may be increased in 75-mg increments every four days as needed until symptoms of depression or anxiety resolve. Most commonly, dosages range between 150 mg to 225

mg daily. although in severe situations, 375 mg per day may be needed. Once patients are stabilized using the rapid-acting tablets, they may be converted over to the appropriate dose of extended-release capsules.

In people with liver disease, the daily dosage of venlafaxine should be cut in half. In patients with kidney disease, the daily dosage of venlafaxine should be reduced 25–50%, depending upon the extent of kidney damage. When stopping venlafaxine, the dosage should be reduced gradually over a period of at least two weeks before the drug is totally stopped.

Precautions

Patients taking venlafaxine should be monitored closely for **insomnia**, anxiety, mania, significant weight loss, **seizures**, and thoughts of **suicide**.

Caution should also be exercised when prescribing venlafaxine to patients with impaired liver or kidney function, the elderly (over age 60) children, individuals with known manic-depressive disorder or a history of seizures, people with diabetes, and individuals expressing ideas of committing suicide.

Individuals should not take MAO inhibitors such as Nardil during venlafaxine therapy, for two weeks prior to beginning venlafaxine therapy, and for five weeks after stopping venlafaxine therapy.

Care should be taken to weigh the risks and benefit of this drug in women who are, or wish to become, pregnant, as well as in breast-feeding mothers.

People with diabetes should monitor their blood or urine sugar more carefully, since venlafaxine may affect blood sugar.

Until an individual understands the effects that venlafaxine may have, he or she should avoid driving, operating dangerous machinery, or participating in hazardous activities. Alcohol should not be used while taking venlafaxine.

Side effects

More common side effects include decreased sexual drive, restlessness, difficulty sitting still, skin rash, hives, and itching.

Less common side effects include fever and/or chills, and pain in joints or muscles.

Rare side effects include pain or enlargement of breasts and/or abnormal milk production in women, seizures, fast heart rate, irregular heartbeats, red or purple spots on the skin, low blood sugar and its symptoms (anxiety, chills, cold sweats, confusion, difficulty concentrating, drowsiness, excess hunger, rapid heart rate, headache, shakiness or unsteadiness, severe **fatigue**), low blood sodium and its symptoms (including confusion, seizures, drowsiness, dry mouth, severe thirst, decreased energy), serotonin syndrome (usually at least three of the following: diarrhea, fever, sweatiness, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking), excitability, agitation, irritability, pressured talking, difficulty breathing, and odd body or facial movements.

Interactions

Venlafaxine interacts with a long list of other medications. Anyone starting this drug should review the other medications they are taking with their physician and pharmacist for possible interactions. Patients should always inform all their health care providers, including dentists, that they are taking venlafaxine.

Dangerously high blood pressure, rapid changes in heart rate, high fever, muscle stiffness, and sudden muscle spasms have resulted from the combination of antidepressants, such as venlafaxine, and members of another class of antidepressants known as monoamine oxidase (MAO) inhibitors. Because of these serious adverse reactions, venlafaxine should never be taken in combination with MAO inhibitors. Patient taking any MAO inhibitors, for example Nardil (**phenelzine** sulfate) or Parmate (**tranylcypromine** sulfate), should stop the MAO inhibitor then wait at least 14 days before starting venlafaxine or a tricyclic antidepressant. The same holds true when discontinuing venlafaxine and starting an MAO inhibitor.

Some other drugs such as **trazodone** (Desyrel), sibutramine (Meridia), and sumatriptan (Imitrex) also interact with venlafaxine and cause a syndrome known as neuroleptic malignant syndrome, characterized by irritability, muscle stiffness, shivering, muscle spasms, and altered consciousness.

The sedative effects (drowsiness, lack of mental clarity) of venlafaxine are increased by other central nervous system depressants such as alcohol, sedatives, sleeping medications, or other medications used for mental disorders such as **schizophrenia**.

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Kelly Karpa, RPh, Ph.D.

Vivactil *see* **Protriptyline**

Vocal tic *see* **Tic disorders**

Vocational rehabilitation

Definition

Vocational rehabilitation (VR) is a set of services offered to individuals with mental or physical disabilities. These services are designed to enable participants to attain skills, resources, attitudes, and expectations needed to compete in the interview process, get a job, and keep a job. Services offered may also help an individual retrain for employment after an injury or mental disorder has disrupted previous employment.

Purpose

Vocational rehabilitation services prepare qualified applicants to achieve a lifestyle of independence and integration within their workplace, family and local community. This transition is achieved through work evaluation and job readiness services, job counseling services, and medical and therapeutic services. For individuals with psychiatric disabilities, situational assessments are generally used to evaluate vocational skills and potential.

Precautions

Vocational rehabilitation as operated by state agencies is not an entitlement program. Only individuals considered eligible can receive VR services. Eligibility criteria require that an individual be at least 16 years old, unemployed or under-employed, and have a physical or mental disability that results in a substantial barrier to employment, such as psychotic disorders, alcohol and other drug abuse dependence, mental and emotional disorders, attention deficit disorders, specific learning disabilities, and physical and sensory disabilities. In addition, the individual must be able to benefit from VR services. An individual must also need help to prepare for, find, and succeed in paid employment. When resources are limited, individuals with the most significant disabilities must be served first.

KEY TERMS

IEP (Individualized Education Plan)—Under federal law governing special education, every child in public schools who is determined through assessment to have special mental disability needs has an IEP. An IEP is typically developed by a team of professionals that may include special education teachers, physical, occupational and speech therapists, psychologists, parents or guardians, and others who may be called on to provide expertise. The team meets at least once a year to set goals for the next school year and to assess progress on already established goals. Parents who are not satisfied with school-based assessments have the right to ask for independent assessments that must be paid for by the school system.

Integrated setting—Placing individuals in usual employment situations rather than making placements into sheltered workshops or other segregated settings.

Natural supports—Using a person's already existing support network to help the person reach a goal, such as the employment of their choice.

Person-centered planning—A technique in which a plan for a person's future is developed by a team consisting of the person, family members, service providers and friends (natural supports). The team develops a practical plan based on the person's wishes and dreams. Each team member agrees to perform certain tasks identified in the plan to help the person reach goals.

Section 504—This section of the Rehabilitation Act of 1973 provides that no person may be discriminated against because of a physical disability. For instance, a child who uses a wheelchair. If a science class is on the second floor and the building has no elevator, the school must find a way to ensure that children in wheelchairs have access to that science class. An educational plan for a child who has both cognitive and physical disabilities is developed under an IEP.

Description

Vocational rehabilitation services are based on individual needs and defined as any goods or services an individual might need to be employable, such as assistive technology devices and services. For instance, a person

who is blind would need screen reading software to access a computer and people with a cognitive or mental disability might need a talking electronic reminder device programmed to prompt them when it is time to perform certain tasks.

Vocational rehabilitation can be provided by private organizations, but is not typically funded under **managed care** arrangements. Thus, most people apply to state vocational rehabilitation agencies that are funded through federal and state monies. Typically, state agencies have offices in their state's major cities and towns. State VR agencies do not necessarily offer the same services or deliver services in the same way in every state, so individuals seeking services must learn how to access the VR program in their own state. The federal VR component is administered by the U.S. Department of Education Rehabilitation Services Administration and authorized by the Rehabilitation Act of 1973 as amended in the 1988 reauthorization.

Most vocational rehabilitation services are free for eligible applicants; however, applicants may be asked to use other benefits, such as: insurance, Pell grants or other financial aid for training or higher education, to pay part of program costs.

Best practices in vocational rehabilitation include individual choice, person-centered planning, integrated setting, natural supports, rapid placement, and career development. The term *integrated setting* refers to placing individuals in usual employment situations rather than making placements into sheltered workshops or other segregated settings. Natural supports are the person's already existing support network, including family members, service providers, and friends, who can help the person reach a goal, such as the employment of their choice. Person-centered planning is a technique in which a plan for a person's future is developed by a team consisting of the person and his or her natural supports, and the team develops a practical plan based on the person's wishes and dreams. Each teammember agrees to perform certain tasks identified in the plan to help the person reach goals. Unfortunately, not all VR programs incorporate all of these best practices.

Preparation

Vocational rehabilitation transition planning services are required for all public and private education students aged 16 and over, who have Individualized Education Plans (IEPs) or Rehabilitation Act Section 504 Plans. Transition services help students make the transition from school to employment, training or higher education. Older individuals who have acquired disabilities and are applying for VR services must undergo medical and psychological assessments at their local VR office to determine the extent of their disabilities, except for individuals receiving SSDI or SSI who are presumed eligible without assessments. Applicants may receive treatment and counseling, if needed, before training and employment. All VR services are described in an applicant's Individualized Plan for Employment (IPE). Applicants may design the IPE either on their own or with the assistance of their assigned VR counselor, usually a person with a master's degree in rehabilitation counseling.

Aftercare

A vocational rehabilitation counselor will assist an applicant gain access to an employment agency to help locate a job. Counselors may provide support (supported employment programs) if applicants need support to keep a job. This support may include job coaching, which includes working with the person in the workplace until the person is comfortable with the work. The counselors also act as resources if a job does not work out by assessing what happened and counseling the person on how to improve performance or change habits that were not perceived favorably in the workplace.

Risks

Applicants may not be satisfied with the pace of progress toward their employment goal through VR or they may not believe their wishes or talents and skills are being taken seriously. Applicants wanting to start their own businesses or engage in telecommuting may not be successful in receiving vocational rehabilitation assistance. Applicants may find that VR counselors tend to recommend low-level and low-paying jobs traditionally recommended for VR applicants, such as food service and janitorial work. Applicants may also be turned away by VR counselors because the counselors decide the applicant's disability is too severe for the person to benefit from VR services. An additional risk for individuals with mental disorders is a usual lack of coordination between VR and mental health systems.

To address these problems in the VR system, the United States Congress passed the Ticket To Work Act. Under this Act, persons with mental or physical disabilities will receive a ticket worth a certain amount of money. They may take this ticket to any private or public entity that provides job training and placement, including state VR programs. The entities providing the employment-related services will be able to redeem the tickets only after the person is gainfully employed for a certain period of time. States are on a staggered schedule to begin implementing the program; persons in the first

states started receiving tickets in 2001. All states will be instituting the Ticket to Work Act by 2004.

Normal results

Individuals with mental or physical disabilities will receive the assessments, counseling, training, placement, accommodations and long-term supports needed to allow them to engage in the gainful employment of their choice.

Abnormal results

Individuals with mental or physical disabilities remain unemployed or under employed. More than 70% of people with disabilities are unemployed; for people with mental disorders, that percentage ranges from 70-90%.

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Association for Persons in Supported Employment (APSE) provides a nationwide supported employment network through its national program and state chapters. APSE works to increase supported employment opportunities, educate consumers regarding their rights in supported employment activities and train professionals to create quality supported employment services. APSE, 1627 Monument Avenue, Richmond, VA 23220. Phone: (804) 278-9187. Fax: (804) 278-9377. <http://www.apse.org/>.

The Office of Special Education and Rehabilitative Services' Rehabilitation Services Administration (RSA) web site describes the programs offered, federal law and regulations governing VR programs, and includes a link to all state VR programs and agencies. <http://www.ed.gov/offices/OSERS/RSA/>.

State Rehabilitation Councils. These councils advise and assist state VR programs in preparing state plans for vocational services to promote employment for persons with disabilities and ensure a link between citizen participation and the legislative process. Persons with disabilities or their family members must make up 60% or more of a Council's membership. The Pennsylvania Rehabilitation Council has a web site with links to various state rehabilitation councils at <http://www.parac.org/>. The Pennsylvania Rehabilitation Council can be reached at: Rehabilitation Council Support Project, 1902 Market Street, Camp Hill, PA 17011. Telephone: (717) 975-2004, or toll free: (888) 250-5175. TTY: (877) 827-9974. Fax: (888) 524-9282.

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Voyeurism

Definition

Voyeurism is a psychosexual disorder in which a person derives sexual pleasure and gratification from looking at the naked bodies and genital organs or observing the sexual acts of others. The voyeur is usually hidden from view of others. Voyeurism is a form of paraphilia.

A variant form of voyeurism involves listening to erotic conversations. This is commonly referred to as telephone sex, although it is usually considered voyeurism primarily in the instance of listening to unsuspecting persons.

Description

The object of voyeurism is to observe unsuspecting individuals who are naked, in the process of undressing or engaging in sexual acts. The person being observed is usually a stranger to the observer. The act of looking or peeping is undertaken for the purpose of achieving sexual excitement. The observer generally does not seek to have sexual contact or activity with the person being observed.

If orgasm is sought, it is usually achieved through masturbation. This may occur during the act of observation or later, relying on the memory of the act that was observed.

KEY TERMS

Paraphilia—A disorder that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) non-human objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

Voyeur—A person who engages in the behavior of voyeurism.

Frequently, a voyeur may have a fantasy of engaging in sexual activity with the person being observed. In reality, this fantasy is rarely consummated.

A number of states have statutes that render voyeurism a crime. Such statutes vary widely regarding definitions of voyeurism. Most states specifically prohibit anyone from photographing or videotaping another person, without consent, while observing that person in the privacy of his home or some other private place.

Causes and symptoms

Causes

There is no scientific consensus concerning the basis for voyeurism. Most experts attribute the behavior to an initially random or accidental observation of an unsuspecting person who is naked, in the process of disrobing, or engaging in sexual activity. Successive repetitions of the act tend to reinforce and perpetuate the voyeuristic behavior.

Symptoms

The act of voyeurism is the observation of an unsuspecting person who is naked, in the process of disrobing, or engaging in sexual activity that provides sexual arousal. To be clinically diagnosed, the symptoms must include the following elements:

- recurrent, intense or sexually arousing fantasies, sexual urges, or behaviors
- fantasies, urges, or behaviors that cause significant distress to an individual or are disruptive of his or her everyday functioning.

Demographics

Voyeurism is apparently more common in men, but does occasionally occur in women. However, the prevalence of voyeurism is not known. Contemporary U.S. society is increasingly voyeuristic (as in the example of "real" television); however **diagnosis** is made only when this is a preferred or exclusive means of sexual gratification.

The onset of voyeuristic activity is usually prior to the age of 15 years. There are no reliable statistics pertaining to the incidence of voyeurism in adulthood.

Diagnosis

According to the mental health professional's handbook, ***Diagnostic and Statistical Manual of Mental Disorders***, two criteria are required to make a diagnosis of voyeurism:

- Over a period of at least six months, an individual must experience recurrent, intense, sexually arousing fantasies, sexual urges, or behaviors that involve the act of observing an unsuspecting person who is naked, in the process of disrobing, or engaging in sexual activity.
- The fantasies, sexual urges, or behaviors must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

In order for a condition to be labeled "voyeurism," the fantasies, urges, or behaviors to watch other persons must cause significant distress in the individual or be disruptive to his or her everyday functioning.

Treatments

For treatment to be successful, a voyeur must want to modify existing patterns of behavior. This initial step is difficult for most voyeurs to admit and then take. Most must be compelled to accept treatment. This may often be the result of a court order.

Behavioral therapy is commonly used to try to treat voyeurism. The voyeur must learn to control the impulse to watch non-consenting victims, and just as importantly to acquire more acceptable means of sexual gratification. Outcomes of behavioral therapy are not known. There are no direct drug treatments for voyeurism.

Voyeurism is a criminal act in many jurisdictions. It is usually classified as a misdemeanor. As a result, legal penalties are often minor. The possibility of exposure and embarrassment may deter some voyeurs. It is also not easy to prosecute voyeurs as intent to watch is difficult to prove. In their defense statements, they usually claim that the observation was accidental.

Prognosis

Once voyeuristic activity is undertaken, it commonly does not stop. Over time, it may become the main form

of sexual gratification for the voyeur. Its course tends to be chronic.

The prognosis for eliminating voyeurism is poor because most voyeurs have no desire to change their pattern of behavior. Since voyeurism involves non-consenting partners and is against the law in many jurisdictions, the possibility of embarrassment may deter some individuals.

Prevention

Most experts agree that providing guidance regarding behavior that is culturally acceptable will prevent the development of a paraphilia such as voyeurism. The origin of some instances of voyeurism may be accidental observation with subsequent sexual gratification. There is no way to predict when such an event and association will occur.

Members of society at large can reduce the incidence of voyeurism by drawing curtains, dropping blinds or closing window curtains. Reducing opportunities for voyeurism may reduce the practice.

See also Paraphilia; Exhibitionism

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American Medical Association. 515 N. State Street, Chicago, IL 60610. Telephone: (312) 464-5000. Web site: <http://www.ama-assn.org/>.

American Psychiatric Association. 1400 K Street NW, Washington, DC 20005. Telephone: (888) 357-7924. Fax: (202) 682-6850. Web site: <http://www.psych.org/>.

American Psychological Association. 750 First Street NW, Washington, DC, 20002-4242. Telephone: (800) 374-2721 or (202) 336-5500. Web site: <http://www.apa.org/>.

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W

WAIS *see* **Wechsler Adult Intelligence Scale**

Wechsler adult intelligence scale

Definition

The Wechsler adult intelligence scale (WAIS) is an individually administered measure of intelligence, intended for adults aged 16–89.

Purpose

The WAIS is intended to measure human intelligence reflected in both verbal and performance abilities. Dr. David Wechsler, a clinical **psychologist**, believed that intelligence is a global construct, reflecting a variety of measurable skills and should be considered in the context of the overall personality. The WAIS is also administered as part of a test battery to make inferences about personality and pathology, both through the content of specific answers and patterns of subtest scores.

Besides being utilized as an intelligence assessment, the WAIS is used in neuropsychological evaluation, specifically with regard to **brain** dysfunction. Large differences in verbal and nonverbal intelligence may indicate specific types of brain damage.

The WAIS is also administered for diagnostic purposes. Intelligence quotient (IQ) scores reported by the WAIS can be used as part of the diagnostic criteria for **mental retardation**, specific learning disabilities, and **attention-deficit/hyperactivity disorder** (ADHD).

Precautions

The Wechsler intelligence scales are not considered adequate measures of extremely high and low intelligence (IQ scores below 40 and above 160). The nature of the scoring process does not allow for scores outside of this range for test takers at particular ages. Wechsler himself was even more conservative, stressing that his scales were not appropriate for people with an IQ below 70 or above 130. Also, when administering the WAIS to people at extreme ends of the age range (below 20 years of age or above 70), caution should be used when interpreting scores. The age range for the WAIS overlaps with that of the **Wechsler Intelligence Scale for Children** (WISC) for people between 16 and 17 years of age, and it is suggested that the WISC provides a better measure for this age range.

Administration and scoring of the WAIS require an active test administrator who must interact with the test taker and must know test protocol and specifications. WAIS administrators must receive proper training and be aware of all test guidelines.

Description

The Wechsler **intelligence tests**, which include the WAIS, the WISC, and the WPPSI (Wechsler preschool and primary scale of intelligence), are the most widely used intelligence assessments and among the most widely used neuropsychological assessments. Wechsler published the first version of the WAIS in 1939, initially called the Wechsler-Bellevue. The newest version is the WAIS-III (the third edition, most recently updated in 1997). Since Wechsler's death in 1981, the Wechsler tests have been revised by the publisher, the Psychological Corporation.

The theoretical basis for the WAIS and the other Wechlser scales came from Wechsler's belief that intelligence is a complex ability involving a variety of skills. Because intelligence is multifaceted, Wechsler believed, a test measuring intelligence must reflect this multitude of skills. After dividing intelligence into two major types of skills—verbal and performance—Wechsler utilized the statistical technique of factor analysis to determine specific skills within these two major domains. These more specific factors formed the basis of the Wechsler subtests.

KEY TERMS

Factor Analysis—A statistical method for summarizing relationships between variables. With the WAIS, items that correlated highly with each other were considered to be part of certain factors underlying intelligence. These factors are the basis for the 14 WAIS subtests.

Indices—Scores based on performance in more than one area. On the WAIS, there are four index scores, each based on an individual's performance in more than one subtest.

Mean—The mathematical average of all scores in a set of scores. The WAIS has been standardized to have a mean of 100.

Percentile rank—The point at which a given percentage of people fall at or below the individual's test score being calculated. For example, if a person's test score was at the 60th percentile, 40% of other test takers received a higher score, while 60% received a score that was at or below that of the test taker.

Standard deviation—A measure of variability in a set of scores. The WAIS has been standardized to have a standard deviation of 15.

Standardization—The administration of a test to a sample group of people for the purpose of establishing scoring norms. Prior to the publication of each version of the WAIS, it is standardized.

The WAIS-III consists of 14 subtests and takes about 60–75 minutes to complete. The test is taken individually, with a test administrator present to give instructions. Each subtest is given separately, and proceeds from very easy items to very difficult ones. There is some flexibility in the administration of the WAIS—the administrator may end some subtests early if test takers seem to reach the limit of their capacity. Tasks on the WAIS include questions of general knowledge, traditional arithmetic problems, a test of vocabulary, completion of pictures with missing elements, arrangements of blocks and pictures, and assembly of objects.

The WAIS is considered to be a valid and reliable measure of general intelligence. When undergoing reliability and validity studies, other intelligence tests are often compared to the Wechsler scales. It is regularly used by researchers in many areas of psychology as a measure of intelligence. Research has demonstrated correlations between WAIS IQ scores and a variety of socioeconomic, physiological, and environmental characteristics.

The WAIS has also been found to be a good measure of both fluid and crystallized intelligence. Fluid intelligence refers to inductive and deductive reasoning, skills considered to be largely influenced by neurological and biological factors. In the WAIS, fluid intelligence is reflected in the performance subtests. Crystallized intelligence refers to knowledge and skills that are primarily influenced by environmental and sociocultural factors. In the WAIS, crystallized intelligence is reflected in the verbal subtests. Wechsler himself did not divide overall intelligence into these two types. However, the consideration of fluid and crystallized intelligence as two major categories of cognitive ability has been a focus for many intelligence theorists.

The Wechshler scales were originally developed and later revised using standardization samples. The samples were meant to be demographically representative of the United States population at the time of the standardization.

Results

The WAIS elicits three intelligence quotient scores, based on an average of 100, as well as subtest and index scores. WAIS subtests measure specific verbal abilities and specific performance abilities.

The WAIS elicits an overall intelligence quotient, called the full-scale IQ, as well as a verbal IQ and a performance IQ. The three IQ scores are standardized in such a way that the scores have a mean of 100 and a standard deviation of 15. Wechsler pioneered the use of deviation IQ scores, allowing test takers to be compared to others of different as well as the same age. WAIS scores are sometimes converted into percentile ranks. The verbal and performance IQ scores are based on scores on the 14 subtests. The 14 subtest scores have a mean of 10 and a standard deviation of three. The WAIS also elicits four indices, each based on a different set of subtests: verbal comprehension, perceptual organization, working memory, and processing speed.

The full-scale IQ is based on scores on all of the subtests and is a reflection of both verbal IQ and performance IQ. It is considered the single most reliable and valid score elicited by the WAIS. However, when an examinee's verbal and performance IQ scores differ significantly, the full-scale IQ should be interpreted cautiously.

The verbal IQ

The verbal IQ is derived from scores on seven of the subtests: information, digit span, vocabulary, arithmetic, comprehension, similarities, and letter-number sequenc-

ing. Letter-number sequencing is a new subtest added to the most recent edition of the WAIS (WAIS-III).

The information subtest is a test of general knowledge, including questions about geography and literature. The digit span subtest requires test takers to repeat strings of digits. The vocabulary and arithmetic subtests are general measures of a person's vocabulary and arithmetic skills. The comprehension subtest requires test takers to solve practical problems and explain the meaning of proverbs. The similarities subtest requires test takers to indicate the similarities between pairs of things. The letter-number sequencing subtest involves ordering numbers and letters presented in an unordered sequence. Scores on the verbal subtests are based primarily on correct answers.

The performance IQ

The performance IQ is derived from scores on the remaining seven subtests: picture completion, picture arrangement, block design, object assembly, digit symbol, matrix reasoning, and symbol search. Matrix reasoning and symbol search are new subtests and were added to the most recent edition of the WAIS (WAIS-III).

In the picture completion subtest, the test taker is required to complete pictures with missing elements. The picture arrangement subtest entails arranging pictures in order to tell a story. The block design subtest requires test takers to use blocks to make specific designs. The object assembly subtest requires people to assemble pieces in such a way that a whole object is built. In the digit symbol subtest, digits and symbols are presented as pairs and test takers then must pair additional digits and symbols. The matrix reasoning subtest requires test takers to identify geometric shapes. The symbol search subtest requires examinees to match symbols appearing in different groups. Scores on the performance subtests are based on both response speed and correct answers.

See also Stanford-Binet intelligence scales

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Tasks on the WAIS include questions of general knowledge, traditional arithmetic problems, a test of vocabulary, completion of pictures with missing elements, arrangements of blocks and pictures, and assembly of objects. The picture arrangement subtest (shown above) entails arranging pictures in order to tell a story. *(Laura Dwight/ CORBIS. Photo reproduced by permission.)*

Wechsler Intelligence Scale for Children

Definition

The Wechsler Intelligence Scale for Children, often abbreviated as WISC, is an individually administered measure of intelligence intended for children aged six years to 16 years and 11 months.

Purpose

The WISC is designed to measure human intelligence as reflected in both verbal and nonverbal (performance) abilities. David Wechsler, the author of the test, believed that intelligence has a global quality that reflects a variety of measurable skills. He also thought that it should be considered in the context of the person's overall personality.

The WISC is used in schools as part of placement evaluations for programs for gifted children and for children who are developmentally disabled.

In addition to its uses in intelligence assessment, the WISC is used in neuropsychological evaluation, specifically with regard to **brain** dysfunction. Large differences in verbal and nonverbal intelligence may indicate specific types of brain damage.

The WISC is also used for other diagnostic purposes. IQ scores reported by the WISC can be used as part

KEY TERMS

Crystallized intelligence—A type of intelligence that reflects knowledge and skills influenced by a person's sociocultural environment.

Factor analysis—A statistical method for summarizing relationships between variables.

Fluid intelligence—A type of intelligence that involves inductive and deductive reasoning ability.

Intelligence quotient (IQ)—A measurement of intelligence obtained by dividing a person's mental age (determined by level of performance on an age-graded test) by his or her chronological age and multiplying by 100. For example, a ten-year-old with a mental age of thirteen would have an IQ of 130.

Standardization—The administration of a test to a sample group of people for the purpose of establishing scoring norms.

of the diagnostic criteria for **mental retardation** and specific learning disabilities. The test may also serve to better evaluate children with **attention-deficit/hyperactivity disorder** (ADHD) and other behavior disorders.

Precautions

The Wechsler intelligence scales are not considered adequate measures of extreme intelligence (IQ scores below 40 and above 160). The scoring process does not allow for scores outside this range for test takers at particular ages. Wechsler himself was even more conservative, stressing that his scales were not appropriate for people with IQs below 70 or above 130. Despite this restriction, many people use the WISC as a measure of the intelligence of gifted children, who typically score above 130. The age range for the WISC overlaps with that of the **Wechsler Adult Intelligence Scale** (WAIS) for people between 16 and 17 years of age, but experts suggest that the WISC provides a better measure for people in this age range.

Administration and scoring of the WISC require a competent administrator who must be able to interact and communicate with children of different ages and must know test protocol and specifications. WISC administrators must receive training in the proper use of the instrument and demonstrate awareness of all test guidelines.

Description

The Wechsler **intelligence tests**, which include the WISC, the WAIS, and the WPPSI (Wechsler Preschool and Primary Scale of Intelligence), are the most widely used intelligence and neuropsychological assessments. The first version of the WISC was written in 1949 by David Wechsler. The newest version of the WISC is the WISC-III (Wechsler Intelligence Scale for Children-Third Edition, most recently updated in 1991). Since Wechsler's death in 1981, the tests have been revised by their publisher, the Psychological Corporation.

The theoretical basis for the WISC and the other Wechsler scales is Wechsler's belief that human intelligence is a complex ability involving a variety of skills. Because intelligence is multifaceted, Wechsler believed, a test measuring intelligence must reflect this diversity. After dividing intelligence into two major types of skills—verbal and performance—Wechsler used a statistical technique called factor analysis to determine which specific skills fit within these two major domains.

The current version of the WISC (the WISC-III) consists of 13 subtests and takes between 50 and 75 minutes to complete. The test is taken individually, with an administrator present to give instructions. Each subtest is given separately. There is some flexibility in the administration of the WISC—the administrator may end some subtests early if the test taker appears to have reached the limit of his or her capacity. Tasks on the WISC include questions of general knowledge, traditional arithmetic problems, English vocabulary, completion of mazes, and arrangements of blocks and pictures.

Children who take the WISC are scored by comparing their performance to other test takers of the same age. The WISC yields three IQ (intelligence quotient) scores, based on an average of 100, as well as subtest and index scores. WISC subtests measure specific verbal and performance abilities. The Wecshler scales were originally developed and later revised using standardization samples. The samples were meant to be representative of the United States population at the time of standardization.

The WISC is considered to be a valid and reliable measure of general intelligence in children. It is regularly used by researchers in many areas of psychology and child development as a general measure of intelligence. It has also been found to be a good measure of both fluid and crystallized intelligence. Fluid intelligence refers to inductive and deductive reasoning, skills that are thought to be largely influenced by neurological and biological factors. Fluid intelligence is measured by the performance subtests of the WISC. Crystallized intelligence refers to knowledge and skills that are primarily influenced by environmental and sociocultural factors. It is

measured by the verbal subtests of the WISC. Wechsler himself did not divide overall intelligence into these two types. The definition of fluid and crystallized intelligence as two major categories of cognitive ability, however, has been a focus of research for many intelligence theorists.

Verbal IQ

The child's verbal IQ score is derived from scores on six of the subtests: information, digit span, vocabulary, arithmetic, comprehension, and similarities.

The information subtest is a test of general knowledge, including questions about geography and literature. The digit span subtest requires the child to repeat strings of digits recited by the examiner. The vocabulary and arithmetic subtests are general measures of the child's vocabulary and arithmetic skills. The comprehension subtest asks the child to solve practical problems and explain the meaning of simple proverbs. The similarities subtest asks the child to describe the similarities between pairs of items, for example that apples and oranges are both fruits.

Performance IQ

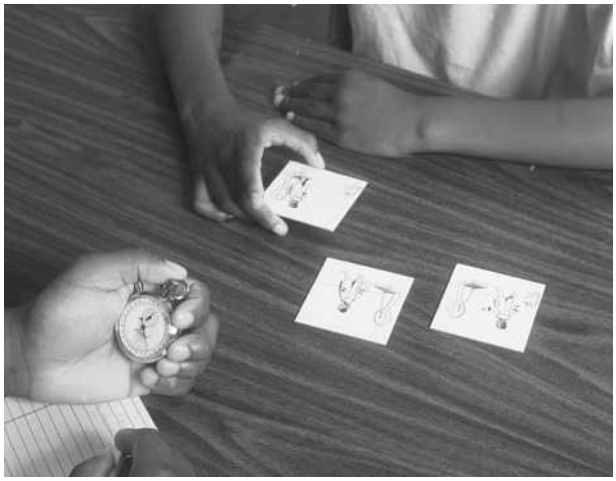
The child's performance IQ is derived from scores on the remaining seven subtests: picture completion, picture arrangement, block design, object assembly, coding, mazes, and symbol search.

In the picture completion subtest, the child is asked to complete pictures with missing elements. The picture arrangement subtest entails arranging pictures in order to tell a story. The block design subtest requires the child to use blocks to make specific designs. The object assembly subtest asks the child to put together pieces in such a way as to construct an entire object. In the coding subtest, the child makes pairs from a series of shapes or numbers. The mazes subtest asks the child to solve maze puzzles of increasing difficulty. The symbol search subtest requires the child to match symbols that appear in different groups. Scores on the performance subtests are based on both the speed of response and the number of correct answers.

Results

WISC scores yield an overall intelligence quotient, called the full scale IQ, as well as a verbal IQ and a performance IQ. The three IQ scores are standardized in such a way that a score of 100 is considered average and serves as a benchmark for higher and lower scores. Verbal and performance IQ scores are based on scores on the 13 subtests.

The full scale IQ is derived from the child's scores on all of the subtests. It reflects both verbal IQ and performance IQ and is considered the single most reliable and valid score obtained by the WISC. When a child's verbal and performance IQ scores are far apart, however, the full scale IQ should be interpreted cautiously.



A child taking the picture arrangement portion of the WISC. *(Lew Merrim/ Science Source/ Photo Researchers, Inc. Photo reproduced by permission.)*

See also Stanford-Binet intelligence scales

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Ali Fahmy, Ph.D.

Wellbutrin *see* **Bupropion**

Wernicke-Korsakoff syndrome

Definition

Wernicke-Korsakoff syndrome is a severe memory disorder usually associated with chronic excessive alcohol consumption, although the direct cause is a deficiency in the B vitamin thiamin.

KEY TERMS

Anterograde amnesia—Amnesia for events that occurred after a physical injury or emotional trauma but before the present moment.

Apathy—Lack of feelings or emotions.

Cognitive—Pertaining to the mental processes of memory, perception, judgment, and reasoning.

Encephalopathy—Brain disease that causes damage or degeneration.

Explicit memory—Consciously recalled memory for facts or events.

Implicit memory—Unconsciously recalled memory for skills, procedures, or associations.

Neurons—Nerve cells in the brain that produce nerve impulses.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Retrograde amnesia—Amnesia for events that occurred before a traumatic injury.

Serotonin—A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

Syndrome—A group of symptoms that together characterize a disease or disorder.

The *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR),* the professional handbook that aids clinicians in diagnosing patients' mental disorders, refers to Korsakoff syndrome as alcohol-induced persisting amnestic disorder and includes it under the category of substance-induced persisting **amnestic disorders**.

Description

The disorder was first identified in the late nineteenth century. The first phase of the condition, called Wernicke's encephalopathy, was described by German neurologist and **psychiatrist** Karl Wernicke in 1881. He noted three key symptoms in three patients—two with alcoholism and one who had swallowed sulfuric acid. These patients suffered from mental confusion, eye **movement disorders**, and ataxia (poor motor coordination). A few years later, S. S. Korsakoff, a Russian psychiatrist, began publishing reports describing a syndrome of anterograde amnesia—an inability to form new memories—and confabulation in individuals with severe alcoholism or certain medical illnesses. (Confabulation refers to the practice of filling in gaps in memory by fabrication.) By 1900, researchers and clinicians studying alcoholism recognized a connection between the two conditions. The typical syndrome begins with acute Wernicke's encephalopathy, with Korsakoff syndrome emerging when the acute phase resolves. The symptoms of Wernicke's encephalopathy appear suddenly. The most prominent symptom initially is mental confusion including memory problems. On examination, patients have difficulty moving their eyes to follow a visual stimulus due to paralysis of the muscles controlling eye movements. For instance, a patient may have trouble looking upward or to the side with one or both eyes. Problems maintaining balance while standing or walking, a condition known as ataxia, are frequently observed as well. If left untreated, most of these symptoms may resolve spontaneously, but the severe memory disorder characteristic of Korsakoff syndrome remains.

The typical person with Korsakoff syndrome appears fairly normal on first impression. Intelligence is intact, and individuals with the syndrome can carry on a conversation quite naturally. They are usually able to recall and talk about incidents that took place before the onset of the disorder and recognize family members and old friends without much difficulty. The ability to form new memories is nearly absent, however. In the course of conversation, people with Korsakoff syndrome may repeat comments or questions several times. They will fail to recognize people they met minutes before or greet a friend with excitement and surprise after a brief trip to another room. These are the characteristics of anterograde **amnesia**. Research shows that anterograde amnesia results from a failure of memory formation and storage. New information is processed normally, but almost immediately forgotten, never making it into the regions of the **brain** where memories of the past are stored. People with Korsakoff syndrome thus have no memories of events that happened after the onset of the illness. Many previously stored memories are still available, however, explaining why individuals with Korsakoff syndrome can usually remember the distant past quite well.

Causes and symptoms

Causes

Wernicke-Korsakoff syndrome is caused by thiamin deficiency. It is most commonly observed in

people with alcoholism since heavy drinkers often eat poorly, and alcoholism interferes with absorption of nutrients from the digestive system. It can also occur in people who are malnourished for other reasons. Thiamin helps produce energy needed to make neurons function properly. Insufficient thiamin can lead to damage or death of neurons.

Thiamin deficiency damages regions of the brain, particularly the thalamus and the mammillary bodies. The thalamus is a structure deep within the brain that serves many important functions. It is often called the major relay station of the brain, and many neurons make connections in the thalamus. The mammillary bodies are part of the hypothalamus, located just below the thalamus. The mammillary bodies receive many neural connections from another part of the brain called the hippocampus, which appears to be the primary part of the brain involved in the formation of memories. Neurons in the mammillary bodies make connections with the thalamus, which in turn makes connections with the cortex of the brain, where long-term memories are stored. This may explain why damage to the mammillary bodies and thalamus can lead to anterograde amnesia. Memories formed in the hippocampus are never stored since connections between hippocampus and cortex are disrupted.

Eye movement disorders observed in the acute phase of the condition are probably due to damage to other nearby brain regions that make connections to the nerves controlling eye muscles. These nerves emerge from the brainstem located right below the thalamus and mammillary bodies. Nerves involved in balance also make connections with other nerves in the brainstem, but a separate part of the brain called the cerebellum may also contribute to ataxia. Reasons why some regions of the brain are selectively affected by thiamin deficiency are not yet fully understood, but selective vulnerability of certain **neurotransmitters** is suspected.

Symptoms

Mental confusion, eye movement disturbances, and ataxia are the primary symptoms of Wernicke's encephalopathy—the first, acute stage of Wernicke-Korsakoff syndrome. At first glance, confusion and ataxia may resemble the effects of severe alcohol intoxication, but they persist after intoxication wears off. Some patients with Wernicke's encephalopathy will recover completely without residual memory deficits, particularly if they are treated quickly with thiamin.

The chronic stage of Wernicke-Korsakoff syndrome, sometimes called Korsakoff **psychosis**, is distinguished by anterograde amnesia, and most untreated patients with Wernicke's encephalopathy will develop this severe memory disorder, which prevents them from forming lasting memories of events or information encountered after the onset of the initial symptoms. Symptoms of Korsakoff syndrome may also develop spontaneously in many patients who never show signs of Wernicke's encephalopathy. Once patients develop Korsakoff's amnesia, recovery is unlikely.

Loss of memory for past events is called retrograde amnesia. Many people with Korsakoff syndrome have some retrograde amnesia in addition to anterograde amnesia, particularly for events that occurred shortly before the onset of illness, but most can recall the distant past without difficulty.

Immediate memory is not affected. For instance, an individual with Korsakoff syndrome could repeat a sentence or string of numbers immediately after hearing them, although this information would likely be forgotten within half a minute. Preservation of immediate memory allows individuals with Korsakoff syndrome to interact with others and respond to questions. Implicit memory is also preserved, so people with Korsakoff syndrome can learn new motor skills or develop conditioned reactions to stimuli. For example, individuals who play computer games can show improved performance each time they play, even if they cannot explicitly remember having played the game before.

Confabulation is another striking feature of Korsakoff syndrome, although it is not always observed. Confabulation refers to falsification of memory. The individual appears to be making up stories to cover up for inability to remember. Confabulation often seems to involve a confusion of the past and present. For example, if patients with Korsakoff syndrome are asked why they are in the hospital, they may say they just had a baby, are recovering from pneumonia, undergoing medical tests, or even applying for a job.

Patients with Wernicke-Korsakoff syndrome may also show signs of **apathy** and a lack of spontaneous behavior. Emotional expression may be lacking as well.

Interestingly, autopsies often reveal brain lesions characteristic of Wernicke-Koraskoff syndrome in alcoholic patients who showed general cognitive problems like those seen in **dementia**, but who never developed anterograde amnesia. These findings suggest that onset may be gradual in some patients.

Demographics

When **diagnosis** is based on postmortem findings, the estimated prevalence of Wernicke-Korsakoff syndrome is between 1 and 2% of the population. The clas-

sic presentation with acute onset of Wernicke's encephalopathy is fairly rare, about 0.05% of all hospital admissions, although this does not account for patients who do not seek medical attention. Wernicke-Korsakoff syndrome usually follows many years of chronic alcoholism or malnutrition and is seldom seen among people under 20. Most patients are 40 years of age or older. The disorder is apparently more common in alcoholic individuals who are particularly vulnerable to malnutrition such as indigent or homeless people.

Diagnosis

Wernicke's encephalopathy is diagnosed when patients seek medical attention and have the classic trio of signs: mental confusion, eye movement disorders, and ataxia. The diagnosis of Korsakoff syndrome is given when anterograde amnesia is present in an individual with a history of chronic, heavy drinking or malnutrition. When Korsakoff syndrome follows Wernicke's encephalopathy, the entire Wernicke-Korsakoff syndrome diagnosis is appropriate. The diagnosis is supported by neuroimaging or autopsy findings showing degeneration of the thalamus and mammillary bodies and loss of brain volume in the area surrounding the fourth ventricle—a fluid-filled cavity near the brainstem.

Although *DSM-IV-TR* criteria for alcohol-induced persisting amnestic disorder apply to most people with Wernicke-Korsakoff syndrome, there are some differences between the two diagnoses. Despite research findings suggesting that severe amnesia is not a necessary symptom of Wernicke-Korsakoff syndrome, the *DSM-IV-TR* requires the presence of either anterograde or retrograde amnesia for a diagnosis of alcohol-induced persisting amnestic disorder. One additional cognitive symptom is also required. Symptoms listed in the *DSM-IV-TR* include language disturbance (aphasia), inability to carry out motor activities (apraxia), inability to recognize objects (agnosia), or deficits in planning, initiation, organization and abstraction (executive functions). Individuals with Wernicke-Korsakoff syndrome frequently demonstrate problems with executive functions that contribute to the symptoms of confabulation and apathy. Aphasia, apraxia, and agnosia are not common signs of Wernicke-Korsakoff syndrome.

The *DSM-IV-TR* also requires that memory impairment must significantly impair a person's ability to perform normal activities and functions, and it must represent a decline from a previous level of functioning. Amnesia cannot occur exclusively during states of **delirium**, alcohol intoxication, or withdrawal, or be exclusively associated with a dementia. Both of the these requirements are consistent with the usual presentation of Wernicke-Korsakoff syndrome.

Finally, the *DSM-IV-TR* requires evidence that amnesia is caused by use of alcohol. Such evidence can include an extensive history of heavy drinking; or physical examination or laboratory findings revealing other signs of heavy alcohol use, such as abnormal liver function tests. Despite this *DSM-IV-TR* requirement, Wernicke-Korsakoff's syndrome can occur in the absence of heavy alcohol use. Emergence of the disorder in people without alcoholism is much less common today than it was in the past, however, since vitamins are now added to many foods. In practice, most people who show the hallmark symptoms of Wernicke-Korsakoff syndrome also qualify for the *DSM-IV-TR* diagnosis.

Treatments

Nutritional

Individuals with signs of Wernicke's encephalopathy should be treated with thiamin immediately. In many cases, prompt administration of thiamin reverses the symptoms and prevents amnesia from developing. Thiamin can be administered intravenously or directly into the digestive system. Unfortunately, thiamin is less effective in the chronic phase of the condition. Based on autopsy findings suggesting the presence of Wernicke-Korsakoff syndrome in people with milder cognitive problems who do not show the classic signs of the disorder, researchers have examined the usefulness of thiamin treatment in people with alcohol dependence who are at risk of developing the syndrome. Results suggest that thiamin treatment improves performance on memory tests in this group, and that higher thiamin doses are associated with better performance. These findings suggest that thiamin treatment can help prevent Wernicke-Korsakoff syndrome in heavy drinkers.

Medication

Recent reports suggest that **donepezil** and **rivastigmine**, drugs used to treat **Alzheimer's disease**, may improve memory in patients with Wernicke-Korsakoff syndrome. Both drugs prevent the breakdown of the neurotransmitter acetylcholine, which is important for the formation of memories. Patients treated with these drugs showed improvements on memory tests and were more able to recognize hospital staff and family members. Although improvements appear to be rather modest, these drugs may be useful for patients who do not respond to thiamin. Antidepressants that increase levels of serotonin may also be helpful, although the reasons why are not clear since these drugs are not effective with other memory disorders.

Conditioning

The fact that implicit memory is not affected by Wernicke-Korsakoff syndrome has led some researchers to explore the use of classical conditioning procedures in helping patients to remember specific people. In classical conditioning, animals and people learn to associate a stimulus with an outcome. The most famous example is the pairing of a ringing bell with food. Dogs naturally salivate when given food. In a famous experiment, Ivan Pavlov rang a bell immediately before serving food to dogs. After doing this repeatedly, Pavlov found that the dogs salivated upon hearing the bell ring even when the food was not presented. This form of learning does not rely on the hippocampus and cortex but appears to involve neurons in other parts of the brain. Patients with Wernicke-Korsakoff syndrome who are given specific rewards for correctly choosing a picture of a face that matches a face they have seen previously are more able to choose the correct face than those who do not receive the rewards. Although these individuals do not explicitly remember the face they saw previously, they are still able to make the correct choice. Training patients in this way could enable them to recognize familiar people and differentiate them from strangers.

Prognosis

The prognosis for full recovery from Wernicke-Korsakoff syndrome is poor. Once chronic Korsakoff's amnesia ensues, approximately 80% of patients will never fully recover the ability to learn and remember new information. Because they cannot learn from experience, individuals with Wernicke-Korsakoff syndrome almost always require some form of custodial care. They are usually unable to work, although some can perform simple tasks they learned prior to onset of the condition if closely supervised.

Prevention

Wernicke-Korsakoff syndrome can be prevented with a nutritious diet containing sufficient thiamin. Because severe chronic alcoholism is the most common cause of thiamin deficiency, treatment of alcohol dependence is extremely important. In order to prevent Wernicke-Korsakoff syndrome among people who are unable to stop drinking or among particularly vulnerable individuals like homeless drinkers, some researchers and clinicians have advocated supplementing alcoholic beverages with thiamin.

See also Alcohol and related disorders; Amnestic disorders; Brain; Dementia; Executive function

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ORGANIZATIONS

Family Caregiver Alliance. 690 Market Street, Suite 600, San Francisco, CA 94104. <http://www.caregiver.org/>.

Medical Council on Alcohol. 3 St. Andrew's Place, Regent's Park, London, UK NW1 4LB. <http://www.medicouncilalcol.demon.co.uk>.

National Institute on Alcohol Abuse and Alcoholism (NIAAA). Willco Building, 6000 Executive Boulevard, Bethesda, MD 20892. <http://www.niaaa.nih.gov>.

OTHER

Memory Loss and the Brain Newsletter, Memory Disorders Project, Rutgers University, 197 University Avenue, Newark, NJ 07102 <http://www.memorylossonline.com/>.

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Wide Range Achievement Test

Definition

Wide Range Achievement Test, 3rd ed. or WRAT-3 is a screening test that can be administered to determine if a more comprehensive achievement test is needed. Achievement tests refer to skills that individuals learn through direct instruction or **intervention**.

Purpose

The WRAT-3 measures basic skills in reading, arithmetic, and spelling. The test covers ages from five to 75 years old and takes approximately 30 minutes to administer.

Precautions

Although screening instruments may save time, these instruments can sometimes have misleading results. For instance, the scores may overestimate or underestimate a person's skills or the test does not measure other important achievement abilities. To obtain a more in-depth result of an examinee's abilities, a more comprehensive achievement test must be administered. For example, the WRAT-3 has no assessment of fundamental skills such as reading comprehension, writing abilities, and applying mathematical concepts to real-life situations. Finally, psychometric testing requires a clinically trained examiner. Therefore, the test should only be administered and interpreted by a trained examiner.

Description

The WRAT-3 has two alternative testing forms (tan and blue). One form is administered with the second form available if needed. Both testing forms (both the tan and blue forms) can be administered. When this is done, a combined scored is obtained. Each testing form consists of one reading test, one arithmetic test, and one spelling test. The reading test is administered individually, but the other two tests may be given in groups of up to five people. The reading test consists of 15 letters and 42 individual words that the examinee is asked to name or pronounce. The spelling test consists of writing one's name, 13 letters, and up to 40 words dictated to the examinee and used in a sentence. The spelling items increase with difficulty. Finally, the arithmetic test consists of two parts. Part I requires counting, reading number symbols, and solving simple arithmetic problems that are verbally presented to the examinee. Part II consists of using paper and a pencil to calculate up to 40 arithmetic problems within 15 minutes. These arithmetic problems are presented in a test booklet.

KEY TERMS

Normal curve equivalents—Standard scores with an average of 100. The normal curve equivalents divide the normal or bell-shaped curve into 100 equal parts. As a result, those scores can be used for statistical analysis because they can be added, subtracted, multiplied and divided.

Percentile ranks—The point at which a given percentage of people fall at or below the individual's test score being calculated. For example, if a person's test score was at the 60th percentile, 40% of other test takers received a higher score, while 60% received a score that was at or below that of the test taker.

Psychometric—Pertaining to testing and measurement of mental or psychological abilities. Psychometric tests convert an individual's psychological traits and attributes into a numerical estimation or evaluation.

Results

Scoring consists of a 1 for a correct answer and a 0 for an incorrect answer. The raw scores are converted to standard scores. These are scores that allow the examiner to compare the individual's score to other people who have taken the test. Additionally, by converting raw scores to standard scores the examiner has uniform scores and can more easily compare an individual's performance on one test with the individual's performance on another test. The average score for each test of the WRAT-3 is 100. An examiner can also obtain grade-equivalent scores, percentile ranks, and normal curve equivalents. A poor performance in any of the three areas assessed by this instrument can indicate the need for further testing.

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Keith Beard, Psy.D.

WISC *see* **Wechsler Intelligence Scale for Children**

X

Xanax *see* **Alprazolam**

Y

Yoga

Definition

Yoga is an ancient system of breathing practices, physical exercises and postures, and **meditation** intended to integrate the practitioner's body, mind, and spirit. It originated in India several thousand years ago, and its principles were first written down by a scholar named Patanjali in the second century B.C. The word *yoga* comes from a Sanskrit word, *yukti*, and means "union" or "yoke." The various physical and mental disciplines of yoga were seen as a method for individuals to attain union with the divine.

In the contemporary West, however, yoga is more often regarded as a beneficial form of physical exercise than as a philosophy or total way of life. As of 2002, more than six million people in the United States were practicing some form of yoga, with 1.7 million claiming to practice it regularly.

Purpose

Yoga has been recommended as an adjunct to **psychotherapy** and standard medical treatments for a number of reasons. Its integration of the mental, physical, and spiritual dimensions of human life is helpful to patients struggling with distorted cognitions or pain syndromes. The stretching, bending, and balancing involved in the asanas (physical postures that are part of a yoga practice) help to align the head and spinal column; stimulate the circulatory system, endocrine glands, and other organs; and keep muscles and joints strong and flexible. Yoga programs have been shown to reduce the risk of heart disease by lowering blood pressure and anxiety levels. The breath control exercises, known as *pranayama*, emphasize slow and deep abdominal breathing. They benefit the respiratory system, help to induce a sense of relaxation, and are useful in pain management. The meditation that is an integral part of classical yoga practice has been shown to strengthen the human immune system. Although Western medical researchers have been studying yoga only since the 1970s, clinical trials in the United States have demonstrated its effectiveness in treating asthma, osteoarthritis, heart disease, stress-related illnesses, high blood pressure, anxiety, and mood disorders. Other reports indicate that yoga merits further research in the treatment of **obsessive-compulsive disorder** (OCD) and substance abuse. Studies done in Germany have focused on the psychological benefits of yoga. One clinical trial done in 1994 at the University of Wurzburg found that the volunteer subjects who had practiced yoga scored higher in life satisfaction, with lower levels of irritability and pychosomatic complaints, than the control group.

One of the advantages of yoga as a complementary therapy is its adaptability to patients with a wide variety of physical and psychiatric conditions. There are a number of different schools of yoga—over 40, according to one expert in the field—and even within a particular school or tradition, the asanas and breathing exercises can be tailored to the patient's needs. One can find special yoga courses for children; for people over 50; for people with fibromyalgia, arthritis, or back problems; for cancer patients; and for people struggling with weight. Although most people who take up yoga attend classes, it is possible to learn the basic postures and breathing techniques at home from beginners' manuals or videotapes. Patients who feel self-conscious about exercising in the presence of others may find yoga appealing for this reason. The American Yoga Association has produced a manual and videotape for beginners, as well as a book called *The American Yoga Association's Easy Does It Yoga* for persons wih physical limitations. In addition, yoga does not require expensive equipment or special courts, tracks, or playing fields. An area of floor space about 6 ft by 8 ft, a so-called "sticky mat" to keep the feet from slipping, and loose clothing that allows the wearer to move freely are all that is needed.

KEY TERMS

Asana—The Indian term for the poses or postures that are done in sequence during hatha yoga practice.

Hatha yoga—The form of yoga most familiar to Westerners; often practiced as a form of physical therapy.

Pranayama—The breathing exercises that accompany the asanas in hatha yoga.

Yogi (feminine, yogini)—A person who is a respected expert in or teacher of yoga.

Woman in child's pose, a hatha yoga position. *(Photo Researchers, Inc. Reproduced by permission.)*

Precautions

Patients with a history of heart disease, severe back injuries, inner ear problems or other difficulties with balance, or recent surgery should consult a physician before beginning yoga. Pregnant women are usually advised to modify their yoga practice during the first trimester.

People diagnosed with a dissociative disorder should not attempt advanced forms of pranayama (yogic breathing) without the supervision of an experienced teacher. Some yogic breathing exercises may trigger symptoms of derealization or **depersonalization** in these patients.

Yoga should not be practiced on a full stomach. It is best to wait at least two hours after a meal before beginning one's yoga practice. In addition, while yoga can be practiced outdoors, it should not be done in direct sunlight.

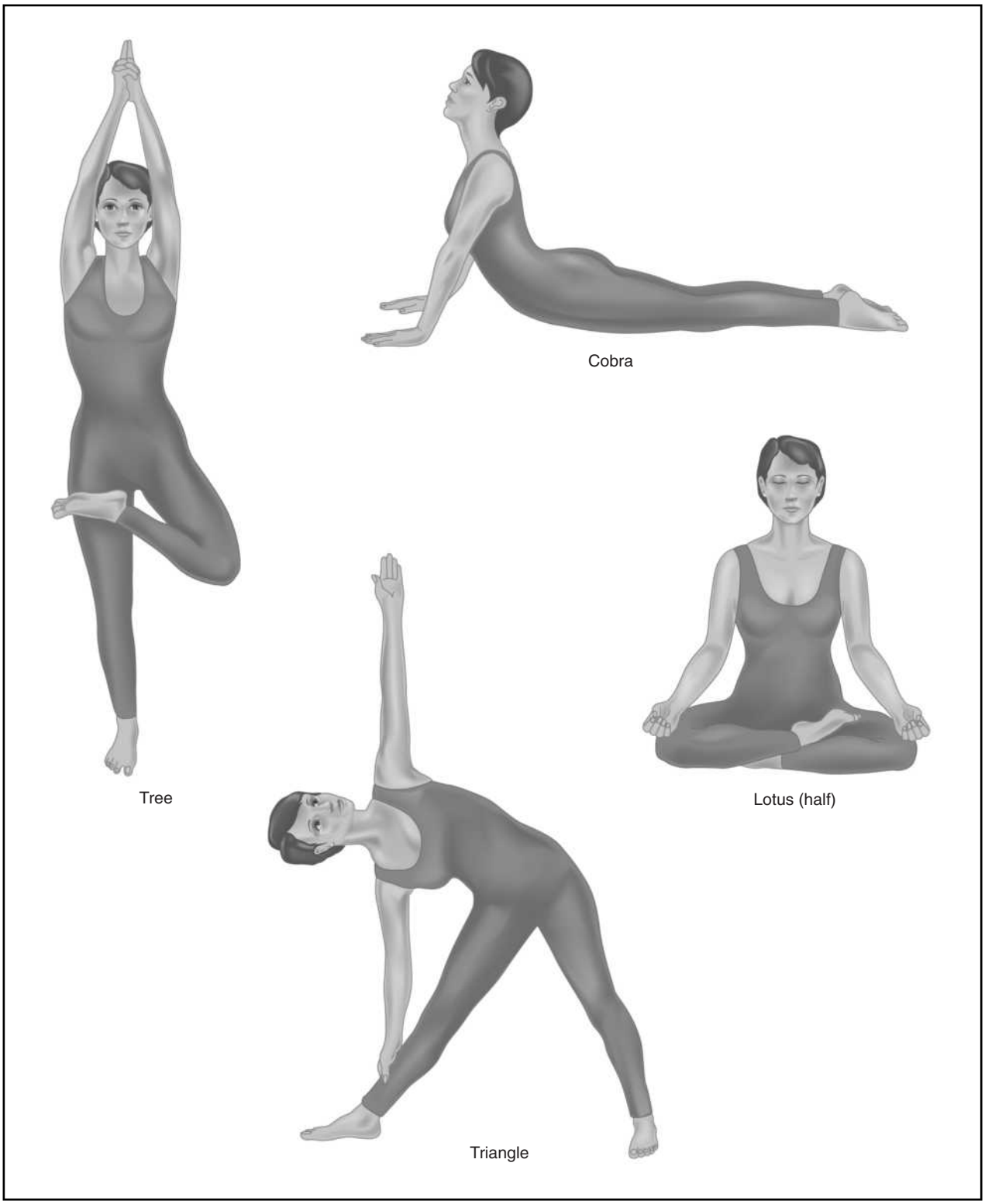
One additional precaution is often necessary for Westerners. Yoga is not a competitive sport, and a "good" practice is defined as whatever one's body and mind are capable of giving on a specific day. Westerners are, however, accustomed to pushing themselves hard, comparing their performances to those of others, and assuming that exercise is not beneficial unless it hurts—an attitude summed up in the phrase "no pain, no gain." Yoga teaches a gentle and accepting attitude toward one's body rather than a punishing or perfectionistic approach. A person should go into the stretches and poses gradually, not forcibly or violently. Stretching should not be done past the point of mild discomfort, which is normal for beginners; frank pain is a warning that the body is not properly aligned in the pose or that the joints are being overstressed. Most people beginning yoga will experience measurable progress in their strength and flexibility after a week or two of daily practice.

Description

There are six major branches of yoga: hatha, raja, karma, bhakti, jnana, and tantra yoga. Hatha yoga, the type most familiar to Westerners, will be discussed more fully in the following paragraph. Raja yoga is a spiritual path of self-renunciation and simplicity; karma yoga emphasizes selfless work as a service to others. Bhakti yoga is the path of cultivating an open heart and single-minded love of God. Jnana yoga is the sage or philosopher's approach; it cultivates wisdom and discernment, and is considered the most difficult type of yoga. Tantra yoga emphasizes transcending the self through religious rituals, including sacred sexuality.

Hatha yoga is the best-known form of yoga in the West because it is often taught as a form of physical therapy. A typical hatha yoga practice consists of a sequence of asanas, or physical poses, designed to exercise all parts of the body in the course of the practice. The asanas incorporate three basic types of movement: forward bends, backward bends, and twists. Practitioners of hatha yoga have over 200 asanas to choose from in creating a sequence for practice. The postures have traditional Indian names, such as Eagle Pose, Half Moon Pose, or Mountain Pose. There are steps for entering and leaving the pose, and the student is taught to concentrate on proper form and alignment. The pose is held for a period of time (usually 10–20 seconds), during which the practitioner concentrates on breathing correctly. Mental focus and discipline is necessary in order to maintain one's poise and balance in the asana. At the close of the practice, most students of yoga rest in a position that allows for a period of meditation. Most yoga practices take about an hour, although some are as short as 20 minutes.

There are a number of different styles of hatha yoga taught in the United States, the best known being Iyengar,



Demonstrations of the tree, cobra, and lotus yoga poses. The tree and triangle are good for balance and coordination. The cobra stretches the pelvic muscles and strengthens the back. Lotus is a meditative pose. *(Illustration by Electronic Illustrators Group.)*

Bikram, Kripalu, and ashtanga yoga. Iyengar yoga, which was developed by B.K.S. Iyengar, emphasizes attention to the details of a pose and the use of such props as blocks and belts to help students gain flexibility. Bikram yoga, taught on the West Coast by Bikram Choudhury, is practiced in heated rooms intended to make participants sweat freely as they warm and stretch their joints and muscles. Kripalu yoga, sometimes called the yoga of consciousness, emphasizes breathing exercises and the proper coordination of breath and movement. It also teaches awareness of one's psychological and emotional reactions to the various poses and movements of the body. Ashtanga yoga, developed by K. Pattabhi Jois, is the basis of so-called power yoga. Ashtanga yoga is a physically demanding workout that is not suitable for beginners.

Preparation

Good preparation for yoga requires spiritual and mental readiness as well as appropriate clothing and a suitable space. Many practitioners of yoga begin their practice with simple breathing exercises and stretches intended to clear the mind as well as open up the lungs.

Clothing should be comfortable and allow free movement. Some women prefer to practice in a dancer's leotard or similar garment made of stretchy fabric, but a simple tunic or beach cover-up worn over a pair of running shorts works just as well. Brassieres should not be worn during practice because they tend to restrict breathing. Men often practice in swim trunks or running shorts. Both men and women can use an oversize men's cotton T-shirt as a practice garment— these are inexpensive, easy to wash, and nonbinding. The feet are bare.

Aftercare

As was mentioned earlier, traditional hatha yoga practice ends the sequence of asanas with a pose in which meditation is possible, either sitting or lying flat on the back. Other than quiet resting, no particular aftercare is necessary.

Risks

Most reported injuries in yoga result from lack of concentration or attempts to perform difficult poses without working up to them. People who have consulted a physician before starting yoga and practice under the supervision of an experienced teacher are unlikely to suffer serious injury.

Normal results

Normal results following yoga practice are improved posture, lowered blood pressure, increased flexibility in the joints, higher energy levels, and a sense of relaxation.

Abnormal results

Abnormal physical results would include serious injuries to joints or muscles; abnormal psychological results would include dissociative episodes.

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ORGANIZATIONS

American Yoga Association. <www.americanyoga association.org>.

International Association of Yoga Therapists (IAYT). 4150 Tivoli Avenue, Los Angeles, CA 90066.

Yoga Research and Education Center (YREC). 2400A County Center Drive, Santa Rosa, CA 95403. (707) 566-0000. <www.yrec.org>.

Rebecca J. Frey, Ph.D.

Z

Zaleplon

Definition

Zaleplon is classified as a hypnotic drug. These drugs help people sleep. Zaleplon is available in the United States as the brand name drug Sonata.

Purpose

Zaleplon is a drug that is used to treat short-term **insomnia**, and it can be habit-forming.

Description

The United States Food and Drug Administration approved Zaleplon in 1999 to treat short-term problems sleeping. Zaleplon is thought to act by mimicking a chemical in the **brain** that helps to facilitate sleep. It is different from other sleeping pills, because it begins to work almost immediately and its effects are rather short-lived (a few hours). These properties make it beneficial both for people who have troubling falling asleep at bedtime and for people who awaken in the middle of the night and have trouble falling back to sleep. Zaleplon may be taken in the middle of the night so long as the person can sleep at least four more hours before having to awaken.

Zaleplon is available as capsules. The drug is broken down by the liver. It is a controlled substance and can be habit-forming.

Recommended dosage

The usual dose of zaleplon for adults is 5–20 mg. For healthy adults, 10 mg is a common dosage. However, people over age 65, small adults with low body weight, and people with serious health problems (especially liver disease) should take a dose at the low end of this range (usually 5 mg). Zaleplon is taken immediately before bedtime. It usually takes only about 30 minutes for the sleep-inducing actions of zaleplon to be felt, and sleep-facilitating effects appear to last only a few hours. If zaleplon is taken with a meal, it will take longer to work. For the fastest sleep onset, it should be taken on an empty stomach. The maximum dose for one day is 20 mg. Under no circumstances should a person take more than 20 mg in one day.

Precautions

Zaleplon can be habit-forming and should be taken exactly as directed by a physician. A person who forgets a dose of zaleplon should skip the dose and take the next dose at the regularly scheduled time.

Because zaleplon is used to help people fall asleep, it should not be used with other drugs (over-the-counter or prescription) that also cause drowsiness. Zaleplon should be used only with close physician supervision in people with liver disease and in the elderly, because these individuals are especially sensitive to the sedative properties of zaleplon. Zaleplon should not be used before driving, operating machinery, or performing activities that require mental alertness. People with a history of drug abuse, psychiatric disorders, or depression should be carefully monitored when using zaleplon since zaleplon may worsen symptoms of some psychiatric disorders and can become a drug of abuse.

If zaleplon is needed for more than seven to ten days, patients should be re-evaluated by a physician to determine if another disorder is causing their difficulty sleeping. When zaleplon or other sleeping pills are used every night for more than a few weeks, they begin to lose their effectiveness and/or people may become dependent upon them to fall asleep. Zaleplon can be addictive. People using zaleplon should not stop taking the drug suddenly because withdrawal symptoms, including sleep disturbances, may occur even if zaleplon has been used only for a short time.

KEY TERMS

Amnesia—A general medical term for loss of memory that is not due to ordinary forgetfulness. Amnesia can be caused by head injuries, brain disease, or epilepsy as well as by dissociation.

Antidepressant—A medication used to treat the symptoms of depression.

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Antipsychotic—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Hypnotic—A type of medication that induces sleep.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals approximately 0.035 ounces.

Tuberculosis—An infection caused by the bacteria *Mycobacterium tuberculosis* that usually affects the lungs. Individuals with tuberculosis may have nighttime sweating, fever, weight loss, cough, and may spit up blood and mucus.

Side effects

Some sleeping pills such as zaleplon can cause aggressiveness, agitation, **hallucinations**, and **amnesia** (memory problems). A patient experiencing these side effects should call a physician immediately. A physician should also be called immediately if a person taking zaleplon develops a fast or irregular heartbeat, chest pains, skin rash, or itching.

The most common side effects of zaleplon are less serious and include dizziness, drowsiness, impaired coordination, upset stomach, nausea, headache, dry mouth, and muscle aches. Other side effects that may occur include: fever, amnesia, tremor, or eye pain. Many side effects appear worse at higher doses, so it is important to use the lowest dose that will induce sleep.

Interactions

Any drug that causes drowsiness may lead to substantially decreased mental alertness and impaired motor skills when taken with zaleplon. Some examples include alcohol, antidepressants such as **imipramine** or **paroxetine**, antipsychotics like **thioridazine**, and some antihistamines.

Because zaleplon is broken down by the liver, it may interact with other drugs broken down by the liver. For example, the drug rifampin, which is used to treat tuberculosis, may cause zaleplon to be less effective. Alternatively, cimetidine (Tagamet), a drug commonly used to treat heartburn, may cause people to be more sensitive to zaleplon.

Resources

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Kelly Karpa, RPh, PhD

Ziprasidone

Definition

Ziprasidone is a drug used to treat **schizophrenia**. It is available with a prescription under the brand name Geodan.

Purpose

Ziprasidone is in a class of drugs called antipsychotics. It is used to control symptoms of schizophrenia.

Description

The United States Food and Drug Administration approved ziprasidone for treatment of schizophrenia in 2001. Mental well-being is partially related to maintaining a balance between naturally occurring chemicals in the **brain** called **neurotransmitters**. Ziprasidone is thought to modify the actions of several neurotransmitters and in this way restore appropriate function to chemical systems in the brain that are out of balance in people with schizophrenia.

Recommended dosage

The dosage of ziprasidone varies widely from one individual to another. A common initially dosage is 20 mg of ziprasidone taken twice daily. The dosage is gradually increased until symptoms of schizophrenia subside.

Dosages of up to 100 mg may be taken twice daily. Ziprasidone should be taken with food.

Precautions

Ziprasidone may alter the rhythm of the heart. Because of the risk of irregular heartbeats or even death, it should not be taken by people with a history of irregular or prolonged heart rhythms (long QT syndrome), those with heart failure, or individuals who have recently had a heart attack. People with a history of heart disease should discuss the risks and benefits of treatment with their doctor before starting ziprasidone. Ziprasidone may lower blood pressure to dangerously low levels, causing people to faint. It should not be taken by people who have slow heartbeats and those with low levels of potassium or magnesium in their blood.

Individuals with a history of seizure, even seizure brought on by drug or alcohol abuse, should use ziprasidone cautiously and with close physician supervision, because it may increase the tendency to have **seizures**.

Ziprasidone may increase body temperatures to dangerously high levels. People who exercise strenuously, those exposed to extreme heat, individuals taking drugs with anticholinergic effects (this includes many common antidepressants), and persons prone to dehydration, should use the drug cautiously and be alert to dehydration-related side effects. Elderly persons with increased risk of developing pneumonia should be carefully monitored while taking ziprasidone. Because there is a high incidence of **suicide** in all patients with psychotic illnesses, people using ziprasidone should be observed carefully for signs of suicidal behavior. Women who are pregnant or breast-feeding should not take ziprasidone.

Side effects

The most common reason that ziprasidone is stopped is due to development of a rash. Another common side effect is drowsiness. This side effect is usually worse when starting the drug and becomes less severe with continued use. People performing tasks that require mental alertness such as driving or operating machinery should refrain from doing so until they see how the drug affects them. Other side effects that may occur are abnormal, involuntary twitching (5%), and respiratory disorders (8%). Nausea, constipation, indigestion, and dizziness due to low blood pressure occur in more than 5% of people taking ziprasidone.

Other, less common, side effects are rapid heartbeats, low blood pressure, agitation, tremor, confusion, **amnesia**, dry mouth, increased salivation, joint pains, and abnormal vision.

The incidence of some adverse effects such as low blood pressure, anorexia, abnormal involuntary movements, sleepiness, tremor, cold symptoms, rash, abnormal vision, dry mouth or increased salivation appears to increase at higher dosages.

People taking ziprasidone should alert their health care provider immediately if they develop a rash or hives since this could indicate a potentially serious adverse reaction. Patients should also notify their health care provider immediately if they experience any abnormal involuntary muscle movements. People who think they may be experiencing any side effects from this or any other medication should talk to their physicians.

Interactions

Ziprasidone interacts with many other drugs. It is a good idea to review all medications being taken with a physician or pharmacist before starting this drug. Since ziprasidone may alter the rhythm of the heart, people who are also taking drugs such as quinidine, dofetilide, **pimozide**, sotalol, erythromycin, **thioridazine**, moxifloxacin, and sparfloxacin should not take it. These drugs may also affect properties of the heart and taken with ziprasidone increase the risk of irregular heart rhythms

KEY TERMS

Anticholinergic—Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

Neurotransmitter—A chemical in the brain that transmits messages between neurons, or nerve cells.

Schizophrenia—A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

and other cardiac problems. Because ziprasidone causes sleepiness, it should be used sparingly and with care with other drugs that also have a tendency to make people drowsy such as antidepressants, antihistamines, some pain relievers, and alcohol. Ziprasidone may lower blood pressure to the point at which people feel dizzy or faint. People taking medication to regulate their blood pressure should have their blood pressure monitored and treatment modified as needed. Ziprasidone may also decrease the effects of drugs used to treat Parkinson's disease such as levodopa.

Other drugs taken in combination with ziprasidone may alter the effects of ziprasidone. For example, drugs such as **carbamazepine**, used to treat seizures, increases liver metabolism and may cause ziprasidone to be less effective. Alternatively, drugs such as ketoconazole slow liver metabolism and may increase negative side effects associated with ziprasidone.

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Kelly Karpa, RPh, Ph.D.

Zoloft *see* **Sertraline**

Zolpidem

Definition

Zolpidem is classified as a hypnotic drug. These drugs help people sleep. In the United States, zolpidem is available as tablets under the brand name of Ambien.

Purpose

Zolpidem is a drug that is used to treat **insomnia**. Zolpidem is especially helpful for people who have trouble falling asleep. However, once individuals have fallen asleep, zolpidem also helps them continue to sleep restfully. Zolpidem should be used only for short periods, approximately seven to ten days. If sleeping pills are needed for a long period, an evaluation by a physician is recommended to determine if another medical condition is responsible for the insomnia.

Description

Although the way zolpidem helps people sleep is not entirely understood, it is believed to mimic a chemical in the **brain** called gamma-aminobutyric acid (GABA) that naturally helps to facilitate sleep. Zolpidem is a central nervous system depressant. This means that it slows down the nervous system. Unlike some sleeping pills, zolpidem does not interfere with the quality of sleep or usually leave the user feeling sedated in the morning. As a result, most people using zolpidem usually awake feeling refreshed in the morning.

Recommended dosage

The usual dose of zolpidem in adults is 5–10 mg. For healthy adults, 10 mg is commonly recommended. However, people taking other drugs that cause drowsiness, people who have severe health problems, especially liver disease, and older people (over age 65) should take a lower dose, usually 5 mg. Zolpidem should be taken immediately before bedtime and only if the person can count on getting seven or eight hours of uninterrupted sleep. It usually takes only about 30 minutes for the sleep-inducing actions of zolpidem to be felt. Unlike some sleeping pills, the sleep-facilitating effects appear to last six to eight hours.

If zolpidem is taken with a meal, it will take longer to work. For the fastest sleep onset, it should be taken on an empty stomach. The maximum dose for one day is 10 mg. People who miss a dose of zolpidem should skip the missed dose, and take the next dose at the regularly scheduled time. Under no circumstances should a person take more than 10 mg in one day. Zolpidem should be taken exactly as directed by the prescribing physician.

Precautions

Because zolpidem is used to help people fall asleep, it should not be used with other drugs (either over-the-counter, herbal, or prescription) that also cause drowsiness (for example, antihistamines or alcohol). Zolpidem should be used only with close physician supervision in people with liver disease and in the elderly, because these individuals are especially sensitive to the sedative properties of zolpidem. Zolpidem should not be used before driving, operating machinery, or performing activities that require mental alertness. People with a history of drug abuse, psychiatric disorders, or depression should be carefully monitored when using zolpidem since zolpidem may worsen symptoms of some psychiatric disorders.

If zolpidem is needed for more than seven to ten days, patients should be re-evaluated by a physician to determine if another disorder is causing their difficulty

KEY TERMS

Amnesia—A general medical term for loss of memory that is not due to ordinary forgetfulness. Amnesia can be caused by head injuries, brain disease, or epilepsy as well as by dissociation.

Antidepressant—A medication used to treat the symptoms of depression.

Antihistamine—A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

Antipsychotic—A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions and delirium. May be used to treat symptoms in other disorders, as well.

Milligram (mg)—One-thousandth of a gram. A gram is the metric measure that equals approximately 0.035 ounces.

Tuberculosis—An infection caused by the bacteria *Mycobacterium tuberculosis* that usually affects the lungs. Individuals with tuberculosis may have nighttime sweating, fever, weight loss, cough, and may spit up blood and mucus.

sleeping. When zolpidem or other sleeping pills are used every night for more than a few weeks, they begin to lose their effectiveness and/or people may become dependent upon them to fall asleep. Zolpidem can be habit-forming when taken over a long period. People using zolpidem should not stop taking the drug suddenly, but gradually reduce the dose over a few days before quitting, even if zolpidem has been used only a for short time.

Side effects

Some sleeping pills such as zolpidem can cause aggressiveness, agitation, **hallucinations**, and **amnesia** (memory problems), rapid, racing heartbeat, and chest pains. These side effects are rare, but the patient should call a physician immediately if they occur.

Side effects that occur in more than 5% of patients are headache, nausea, muscle aches, and drowsiness. Although drowsiness is desired when trying to fall asleep, a few people continue to be drowsy the next day. Daytime drowsiness may cause people, especially the elderly, to be less coordinated and more susceptible to falls. Other less common side effects are anxiety, confusion, dizziness, and stomach upset.

Interactions

Any drug that causes drowsiness may lead to substantially decreased mental alertness and impaired motor skills when taken with zolpidem. Some examples include alcohol, antidepressants such as **imipramine** or **paroxetine**, antipsychotics such as **thioridazine**, and antihistamines (commonly found in allergy and cold medications).

The effectiveness of zolpidem may be reduced if taken with rifampin, an antibiotic that is commonly used to treat tuberculosis infections.

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Kelly Karpa, RPh, Ph.D.

Zyprexa *see* **Olanzapine**

SYMPTOMS LIST

The following list of symptoms is intended *not* for diagnosis, but to reveal patterns in symptoms and disorders and to provide a starting point for research or discussion with a health care provider. Not every symptom with all of its accompanying disorders could be included.

A

Admiration, need for
- narcissistic personality disorder

Aggression
- Alzheimer's disease
- antisocial personality disorder
- borderline personality disorder
- conduct disorder
- intermittent explosive disorder
- may occur with tic disorders
- may occur with vascular dementia
- oppositional defiant disorder
- paranoid personality disorder
- substance intoxication

Agitation
- Alzheimer's disease
- major depressive disorder
- substance use, abuse, or dependence

Agoraphobia
- may occur with panic disorder

Amenorrhea (loss of menstrual periods)
- anorexia nervosa
- bulimia nervosa

Amnesia (memory loss)
- acute stress disorder
- Alzheimer's disease
- dissociative amnesia
- dissociative fugue
- dissociative identity disorder
- post-traumatic stress disorder
- vascular dementia
- Wernicke-Korsakoff syndrome

Anxiety
- acute stress disorder
- adjustment disorder
- agoraphobia
- borderline personality disorder
- generalized anxiety disorder
- may occur with tic disorders
- nightmare disorder
- pain disorder
- panic disorder
- schizotypal personality disorder
- separation anxiety disorder
- sleep terror disorder
- social phobia
- specific phobias
- substance abuse
- substance intoxication

Apathy
- dysthymic disorder
- feeding disorder of infancy or early childhood
- Internet addiction
- major depressive disorder
- may occur with Wernicke-Korsakoff syndrome
- schizoaffective disorder
- schizoid personality disorder
- schizophrenia
- schizotypal personality disorder

Appetite, loss of
- major depressive disorder
- postpartum depression
- seasonal affective disorder
- substance dependence
- undifferentiated somatoform disorder

Arousal, sexual
- from receiving pain: sexual masochism
- from administering pain: sexual sadism

Attention difficulties
- attention-deficit/hyperactivity disorder
- major depressive disorder
- nightmare disorder
- post-traumatic stress disorder

Attention-seeking behavior
- histrionic personality disorder
- narcissistic personality disorder

Aversion to sex
- hypoactive sexual desire disorder
- sexual aversion disorder

Avoidance
- acute stress disorder
- agoraphobia
- of conflict or disagreement: dependent personality disorder
- of pleasure: schizoid personality disorder; schizotypal personality disorder
- of relationships: schizoid personality disorder; schizotypal personality disorder
- of sex: hypoactive sexual desire disorder; schizoid personality disorder; sexual aversion disorder
- of social situations: avoidant personality disorder; seasonal affective disorder; separation anxiety disorder; may occur with tic disorders
- of specific feared situations: social phobia
- of specific feared situations or objects: specific phobias

B

Binge eating, followed by purging
- bulimia nervosa

Bizarre behavior
- brief psychotic disorder
- delusional disorder
- schizoaffective disorder
- schizophrenia
- schizophreniform disorder

C

D

E

F

G

H

I

L

M

Manic episode
  bipolar disorder
Math, difficulties with
  mathematical disorder
Medical history, vague or inconsistent
  factitious disorder
Memory impairment
  substance abuse
  substance dependence
  substance intoxication
  See also amnesia
Mental clarity. *See* thinking impairments
Misinterpretation of events on regular basis
  schizotypal personality disorder
Mixed episode
  bipolar disorder
  cyclothymic disorder
Mood, abrupt changes in
  cyclothymic disorder
Movement difficulties
  Alzheimer's disease
  developmental coordination disorder
  slowed movements: major depressive disorder
  See also coordination impairment
Movement, involuntary
  medication-induced movement disorder
  tardive dyskinesia
  tic disorders
  vaginismus
Muscle, loss of control
  narcolepsy

N

Nausea
  agoraphobia
  bulimia nervosa
  social phobia
  somatization disorder
  substance intoxication
Nightmares
  nightmare disorder
  post-traumatic stress disorder
  separation anxiety disorder
Normal development, then loss of skills
  childhood disintegrative disorder
Numbers, difficulty with
  mathematical disorder

O

Object needed for sexual arousal
  fetishism
Obsessions
  obsessive-compulsive disorder
Odd beliefs
  schizotypal personality disorder
Orgasm difficulties
  female orgasmic disorder
  male orgasmic disorder

P

Pain
  administering: sexual sadism
  during sexual intercourse: dyspareunia; female sexual arousal disorder; somatization disorder; vaginismus
  pain disorder
  receiving: sexual masochism
  undifferentiated somatoform disorder
Panic attacks
  may occur with tic disorders
  panic disorder
  substance use; substance abuse
Paranoia
  borderline personality disorder
  paranoid personality disorder
  schizotypal personality disorder
  substance abuse
Perfectionism
  obsessive-compulsive personality disorder
Personal care difficulties
  Alzheimer's disease
Preoccupation with fears of serious physical illness or injury
  hypochondriasis
Preoccupation with others' reactions
  social phobia
Preoccupation with particular body part
  body dysmorphic disorder
Preoccupation with seeking and using substance
  Substance dependence
Preoccupation with weight/ body image
  anorexia nervosa
  bulimia nervosa
Producing symptoms deliberately
  malingering

R

Reading difficulties
  reading disorder
Recognition difficulties
  Alzheimer's disease
  vascular dementia
Regurgitation
  rumination disorder
Relationship problems. *See* interpersonal problems
Repetitive movements
  autism
  childhood disintegrative disorder
  Rett's disorder
  stereotypic movement disorder
Responsibilities, failure to live up to
  pathological gambling
  substance abuse
  substance dependence
Restlessness
  attention-deficit/hyperactivity disorder
  generalized anxiety disorder
Rigidity, muscular
  medication-induced movement disorder
  Rett's disorder
  See also inflexibility
Risk-taking behaviors
  substance abuse
Rule violations
  conduct disorder

S

Sadness. *See* depression
Scars, multiple
  from surgery: factitious disorder
  on hand from self-induced vomiting: bulimia nervosa
Seductive behavior

T

U

V

W

GLOSSARY

A

ABSENCE SEIZURE. An epileptic seizure characterized by a sudden, momentary loss of consciousness, occasionally accompanied by some minor, jerky movements in the neck or upper arms, a twitching of the face, or a loss of muscle tone.

ABSTINENCE. Refraining from sexual intercourse for a period of time; may also refer to refraining from use of a substance, such as alcohol.

ABSTRACTION. Ability to think about concepts or ideas separate from specific examples.

ABUSE. Substance abuse is a milder form of addiction than substance dependence. Generally, people who have been diagnosed with substance abuse don't experience the tolerance or withdrawal symptoms—the signs of physiological dependence—that people dependent on a substance experience.

ACETYLCHOLINE. A naturally occurring chemical in the body that transmits nerve impulses from cell to cell. Generally, it has opposite effects from dopamine and norepinephrine; it causes blood vessels to dilate, lowers blood pressure, and slows the heartbeat. Central nervous system well-being is dependent on a balance among acetylcholine, dopamine, serotonin, and norepinephrine.

ACETYLCHOLINESTERASE. The chemical responsible for the breakdown of acetylcholine.

ACTIVE COPING STRATEGIES. Ways of handling stress that affect the problem or situation in some way.

ACUTE PSYCHOSIS. A severe mental disorder marked by delusions, hallucinations, and other symptoms that indicate that the patient is not in contact with reality.

ACUTE STRESS DISORDER. Symptoms occuring in an individual following a traumatic event to oneself or surrounding environment. Symptoms include a continued response of intense fear, helplessness, or terror within four weeks of the event, extreme nervousness, sleep disorders, increased anxiety, poor concentration, absence of emotional response to surroundings, and sometimes a dissociative amnesia—not recalling the significance of the trauma. Symptoms last a minimum of two days and maximum of four weeks. Can become post-traumatic stress disorder.

ADAPTOGEN. A remedy that helps the body adapt to change, and thus lowers the risk of stress-related illnesses.

ADDICTION. A compulsive need for, and use of, a habit-forming substance or behavior.

ADDICTIVE DISORDER. A disorder involving repetitive participation in a certain activity, in spite of negative consequences and despite attempts to stop the behavior. Alcohol abuse is an example.

ADDISON'S DISEASE. Disease caused by malfunctioning adrenal glands that can be treated with cortisol replacement therapy. Symptoms include anemia, low blood pressure, digestive complaints, and diarrhea.

ADENOSINE. A compound that serves to modulate the activities of nerve cells (neurons) and to produce a mild sedative effect when it activates certain types of adenosine receptors. Caffeine is thought to produce its stimulating effect by competing with adenosine for activation of these receptors.

ADJUNCT. A form of treatment that is not strictly necessary to a therapy regimen but is helpful.

ADJUSTMENT DISORDER. A disorder defined by the development of significant emotional or behavioral symptoms in response to a stressful event or series of events. Symptoms may include depressed mood, anxiety, and impairment of social and occupational functioning.

ADRENAL GLAND. A small organ located above each kidney that produces hormones related to the sex drive.

ADRENALINE. Another name for epinephrine, the hormone released by the adrenal glands in response to

stress. It is the principal blood pressure-raising hormone and a bronchial and intestinal smooth muscles relaxant.

AEROSOL. A liquid substance sealed in a metal container under pressure with an inert gas that propels the liquid as a spray or foam through a nozzle.

AFFECT. The expression of emotion displayed to others through facial expressions, hand gestures, tone of voice, etc. Types of affect include: flat (inanimate, no expression), blunted (minimally responsive), inappropriate (incongruous expressions of emotion relative to the content of a conversation), and labile (sudden and abrupt changes in type and intensity of emotion).

AFFECTIVE DISORDER. A disorder involving extreme emotional experience that is not congruent with the environmental circumstances (for example, feeling sad when there is no easily identifiable reason, as in depression).

AGE-ASSOCIATED MEMORY IMPAIRMENT (AAMI). A condition in which an older person suffers some memory loss and takes longer to learn new information. AAMI is distinguished from dementia in that it is not progressive and does not represent a serious decline from the person's previous level of functioning. Benign senescent forgetfulness is another term for AAMI.

AGITATION. Excessive restlessness or emotional disturbance often associated with anxiety or psychosis. Agitation may be associated with middle-stage Alzheimer's disease.

AGNOSIA. Loss of the ability to recognize familiar people, places, and objects.

AGONIST. A chemical that reproduces the mechanism of action of a neurotransmitter.

AGORAPHOBIA. People with this condition worry that they will not be able to get help or flee a place if they have a panic attack and may refuse to go to places that might trigger a panic attack.

AGRANULOCYTOSIS. A blood disorder characterized by a reduction in the number of circulating white blood cells (granulocytes). White blood cells defend the body against infections. Agranulocytosis is a potential side effect of some of the newer antipsychotic medications used to treat schizophrenia.

AKATHISIA. Agitated or restless movement, usually affecting the legs. Movement is accompanied by a sense of discomfort and an inability to sit, stand still, or remain inactive for periods of time. Akathisia is a common side effect of some neuroleptic (antipsychotic) medications.

AKINESIA. Absence of physical movement.

ALBUMIN. A simple protein that is widely distributed in human blood.

ALLOSTASIS. The process of an organism's adaptation to acute stress.

ALOPECIA. Hair loss (also, loss of feathers or wool in animals).

ALTER. An alternate or secondary personality in a person with dissociative identity disorder. Each alter has a unique way of looking at and interacting with the world.

ALVEOLAR. Pertaining to alveoli, which are tiny air sacs at the ends of the small air passages in the lungs.

ALZHEIMER'S DISEASE. An incurable dementia marked by the loss of cognitive ability and memory over a period of 10–15 years. Usually affects elderly people.

AMBULATION. Ability to walk.

AMENORRHEA. Absence of menstrual periods.

AMINO ACID. A building block of protein.

AMNESIA. A general medical term for loss of memory that is not due to ordinary forgetfulness. Amnesia can be caused by head injuries, brain disease, or epilepsy, as well as by dissociation.

AMNIOCENTESIS. A test usually done between 16 and 20 weeks of pregnancy to detect any abnormalities in the development of the fetus. A small amount of the fluid surrounding the fetus (amniotic fluid) is drawn out through a needle inserted into the mother's womb. Laboratory analysis of this fluid can detect various genetic defects, such as Down syndrome, or neural tube defects.

AMOTIVATIONAL SYNDROME. Loss of ambition associated with chronic cannabis (marijuana) use.

AMPHETAMINE ABUSE. An amphetamine problem in which the user experiences negative consequences from the use, but has not reached the point of dependence.

AMPHETAMINE DEPENDENCE. The most serious type of amphetamine problem.

AMPHETAMINE INTOXICATION. The effects on the body that develop during or shortly after amphetamine use.

AMPHETAMINE WITHDRAWAL. Symptoms that develop shortly after reducing or stopping heavy amphetamine use.

AMPHETAMINES. A group of powerful and highly addictive substances that stimulate the central nervous system. May be prescribed for various medical conditions, but are often purchased illicitly and abused.

AMYGDALA. An almond-shaped brain structure in the limbic system that is activated in stressful situations to trigger the emotion of fear. It is thought that the emotional overreactions in Alzheimer's patients are related to the destruction of neurons in the amygdala.

AMYLOID. A waxy translucent substance composed mostly of protein, that forms plaques (abnormal deposits) in the brain during the progression of Alzheimer's disease.

ANALEPTIC. A substance that acts as a stimulant of the central nervous system. Caffeine is classified as an analeptic.

ANALGESIC. A substance that provides relief from pain.

ANANDAMIDE. One type of endocannabinoid that appears to help regulate early pregnancy.

ANANKASTIC PERSONALITY DISORDER. The European term for obsessive-compulsive personality disorder.

ANDROGYNY. A way of behaving that includes high levels of both masculinity and femininity.

ANEMIA. Condition that results when there is a deficiency of oxygen in the blood. Can cause fatigue and impair mental functions.

ANEURYSM. A symptomless bulging of a weak arterial wall that can rupture, leading to stroke.

ANGINA. Severe pain and a feeling of constriction around the heart.

ANGIOGRAPHY. A procedure in which a contrast medium is injected into the bloodstream (through an artery in the neck) and its progress through the brain is tracked. This illustrates where a blockage or hemorrhage has occurred.

ANHEDONIA. Loss of the capacity to experience pleasure. Anhedonia is one of the so-called negative symptoms of schizophrenia, and is also a symptom of major depression.

ANOREXIA. Loss of appetite or unwillingness to eat. Can be caused by medications, depression, or many other factors.

ANOREXIA NERVOSA. An eating disorder characterized by an intense fear of weight gain accompanied by a distorted perception of one's own underweight body.

ANOSOGNOSIA. Lack of awareness of the nature of one's illness. The term is usually applied to stroke patients, but is sometimes used to refer to lack of insight on the part of patients with schizophrenia. Anosognosia appears to be caused by the illness itself; it does not appear to be a form of denial or inappropriate coping mechanism. It is, however, a factor in nonadherence to treatment regimens and the increased risk of relapse.

ANOXIA. Lack of oxygen.

ANTI-ANXIETY AGENT. A medication that is used to treat symptoms of generalized fear that dominates a person's life.

ANTAGONIST. A substance whose actions counteract the effects of or work in the opposite way from another chemical or drug.

ANTECEDENTS. Events that occur immediately before the target behavior.

ANTEROGRADE AMNESIA. Amnesia for events that occurred after a physical injury or emotional trauma but before the present moment.

ANTHELMINTHIC. A type of medication given to expel or eliminate intestinal worms.

ANTICHOLINERGIC. Related to the ability of a drug to block the nervous system chemical acetylcholine. When acetylcholine is blocked, patients often experience dry mouth and skin, increased heart rate, blurred vision, and difficulty in urinating. In severe cases, blocking acetylcholine may cloud thinking and cause delirium.

ANTICHOLINERGIC TOXICITY. A poisonous effect brought about by ingestion of medications or other toxins that block acetylcholine receptors. When these receptors are blocked, the person taking the medication may find that he or she gets overheated, has dry mouth, has blurry vision, and his or her body may retain urine.

ANTICIPATION. In medicine, a phenomenon in which certain diseases manifest at earlier ages or in more severe phenotypes in each successive generation of an affected family.

ANTICOAGULANT. A medication (such as warfarin, Coumadin, or Heparin) that decreases the blood's clotting ability, preventing the formation of new clots. Although anticoagulants will not dissolve existing clots, they can stop them from getting larger. These drugs are commonly called blood thinners.

ANTICONVULSANT MEDICATION. A medication that prevents convulsions or seizures; often prescribed in the treatment of epilepsy. Several anticonvulsant medications have been found effective in the treatment of bipolar disorder.

ANTIDEPRESSANT. A medication used to treat the symptoms of depression.

ANTIHISTAMINE. A medication used to alleviate allergy or cold symptoms such as runny nose, itching, hives, watering eyes, or sneezing.

ANTIHYPERTENSIVE. An agent used in the treatment of hypertension (high blood pressure).

ANTIOXIDANT. Substance that protects the body from damaging reactive oxygen molecules in the body. These reactive oxygen molecules can come from inside the body or from environmental pollution and are thought to play a role in the aging process and the development of degenerative disease.

ANTIPSYCHOTIC MEDICATION. A medication used to treat psychotic symptoms of schizophrenia such as hallucinations, delusions, and delirium. May be used to treat symptoms in other disorders, as well.

ANTISOCIAL BEHAVIOR. Behavior characterized by high levels of anger, aggression, manipulation, or violence.

ANTISOCIAL PERSONALITY DISORDER. Disorder characterized by behavior pattern of disregard for others' rights. People with this disorder often deceive and manipulate, or their behavior might include aggression to people or animals or property destruction, for example. This disorder has also been called sociopathy or psychopathy.

ANTISPASMODIC. A medication or preparation given to relieve muscle or digestive cramps.

ANXIETY. A feeling of apprehension and fear characterized by physical symptoms (heart palpitations, sweating, and feelings of stress, for example).

ANXIETY AND ANXIETY DISORDERS. Chronic conditions that can be characterized by an excessive and regular sense of apprehension, with physical symptoms such as sweating, palpitations, and feelings of stress. Anxiety disorders can be caused by biological and environmental events.

ANXIETY-REDUCTION TECHNIQUES. Skills taught by a therapist to help an individual overcome anxiety, stress, and tension, and can include relaxation, visualization and imagery, diaphragmatic breathing, stress inoculation, and meditation.

ANXIOLYTIC. A preparation or substance given to relieve anxiety; a tranquilizer.

APATHY. Lack of feelings or emotions.

APHASIA. Loss of language abilities.

APHONIA. Inability to speak caused by a functional disturbance of the voice box or vocal cords.

APHRODISIAC. A medication or preparation given to stimulate sexual desire.

APLASTIC ANEMIA. A form of anemia in which the bone marrow does not produce adequate amounts of peripheral blood components such as red cells, white cells, and platelets.

APNEA. A brief suspension or interruption of breathing.

APOLIPOPROTEIN E. A protein that transports cholesterol through the body. One form of this protein, apoE4, is associated with a 60% risk of late-onset Alzheimer's disease.

APPERCEPTION. The process of understanding through linkage with previous experience. The term was coined by one of the authors of the Thematic Apperception Test to underscore the fact that people don't "perceive" the story cards in a vacuum; rather, they construct their stories on the basis of past experiences as well as present personality traits.

APPETITE SUPPRESSANTS. Medications that assist in weight loss by reducing appetite or increasing the sensation of fullness.

APRAXIA. Inability to perform purposeful movements that is not caused by paralysis or loss of feeling.

ARRHYTHMIA. Any disturbance in the normal rhythm of the heartbeat.

ARSON. The deliberate setting of fires for criminal purposes, usually to collect insurance money or to cover up evidence of another crime. It is distinguished from pyromania by its connection with planning and forethought rather than failure of impulse control.

ARTERIOSCLEROSIS. A thickening, hardening, and loss of elasticity of the walls of the arteries.

ASANA. The Indian term for the poses or postures that are done in sequence during hatha yoga practice.

ASPERGER'S DISORDER. A condition in which young children experience impaired social interactions and develop limited repetitive patterns of behavior.

ASSAULTIVE. An act with intent of causing harm.

ASSERTIVE. Confidently self-assured; able to express oneself constructively and directly.

ASSESSMENT. In the context of psychological assessment (a structured interview), assessment is information-gathering to diagnose a mental disorder.

ASSISTED SUICIDE. A form of self-inflicted death in which a person voluntarily brings about his or her own

death with the help of another, usually a physician, relative, or friend.

ASSOCIATIONISM. A theory about human learning that explains complex psychological phenomena in terms of coincidental relationships. For example, a person with agoraphobia who is afraid of riding in a car may have had a panic attack in a car on one occasion and has learned to associate cars with the physical symptoms of a panic attack.

ASTRINGENT. A substance or compound that causes contraction or constriction of soft tissue.

ATAQUE DE NERVIOS. A culture-specific anxiety syndrome found among some Latino groups in the United States and in Latin America. It resembles panic disorder in some respects but also includes dissociative symptoms, and frequently occurs in response to a stressful event.

ATHEORETICAL. Unrelated to any specific theoretical approach or conceptual framework. The classification system of *DSM-IV-TR* is atheoretical.

ATHEROSCLEROSIS. Clogging of the arteries, creating a risk factor for stroke.

ATRIAL FIBRILLATION. A disorder in which the upper chambers (atria) of the heart do not completely empty with each contraction (heartbeat). This can allow blood clots to form and is associated with a higher risk of stroke.

ATTENTION DEFICIT DISORDER. A condition that mostly affects children and involves the inability to concentrate on various tasks.

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER (ADHD). A learning and behavioral disorder characterized by difficulty in sustaining attention, impulsive behavior, and excessive activity.

ATYPICAL ANTIPSYCHOTICS. A group of newer medications for the treatment of psychotic symptoms that were introduced in the 1990s. The atypical antipsychotics include clozapine, risperidone, quetiapine, ziprasidone, and olanzapine. They are sometimes called serotonin dopamine antagonists, or SDAs.

AUDITORY. Pertaining to the sense of hearing.

AURA. An energy field that is thought to emanate from the human body and to be visible to people with special psychic or spiritual powers.

AUTISM. A developmental disability that appears early in life, in which normal brain development is disrupted and social and communication skills are retarded, sometimes severely.

AUTISTIC PSYCHOPATHY. Hans Asperger's original name for the condition now known as Asperger's disorder. It is still used occasionally as a synonym for the disorder.

AUTONOMIC NERVOUS SYSTEM. The part of the nervous system that governs the heart, involuntary muscles, and glands.

AVERSION. A strong feeling of dislike or disgust. Aversion therapy makes use of this feeling to reduce or eliminate an undesirable behavior.

AVERSION THERAPY. An approach to treatment in which an unpleasant or painful stimulus is linked to an undesirable behavior in order to condition the patient to dislike or avoid the behavior. Chemicals or medications used to produce unpleasant effects are called aversants.

AVOIDANT COPING STRATEGIES. Ways of coping with stress that do not alter the problem in any way, but instead provide temporary relief or distraction.

AYURVEDIC MEDICINE. The traditional medical system of India. Ayurvedic treatments include diet, exercises, herbal treatments, meditation, massage, breathing techniques, and exposure to sunlight.

B

BACK-UP REINFORCER. A desirable item, privilege, or activity that is purchased with tokens and serves as a delayed reward and subsequent motivation for desired behavior.

BARBITURATES. A class of medications (including Seconal and Nembutal) that causes sedation and drowsiness. They may be prescribed legally but may also be used as drugs of abuse.

BASAL GANGLIA. A group of masses of gray matter located in the cerebral hemispheres of the brain that control movement as well as some aspects of emotion and cognition.

BASELINE DATA. Information regarding the frequency and severity of behavior, gathered before treatment begins.

BATTERY. A number of separate items (such as tests) used together. In psychology, a group or series of tests given with a common purpose, such as personality assessment or measurement of intelligence.

BEHAVIOR. A stereotyped motor response to an internal or external stimulus.

BEHAVIOR DISORDERS. Disorders characterized by disruptive behaviors such as conduct disorder, oppositional defiant disorder, and attention-deficit/hyperactivity disorder.

BEHAVIOR MODIFICATION. An approach to therapy based on the principles of operant conditioning. Behavior modification seeks to replace undesirable behaviors with preferable behaviors through the use of positive or negative reinforcement.

BEHAVIOR THERAPIES. Numerous techniques all having their roots in principles of learning.

BEHAVIORAL CONTRACTS. A behavioral contract is a written agreement that defines the behaviors to be performed and the consequences of the specified behaviors.

BEHAVIORAL DEFICIENCY. Failure to engage in a positive, desirable behavior frequently enough.

BEHAVIORAL EXCESS. Engaging in negative, undesirable behavior too often.

BEHAVIORAL INHIBITION. A set of behaviors that appear in early infancy that are displayed when the child is confronted with a new situation or unfamiliar people. These behaviors include moving around, crying, and general irritability, followed by withdrawing and seeking comfort from a familiar person. These behaviors are associated with an increased risk of social phobia and panic disorder in later life. Behavioral inhibition in children appears to be linked to anxiety and mood disorders in their parents.

BEHAVIORAL PHENOTYPE. A term that refers to the greater likelihood that people with a specific genetic syndrome will have certain behavioral or developmental characteristics, compared to people who do not have the syndrome.

BEHAVIORAL THERAPY. An approach to treatment that focuses on extinguishing undesirable behavior and replacing it with desired behavior.

BENIGN PROSTATE HYPERTROPHY. Enlargement of the prostate gland.

BENZODIAZEPINES. A group of central nervous system depressants used to relieve anxiety or to induce sleep.

BEREAVEMENT. The emotional experience of loss after the death of a friend or relative.

BETA AMYLOID PROTEIN. A starchy substance that builds up in the brains of people with Alzheimer's disease to form the plaques that are characteristic of the disease. Beta amyloid is formed when amyloid precursor protein, or APP, is not broken down properly by the body.

BETA BLOCKER. Drugs that block beta-adrenergic receptors on neurons in the central nervous system. When these sites are blocked, heart rate, blood pressure, and anxiety levels decrease.

BEZOAR. A hard ball of hair or vegetable fiber that may develop in the stomach of humans as the result of ingesting nonfood items.

BINGE. An excessive amount of food consumed in a short period of time. Usually, while a person binge-eats, he or she feels disconnected from reality, and feels unable to stop. The bingeing may temporarily relieve depression or anxiety, but after the binge, the person usually feels guilty and depressed.

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BIOAVAILABILITY. Medication that is available in the body. If the bioavailability of a drug is increased, more is available to the body for use, and if it is decreased, less is available for use.

BIOCHEMICAL. Chemical reactions occurring in living systems.

BIOFEEDBACK. Biofeedback is a technique that uses monitoring instruments to measure and feed back information about muscle tension, heart rate, sweat responses, skin temperature, or brain activity.

BIOFIELD THERAPIES. A subgroup of energy therapies that make use of energy fields (biofields) thought to exist within or emanate from the human body. Biofield therapies include such approaches as Reiki, therapeutic touch, qigong, and polarity balancing.

BIOLOGICAL MARKER. An indicator or characteristic trait of a disease that facilitates differential diagnosis (the process of distinguishing one disorder from other, similar disorders).

BIOPSYCHOSOCIAL HISTORY. A history of significant past and current experiences that influence client behaviors, including medical, educational, employment, and interpersonal experiences. Alcohol or drug use and involvement with the legal system are also assessed in a biopsychosocial history.

BIOPSYCHOSOCIAL MODEL. A hypothetical explanation for why something occurs that includes biological, psychological, and social causes or correlates.

BIOSOCIAL. A biosocial model in psychology asserts that social and biological factors contribute toward the development of personality.

BIPOLAR AFFECTIVE DISORDER. A disorder in which a person alternates manic and depressive episodes.

BIPOLAR DISORDER (FORMERLY MANIC-DEPRESSIVE DISORDER). A mental disorder characterized by dramatic and sometimes rapid mood swings, resulting in both manic and depressive episodes.

BIPOLAR DISORDER NOT OTHERWISE SPECIFIED. Disorder of mood involving mood swings that do not meet criteria for other disorders specified above.

BIPOLAR DISORDERS. Disorders characterized by wide fluctuations in mood.

BIPOLAR I DISORDER. A major mood disorder characterized by full-blown manic episodes, often interspersed with episodes of major depression.

BIPOLAR II DISORDER. Disorder with major depressive episodes and mild manic episodes known as hypomania.

BLACKOUT. A period of loss of consciousness or memory.

BLENDED FAMILY. A family formed by the remarriage of a divorced or widowed parent. It includes the new husband and wife, plus some or all of their children from previous marriages.

BLEOMYCIN HYDROLASE. An enzyme involved in the body's processing of amyloid precursor protein. If the gene that governs production of BH mutates, the APP accumulates, producing the plaques in the brains of patients with Alzheimer's disease.

BLUNTED AFFECT. A term that refers to the loss of emotional expressiveness sometimes found in patients with schizophrenia. It is sometimes called flattened affect.

BODY IMAGE. A term that refers to a person's inner picture of his or her outward appearance. It has two components: perceptions of the appearance of one's body, and emotional responses to those perceptions.

BODY MASS. The quantity of matter in the body (measured by dividing weight by acceleration due to gravity).

BODY MASS INDEX, OR BMI. A measure of body fat, calculated as weight in kilograms over the square of height in meters.

BODYWORK. Any technique involving hands-on massage or manipulation of the body.

BORDERLINE PERSONALITY DISORDER. A severe and usually life-long mental disorder characterized by violent mood swings and severe difficulties in sustaining interpersonal relationships.

BRACHYCARDIA. Slow heartbeat, defined as a rate of less than 60 beats per minute.

BRAIN STEM. The part of the brain that is continuous with the spinal cord and controls most basic life functions. It is the last part of the brain that is destroyed by Alzheimer's disease.

BREEMA. An alternative therapy that originated in California in the 1980s. Breema combines biofield therapy with certain elements of chiropractics and bodywork.

BRUXISM. Habitual, often unconscious, grinding of the teeth.

BULIMIA NERVOSA. An eating disorder characterized by binges in which large amounts of food are consumed, followed by forced vomiting.

BUPRENORPHINE. A medication that blocks some of the withdrawal effects during heroin detoxification.

BURDEN. First described by Treudley in 1946, this term generally refers to the consequences for the family of close contact with a person who is severely mentally ill.

BURNOUT. An emotional condition that interferes with job performance, marked by fatigue, loss of interest, or frustration; usually regarded as the result of prolonged stress.

C

CAFFEINISM. A disorder caused by ingesting very high doses of caffeine (10g or more per day) and characterized by seizures and respiratory failure.

CALORIE. The quantity of heat necessary to raise the temperature of 1kg of water 1°C.

CANCER SCREENING. A procedure designed to detect cancer even though a person has no symptoms, usually performed using an imaging technique.

CANNABIS. The collective name for several varieties of Indian hemp plant. Also known as marijuana.

CANNABIS ABUSE. Periodic use of cannabis, less serious than dependence, but still capable of causing problems for the user.

CANNABIS DEPENDENCE. The compulsive need to use cannabis, leading to problems.

CANNABIS INTOXICATION. The direct effects of acute cannabis use and the reactions that accompany those effects.

CAPITATED PAYMENT SYSTEM. A contract between managed care organizations and health care providers involving a prepaid amount for blocks of services.

CARDIAC TAMPONADE. A condition in which blood leaking into the membrane surrounding the heart puts pressure on the heart muscle, preventing complete filling of the heart's chambers and normal heartbeat.

CARMINATIVE. A substance or preparation that relieves digestive gas.

CARPAL TUNNEL SYNDROME. A disorder of the hand and wrist characterized by pain, weakness, or numbness in the thumb and other fingers. It is caused by pressure on a nerve in the wrist. Carpal tunnel syndrome is frequently associated with heavy use of a computer, typewriter, or musical keyboard.

CARRIER. A vegetable oil such as safflower, olive, grapeseed, or wheatgerm oil used to dilute essential oils for massage.

CARVE-OUT PLANS. Managed care plans that make provision for mental health services by creating subcontracts involving different terms of payment and utilization review from those used for general health care.

CASE MANAGER. A professional who designs and monitors implementation of comprehensive care plans (i.e., services addressing medical, financial, housing, psychiatric, vocational, social needs) for individuals seeking mental health or social services.

CASE RATE. A type of contract between managed care organizations and health care providers involving a prepaid amount for services on a case-by-case basis.

CASTRATION. Desexing a person or animal by surgical removal of the testes (in males) or ovaries (in females). Castration is sometimes offered as a treatment option to violent rapists and pedophiles who are repeat offenders.

CATALEPSY. An abnormal condition characterized by postural rigidity and mental stupor, associated with certain mental disorders.

CATAPLEXY. A symptom of narcolepsy marked by a sudden episode of muscle weakness triggered by strong emotions. The muscle weakness may cause the person's knees to buckle, or the head to drop. In severe cases, the patient may become paralyzed for a few seconds to minutes.

CATATONIC BEHAVIOR OR CATATONIA. Term that describes both possible extremes related to movement. Catalepsy is the motionless aspect of catatonia—a person with catalepsy may remain fixed in the same position for hours on end. Rapid or persistently repeated movements, frequent grimacing and strange facial expressions, and unusual gestures are the opposite end of the catatonia phenomenon, involving an excess or distorted extreme of movement.

CATATONIC DISORDER. A severe disturbance of motor behavior characterized by either extreme immobility or stupor, or by random and purposeless activity.

CATATONIC SCHIZOPHRENIA. A subtype of a severe mental disorder that affects thinking, feeling, and behavior, and that is also characterized by catatonic behaviors—either extreme stupor or random, purposeless activity.

CATCHMENT. In mental health, a term that refers to a particular geographical area served by a particular mental health agency.

CATECHOLAMINE. A group of neurotransmitters synthesized from the amino acid tyrosine and released by the hypothalamic-pituitary-adrenal system in the brain in response to acute stress. The catecholamines include dopamine, serotonin, norepinephrine, and epinephrine.

CATHARSIS. A powerful emotional release followed by a feeling of great relief.

CATHETERIZATION. Placing a tube in the bladder so that it can be emptied of urine.

CENTRAL NERVOUS SYSTEM DEPRESSANT. Any drug that lowers the level of stimulation or excitement in the central nervous system.

CENTRAL NERVOUS SYSTEM STIMULANT. Any drug that raises the level of activity in the central nervous system.

CEREBRAL ATERIOGRAPHY. A procudure that allows a wire to be inserted in blood vessels in the brain, which generates an image of diseases in these arteries.

CEREBROVASCULAR. Blood flow in the brain.

CERVIX. The neck or narrow lower end of a woman's uterus.

CHAKRA. One of the seven major energy centers in the body, according to traditional Indian yoga.

CHASING. Betting larger and larger sums of money, or taking greater risks, in order to make up for money previously lost in gambling.

CHELATION. A method of treating lead or mercury poisoning by giving medications that remove heavy metals from the bloodstream. The medications that are used are called chelating agents.

CHOLINESTERASE INHIBITORS. A group of medications given to slow the progression of Alzheimer's disease.

CHOREATHETOID MOVEMENTS. Repetitive dance-like movements that have no rhythm.

CHROMATHERAPY. An alternative form of light therapy in which colored light is directed toward a specific chakra or part of the body in order to heal or correct energy imbalances. Practitioners of chromatherapy are sometimes called chromapaths.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE. Disorder characterized by the decreasing ability of the lungs to ventilate adequately.

CHRYSIN. A flavonoid found in passionflower that may be the source of its anxiolytic properties.

CLASSICAL CONDITIONING. In psychology, a process in which a previously neutral stimulus eventually produces a specific response by being paired repeatedly with another stimulus that produces that response. The best-known example of classical conditioning is Pavlov's dogs, who were conditioned to salivate when they heard a bell ring (the previously neutral stimulus) because the sound had been paired repeatedly with their feeding time.

CLEARINGHOUSE. A centralized organization that is a repository of information and that facilitates access to information.

CLITORIS. The most sensitive area of the female external genitals. Stimulation of the clitoris causes most women to reach orgasm.

CLONIC-TONIC SEIZURE. This is the most common type of seizure among all age groups and is categorized into several phases beginning with vague symptoms hours or days before an attack. These seizures are sometimes called grand mal seizures.

CLOZAPINE. A newer antipsychotic medication that is often given to patients who are developing signs of tardive dyskinesia.

CLUSTER SUICIDE. Refers to the phenomenon of additional suicides being attempted or completed after one suicide has occurred within a small community, such as a group of high school students.

COCA PLANT. The plant that is the source of cocaine.

COCAINE. An illegal drug that increases energy and induces euphoria. It is addictive and is often abused.

CODEINE. A medication that may be prescribed but also may be purchased illegally and is used to reduce pain.

CODON. A three-member nucleotide sequence in messenger RNA that codes for a specific amino acid in synthesizing protein molecules.

COGNITION. The act or process of knowing or perceiving.

COGNITIVE. Pertaining to the mental processes of memory, perception, judgment, and reasoning.

COGNITIVE RESTRUCTURING. An approach to psychotherapy that focuses on helping the patient examine distorted patterns of perceiving and thinking in order to change their emotional responses to people and situations.

COGNITIVE STYLE. A way in which an individual works with and performs cognitive tasks such as reasoning, learning, thinking, understanding, making decisions, and using memory.

COGNITIVE THERAPY. Psychological treatment aimed at changing a person's way of thinking in order to change his or her behavior and emotional state.

COGNITIVE-BEHAVIORAL THERAPY (CBT). An approach to psychotherapy that emphasizes the correction of distorted thinking patterns and changing one's behaviors accordingly.

COGWHEEL RIGIDITY. An abnormal rigidity in muscles, characterized by jerky movements when the muscle is passively stretched.

COITUS. Sexual intercourse.

COLD TURKEY. A slang term for stopping the use of nicotine (or any other addictive drug) suddenly and completely.

COMA. Unconsciousness.

COMMUNITY MENTAL HEALTH CENTERS. Organizations that manage and deliver a comprehensive range of mental health services, education, and outreach to residents of a given community.

COMMUNITY MENTAL HEALTH CENTERS ACT OF 1963. Federal legislation providing grants for the operation of community mental health centers and related services.

COMORBID. Having another disorder or condition simultaneously.

COMORBID PSYCHOPATHOLOGY. The presence of other mental disorders in a patient together with the disorder that is the immediate focus of therapy.

COMORBIDITY. Association or presence of two or more mental disorders in the same patient. A disorder that is said to have a high degree of comorbidity is likely to occur in patients diagnosed with other disorders that may share or reinforce some of its symptoms.

COMPENSATORY. Counterbalancing or offsetting. A compensatory strategy is one that makes up for or balances a weakness in some area of functioning.

COMPETING BEHAVIORS. Behaviors that interfere with the target behavior because they are preferred by the individual.

COMPLEX ABSENCE SEIZURE. Absence (petit mal) seizures usually begin with a brief loss of consciousness and last between one and 10 seconds. People having a petit mal seizure become very quiet and may blink, stare blankly, roll their eyes, or move their lips. A petit mal seizure lasts 15–20 seconds. When it ends, the individual resumes whatever he or she was doing before the seizure began, and may not realize that anything unusual happened.

COMPLEX SEIZURE. In complex seizures, the person experiences impaired consciousness.

COMPLIANCE. In medicine or psychiatry, cooperation with a treatment plan or schedule of medications.

COMPULSION. A strong impulse to perform an act, particularly one that is irrational or contrary to one's will.

CONDUCT DISORDER. A behavioral and emotional disorder of childhood and adolescence in which children display physical aggression and infringe on or violate the rights of others. Youths diagnosed with conduct disorder may set fires, exhibit cruelty toward animals or other children, sexually assault others, or lie and steal for personal gain.

CONFABULATION. In psychiatry, the filling-in of gaps in memory with false information that the patient believes to be true. It is not deliberate telling of lies.

CONGENITAL. Present at birth.

CONGESTIVE HEART FAILURE. Condition characterized by abdominal pain, swelling in the lower extremities, and weakness caused by a reduced output of blood from the left side of the heart.

CONGRUENCE. A quality of the client-centered therapist, consisting of openness to the client.

CONSEQUENCES. Events that occur immediately after the target behavior.

CONSTIPATION. Difficult bowel movements caused by the infrequent production of hard stools.

CONTINGENCIES. Naturally occurring or artificially designated reinforcers or punishers that follow a behavior.

CONTRAST AGENT, or MEDIUM. A substance injected into the body that illuminates certain structures that would otherwise be hard to see on the radiograph (film).

CONTROLLED BEHAVIOR. The behavior to be changed by self-control strategies; also known as the target behavior.

CONTROLLING BEHAVIORS. Self-control strategies used to change the controlled or target behavior.

CONVERSION. In psychiatry, a process in which a repressed feeling, impulse, thought, or memory emerges in the form of a bodily symptom.

CONVERSION DISORDER. A type of somatoform disorder in which unconscious psychological conflicts or other factors take the form of physical symptoms that are produced unintentionally.

CONVULSION. A violent, involuntary contraction or series of contractions of muscles.

COPING. In psychology, a term that refers to a person's patterns of response to stress.

COPROLALIA. A vocal tic characterized by uttering obscene, hostile, or inappropriate words. A motor tic characterized by obscene gestures is called copropraxia.

CORONARY OCCLUSION. Blockage of the arteries supplying the blood to the heart.

CORPUS CALLOSUM. (plural, corpora callosa) A thick bundle of nerve fibers lying deep in the brain that connects the two cerebral hemispheres and coordinates their functions.

CORTEX. Region in the brain where sensation and perception are processed and integrated into thoughts, memories, and abilities; also where actions are planned and initiated.

CORTICOSTEROIDS. Any one of a number of hormonal steroid compounds that are derived from the adrenal gland.

CORTISOL. A steroid hormone released by the cortex (outer portion) of the adrenal gland when a person is under stress.

COVERT. Concealed, hidden, or disguised.

CRACK. A slang term for a form of cocaine that is smokable.

CREUTZFELDT-JAKOB DISEASE. A degenerative disease of the central nervous system caused by a prion, or "slow virus."

CRITICAL INCIDENT. Also known as a crisis event. An event that is stressful enough to overwhelm the coping skills of a person or group.

CROSS-DRESSING. Wearing clothing and other attire appropriate to the opposite sex.

CRYSTALLIZED INTELLIGENCE. A type of intelligence that reflects knowledge and skills influenced by a person's sociocultural environment.

CT SCAN. An imaging technique that uses a computer to combine multiple x-ray images into a two-dimensional cross-sectional image. The full name is *computed tomography*.

CUE. Any behavior or event in a person's environment that serves to stimulate a particular response. For example, the smell of liquor may be a cue for some people to pour themselves a drink.

CUTOFF SCORES. In psychological testing, scores that indicate the borderline between normal and impaired functioning.

CYCLIC AMP. A small molecule of adenosine monophosphate (AMP) that activates enzymes and increases the effects of hormones and other neurotransmitters.

CYCLOTHYMIA. An alternate name for cyclothymic disorder.

CYCLOTHYMIC DISORDER. A relatively mild mood disorder characterized by mood swings between mild depression to mild mania.

CYTOGENETICS. The branch of biology that combines the study of genetic inheritance with the study of cell structure.

D

DAWN SIMULATION. A form of light therapy in which the patient is exposed while asleep to gradually brightening white light over a period of an hour and a half.

DECISION-MAKERS. In some mental health contexts, the term refers to prison or court officials, treatment facility administrators, or family members.

DECONDITIONING. Loss of physical strength or stamina resulting from bed rest or lack of exercise.

DEDUCTIBLE. The amount of money that must be paid out of pocket by health care consumers before the insurance provider will make payments.

DEFENSE. An unconscious mental process that protects the conscious mind from unacceptable or painful thoughts, impulses, or desires. Examples of defenses include denial, rationalization, projection, and repression. Some defenses are considered to represent lower levels of maturation than others; thus, identifying a child's defenses may be helpful in evaluating his or her level of psychological maturity.

DEFENSE MECHANISMS. Indirect strategies used to reduce anxiety rather than directly facing the issues causing the anxiety.

DEINSTITUTIONALIZATION. The process of moving people out of mental hospitals into treatment programs or halfway houses in local communities. With this movement, the responsibility for care shifted from large (often governmental) agencies to families and community organizations.

DELIRIUM. A disturbance of consciousness marked by confusion, difficulty paying attention, delusions, hallucinations, or restlessness. It can be distinguished from dementia by its relatively sudden onset and variation in the severity of the symptoms.

DELIRIUM TREMENS. Serious alcohol withdrawal symptoms that must be treated in a hospital and that may include shaking, delirium, and hallucinations.

DELTA-9-TETRAHYDROCANNABINOL(THC). The primary active ingredient in marijuana.

DELUSION. A false belief that is resistant to reason or contrary to actual fact. A patient may be convinced, for example, that someone is trying to poison him or her, or that he or she has a fatal illness despite evidence to the contrary.

DELUSIONAL DISORDER OF THE PERSECUTORY TYPE. A psychotic disorder characterized by a patient's belief that others are conspiring against him or her.

DEMENTIA. A group of symptoms (syndrome) associated with a progressive loss of memory and other intellectual functions that is serious enough to interfere with a person's ability to perform the tasks of daily life. Dementia impairs memory, alters personality, leads to

deterioration in personal grooming, impairs reasoning ability, and causes disorientation.

DEMENTIA INFANTILIS. Another term for childhood disintegrative disorder, used more frequently in the European medical literature. The Latin name literally means "early childhood dementia."

DEMENTIA PRAECOX. A late nineteenth-century term for schizophrenia.

DENIAL. A psychological defense mechanism that reduces anxiety by excluding recognition of an addiction or similar problem from the conscious mind.

DEPENDENCE. The adaptation of neurons and other physical processes to the use of a drug, followed by withdrawal symptoms when the drug is removed; physiological and/or psychological addiction.

DEPENDENT PERSONALITY DISORDER. Personality disorder characterized by a constant, unhealthy need to be liked and appreciated by others at all costs.

DEPERSONALIZATION. A dissociative symptom in which the patient feels that his or her body is unreal, changing, or dissolving.

DEPERSONALIZATION NEUROSIS. Another name for depersonalization disorder.

DEPRESSANT. Something that slows down functioning.

DEPRESSION. A mental state characterized by excessive sadness. Other symptoms include altered sleep patterns, thoughts of suicide, difficulty concentrating, agitation, lack of energy, and loss of enjoyment in activities that are usually pleasurable.

DEREALIZATION. A dissociative symptom in which the external environment is perceived as unreal or dreamlike.

DERVISH. A person who belongs to one of the various mystical and ascetic Muslim orders, such as the Sufis. A whirling dervish meditates by whirling or spinning in an ecstatic dance.

DESENSITIZATION. The reduction or elimination of an overly intense reaction to a cue by controlled, repeated exposures to the cue.

DESIGNER AMPHETAMINES. Substances close in chemical structure to classic amphetamines that provide both stimulant and hallucinogenic effects.

DETOXIFICATION. A process in which the body is allowed to free itself of a drug while the symptoms of withdrawal are treated. It is the primary step in any treatment program for drug or alcohol abuse.

DEVELOPMENTAL DELAY. The failure to meet certain developmental milestones, such as sitting, walking, and talking, at the average age. Developmental delay may indicate a problem in development of the central nervous system.

DEVELOPMENTAL DISABILITIES. Disabilities that are present from birth and delay or prevent normal development, such as mental retardation or autism.

DEXFENFLURAMINE (REDUX). A prescription appetite suppressant for weight loss that was withdrawn from the market due to unacceptable health risks.

DIABETES MELLITUS. A chronic disease affecting the metabolism of carbohydrates that is caused by insufficient production of insulin in the body.

DIABETIC NEUROPATHY. A condition in which the nerve endings, particularly in the legs and feet, become less sensitive. Minor injuries, such as blisters or calluses, are not felt and can thus become infected and become more serious problems.

DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS. A handbook for mental health professionals that includes lists of symptoms that indicate diagnoses of mental disorders.

DIATHESIS. The medical term for predisposition. The stress/diathesis model is a diagram that is used to explain why some people are at greater risk of suicidal behavior than others.

DIETHYLPROPION (TENUATE, TENUATE DOSPAN). A prescription appetite suppressant currently on the market for weight loss.

DIFFERENTIAL DIAGNOSIS. The process of distinguishing one disorder from other, similar disorders.

DIFFERENTIATION. The ability to retain one's identity within a family system while maintaining emotional connections with the other members.

DIGRAPH. A pair of letters that represents a single speech sound. In English, the *th* in "thumb" and the *ei* in "vein" are examples of digraphs.

DIPLOPIA. A disorder of vision in which a single object appears double. Diplopia is sometimes called double vision.

DISFLUENCY. Disruptions, breakage, or blockages in the forward flow of speech.

DISPLACEMENT. A psychological process in which feelings originating from one source are expressed outwardly in terms of concern or preoccupation with an issue or problem that the patient considers more accept-

able. In some patients with body dysmorphic disorder, obsession about the body includes displaced feelings, often related to a history of childhood abuse.

DISSOCIATED. Feelings of experiencing an altered state of reality, similar to a trance state. During the period of dissociation, the affected person may feel as if he or she is an observer instead of a participant in events, and may feel as if surroundings are unreal or distorted.

DISSOCIATION. A reaction to trauma in which the mind splits off certain aspects of the traumatic event from conscious awareness. Dissociation can affect the patient's memory, sense of reality, and sense of identity.

DISSOCIATIVE AMNESIA. A dissociative disorder characterized by loss of memory for a period or periods of time in the patient's life. May occur as a result of a traumatic event.

DISSOCIATIVE DISORDERS. A group of disorders marked by the separation (dissociation) of perception, memory, personal identity, and consciousness. Depersonalization disorder is one of five dissociative disorders defined by *DSM-IV-TR*.

DISSOCIATIVE IDENTITY DISORDER (DID). Term that replaced *multiple personality disorder*. A condition in which two or more distinctive identities or personality states alternate in controlling a person's consciousness and behavior.

DISTENSION. The condition of being stretched or expanded, as the abdomen of a pregnant woman.

DISULFIRAM. A medication helps reinforce abstinence from alcohol in people who are recovering from alcohol abuse. If a person taking disulfiram drinks even a small amount of alcohol, he or she experiences facial flushing, headache, nausea, and vomiting.

DIURETIC. A medication or substance given to increase the amount of urine excreted.

DIZYGOTIC. Developed from two fertilized ova. Dizygotic twins are sometimes called fraternal twins.

DOMINANT HAND. The hand that one prefers to use when performing various tasks such as writing or throwing an object.

DOPAMINE. A chemical in brain tissue that serves to transmit nerve impulses (is a neurotransmitter) and helps to regulate movement and emotions.

DOUBLE ANXIETY. Acute anxiety from a recent stressful event combined with underlying persistent anxiety associated with generalized anxiety disorder.

DOUBLE-BLIND PLACEBO-CONTROLLED STUDY. A study in which patients are divided into two groups—those who will recive a medication, and those who will receive a placebo (a pill that looks like the medication but has no active ingredients). Neither the patients nor their physicians know which pill any specific patient is receiving.

DOUCHE. A jet or current of water, often with a medication or cleansing agent dissolved in it, applied to a body cavity for medicinal or hygienic purposes.

DOWN SYNDROME. A genetic disorder characterized by an extra chromosome 21 (trisomy 21), mental retardation, and susceptibility to early-onset Alzheimer's disease.

DREAM ANXIETY DISORDER. Another name for nightmare disorder.

DSM. Abbreviation for the *Diagnostic and Statistical Manual of Mental Disorders,* a handbook for mental health professionals that includes lists of symptoms that indicate specific diagnoses. The text is periodically revised, and the latest version was published in 2000 and is called *DSM-IV-TR*, for Fourth Edition, Text Revised.

DUE PROCESS. A term referring to the regular administration of a system of laws that conform to fundamental legal principles and are applied without favor or prejudice to all citizens. In the context of involuntary commitment or hospitalization, due process means that people diagnosed with a mental illness cannot be deprived of equal protection under the laws of the United States on the basis of their diagnosis.

DYSARTHRIA. A group of speech disorders caused by disturbances in the strength or coordination of the muscles of the speech mechanism as a result of damage to the brain or nerves. Difficulty talking and speaking.

DYSKINESIA. Difficulty in performing voluntary muscular movements.

DYSLEXIA. A type of reading disorder.

DYSMORPHIC. Malformed.

DYSPAREUNIA. Painful sexual intercourse.

DYSPRAXIA. Developmental dyspraxia is an impairment or immaturity of the organization of movement. It is a defect in the way the brain processes information, resulting in messages not being correctly or fully transmitted. The term dyspraxia comes from the word "praxis," meaning "doing" or "acting." Dyspraxia is associated with problems of perception, language, and thought.

DYSSOMNIA. A type of sleep disorder characterized by a problem with the amount, quality, or timing of the patient's sleep.

DYSTHYMIA. Depression of low intensity.

DYSTHYMIC DISORDER. A mood disorder that is less severe than depression but usually more chronic.

DYSTONIA. A neurological disorder characterized by involuntary muscle spasms. The spasms can cause a painful twisting of the body and difficulty walking or moving. Some medications can cause dystonia.

E

ECHOLALIA. Meaningless repetition of words or phrases spoken by another.

ECHOPRAXIA. Imitation of another person's physical movements in a repetitious or senseless manner.

ECSTASY. Best known of the so-called designer amphetamines, also known as MDMA. It produces both stimulant and hallucinogenic effects.

ECT. Electroconvulsive therapy is sometimes used to treat depression or mania when pharmaceutical treatment fails.

ECZEMA. An inflammation of the skin characterized by itching and oozing of a clear fluid.

EDEMA. Abnormal accumulation of fluid in the interstitial spaces of bodily tissue.

EGO. In Freudian psychology, the conscious, rational part of the mind that experiences and reacts to the outside world.

EGOCENTRICITY. Self-centeredness.

EJACULATION. The discharge of semen by the male reproductive organs.

ELECTROACUPUNCTURE. A variation of acupuncture in which the practitioner stimulates the traditional acupuncture points electronically.

ELECTROCARDIOGRAM. (EKG) A test that measures the electrical activity of the heart as it beats. An abnormal EKG can indicate possible cardiac disease.

ELECTROCONVULSIVE THERAPY. Medical treatment that uses electrical currents to cause seizures; sometimes used to treat depression.

ELECTROENCEPHALOGRAM. (EEG) A test that measures the electrical activity of the brain by means of electrodes placed on the scalp or on or in the brain itself.

ELECTROENCEPHALOGRAPH. (EEG) An instrument that measures the normal and abnormal electrical activity in the brain.

ELECTROENCEPHALOGRAPHY. The measurement and recording of the brain's electrical activity.

ELECTROLYTES. Substances or elements that dissociate into electrically charged particles (ions) when dissolved in the blood. The electrolytes in human blood include potassium, magnesium, and chloride.

ELECTRON. One of the small particles that make up an atom. An electron has the same mass and amount of charge as a positron, but the electron has a negative charge.

ELIMINATION. The medical term for expelling wastes from the body.

EMETIC. A medication intended to cause vomiting. Emetics are sometimes used in aversion therapy in place of electric shock. Their most common use in mainstream medicine is in treating accidental poisoning.

EMPATHY. A quality of the client-centered therapist, characterized by the therapist's conveying appreciation and understanding of the client's point of view.

EMPIRICAL. Verified by actual experience or by scientific experimentation.

ENCEPHALITIS. Inflammation of the brain.

ENCEPHALOPATHY. Brain disease that causes damage or degeneration.

ENCOUNTER GROUPS. A term coined by Carl Rogers for therapist-run groups that focus on personal exploration, experiencing in the here-and-now (that is, feelings and interpersonal exchanges occurring in the group setting), and genuine concern and honesty among the members.

ENDOCANNABINOIDS. Cannabis-like compounds produced naturally in the human body.

ENDOGENOUS DEPRESSION. Depression arising from causes within a person, such as chemical or hormonal imbalances.

ENDORPHINS. A group of peptide compounds released by the body in response to stress or traumatic injury. Endorphins react with opiate receptors in the brain to reduce or relieve pain.

ENERGY. The capability of producing force, performing work, or generating heat.

ENFLEURAGE. A technique for extracting essential oils from flower petals by placing them on a layer of purified fat.

ENURESIS. The inability to control urination; bedwetting.

EPHEBOPHILIA. Sexual desire on the part of an adult for youths in the early stages of puberty, as distinct from prepubertal children.

EPHEDRINE. An amphetamine-like substance used as a nasal decongestant.

EPIDEMIOLOGY. The study of the causes, incidence, transmission, and control of diseases.

EPILEPSY. A neurological disorder characterized by the onset of seizures. Seizures are caused by a disturbance in the electrical activity in the brain and can cause loss of consciousness, muscle spasms, rhythmic movements, abnormal sensory experiences, or altered mental states.

EPINEPHRINE (ADRENALINE). The principal blood-pressure raising hormone secreted by the adrenal glands in response to stress; a bronchial and intestinal smooth muscles relaxant.

EPISODIC DYSCONTROL. Another term for intermittent explosive disorder.

EROTOMANIC DELUSIONS. Erotomanic delusions involve the mistaken conviction that someone is in love with the delusional person. Often, the love object is a public figure of some prominence, such as an actress, rock star, or political figure. David Letterman and Jodie Foster are celebrities who have both been victimized by persons with erotomanic delusions.

ESSENTIAL FATTY ACIDS (EFAS). a group of polyunsaturated fats that are essential to life and growth but cannot be produced by the body.

ESSENTIAL OIL. The product of special ducts or cells in the tissues of aromatic plants (or the sap of certain trees) that gives the plant its characteristic aroma and therapeutic properties. Essential oils are sometimes called volatile oils because they evaporate readily at room temperature.

ETIOLOGY. The cause or origin of a disease or disorder. The word is also used to refer to the study of the causes of disease.

EUPHORIA. A feeling or state of well-being or elation.

EUSTRESS. A term that is sometimes used to refer to positive stress.

EUTHANASIA. The act of putting a person or animal to death painlessly or allowing them to die by withholding medical services, usually because of a painful and incurable disease. Mercy killing is another term for euthanasia.

EXECUTIVE. Pertaining to supervision, planning, and carrying out duties or actions.

EXECUTIVE FUNCTIONS. A set of cognitive abilities that control and regulate other abilities and behaviors. Necessary for goal-directed behavior, they include the ability to initiate and stop actions, to monitor and change behavior as needed, and to plan future behavior when faced with novel tasks and situations.

EXISTENTIAL FACTORS. Realities of life including death, isolation, freedom, and meaninglessness that must be faced by all individuals.

EXON. A segment of DNA that is transcribed to RNA and encodes information about the protein sequence.

EXPANSION MUTATION. A genetic mutation caused by additional repetitions of a triplet, or trinucleotide sequence, during the process of genetic transmission. In Huntington's disease, the expansion mutation produces more of a toxic gene product.

EXPERIENTIAL KNOWLEDGE. Knowledge gained from experience, often practical, in contrast with theoretical or professional knowledge.

EXPERIENTIAL THERAPY. An approach to therapy that focuses on experiencing inner feelings, rather than talking about problems in a disconnected, intellectual way. Although it is based on person-centered therapy, experiential therapy is more directive because it uses techniques from a variety of therapeutic approaches to draw out a person's inner experiences.

EXPLICIT MEMORY. Consciously recalled memory for facts or events.

EXPOSURE THERAPY. A form of cognitive-behavioral therapy in which patients suffering from phobias are exposed to their feared objects or situations while accompanied by the therapist. The length of exposure is gradually increased until the association between the feared situation and the patient's experienced panic symptoms is no longer present.

EXPRESSIVE THERAPY. An approach to psychotherapy that seeks to relieve the patient's symptoms through exploration of previously unconscious material, leading to greater insight and more adaptive behaviors.

EXTENDED FAMILY FIELD. A person's family of origin plus grandparents, in-laws, and other relatives.

EXTENSIVE SUPPORT. Ongoing daily support required to assist an individual in a specific adaptive area, such as daily help with preparing meals.

EXTERNALIZING DISORDERS. Mental disorders with primary symptoms that involve outward behavior as opposed to inner emotions.

EXTINCTION. The elimination or removal of a person's reaction to a cue as a result of exposure treatment.

EXTRAPYRAMIDAL. Brain structures located outside the pyramidal tracts of the central nervous system.

EXTRAPYRAMIDAL MOVEMENT DISORDERS. Involuntary movements that occur as a side effect of some psychiatric medications.

EXTRAPYRAMIDAL SIDE EFFECTS. A group of neurological side effects including muscle spasms, involuntary movements, and symptoms that resemble Parkinson's disease (also called drug-induced Parkinsonism).

F

FACTITIOUS DISORDER. A type of mental disturbance in which patients intentionally act physically or mentally ill without obvious benefits. It is distinguished from malingering by the absence of an obvious motive, and from conversion disorder by intentional production of symptoms.

FACTOR ANALYSIS. A statistical method for summarizing relationships between variables. For the HAS, factor analysis was utilized to determine the specific sets of symptoms relating to overall anxiety, somatic anxiety, and psychic anxiety.

FADING. Gradually decreasing the amount or frequency of a reinforcer so that the target behavior will begin to occur independent of any rewards.

FAIR HOUSING ACT OF 1968. Federal legislation regarding access to housing that prohibits discrimination based on race, color, national origin, sex, religion, disability, or familial status.

FALSE-POSITIVE. A test result that is positive for a specific condition or disorder, but this result is inaccurate.

FAMILY SYSTEMS THEORY. An approach to treatment that emphasizes the interdependency of family members rather than focusing on individuals in isolation from the family. This theory underlies the most influential forms of contemporary family therapy.

FARADIC. A type of discontinuous alternating electric current sometimes used in aversion therapy. It is named for Michael Faraday, an eminent British physicist.

FASCIA (PLURAL, FASCIAE). A band or sheath of connective tissue that covers, supports, or connects the muscles and the internal organs.

FECES. Waste products eliminated from the large intestine; excrement.

FEEDBACK. A reaction or response from others to a particular behavior or activity.

FEEDBACK LOOP. A naturally occurring process whereby individuals control their behavior by self-monitoring, self-evaluation, and self-reinforcement.

FEEDBACK LOOPS. Chains of biochemical reactions in which the products of reactions limit or enhance the subsequent reactions, and in which the chain ends up back at the first reaction, either limiting or enhancing it.

FEMININITY. Prescribed behavior for females, characterized by interpersonal warmth, passivity, and lack of aggression.

FENFLURAMINE (PONDIMIN). A prescription appetite suppressant for weight loss that was withdrawn from the market due to unacceptable health risks.

FETISHISM. A paraphilia in which a person requires a nonliving object (or occasionally a nongenital part of the body, such as the partner's feet) in order to achieve sexual arousal and satisfaction.

FIRST-RANK SYMPTOMS. A list of symptoms that have been considered to be diagnostic of schizophrenia. They include, delusions, somatic hallucinations, hearing voices commenting on one's behavior, and thought insertion or withdrawal. First-rank symptoms are sometimes called Schneiderian symptoms, after the name of Kurt Schneider, the German psychiatrist who listed them in 1959.

FLASHBACK. The re-emergence of a traumatic memory as a vivid recollection of sounds, images, and sensations associated with the trauma. The person having the flashback typically feels as if he or she is reliving the event.

FLAVONOIDS. Plant pigments that have a variety of effects on human physiology. Some of these pigments have anti-inflammatory, anti-carcinogenic, and antioxidant effects, for example.

FLOODING. A type of exposure treatment in which the patient is exposed to an anxiety-provoking or feared situation all at once and kept in it until the anxiety and fear subside.

FLUID INTELLIGENCE. A type of intelligence that involves inductive and deductive reasoning ability.

FOLIC ACID. An essential B-vitamin that humans obtain through diet.

FOOD FREQUENCY QUESTIONNAIRE. A listing of how often a person consumes foods from certain food groups in a given period of time.

FORENSIC. Pertaining to courtroom procedure or evidence used in courts of law.

FORMICATION. The sensation of bugs creeping on the skin.

FREE-FLOATING. A term used in psychiatry to describe anxiety that is unfocused or lacking an apparent cause or object.

FREQUENCY DISTRIBUTION. In statistics, the correspondence between a set of frequencies and the set of categories used to classify the group being tested.

FRONTAL CORTEX. The part of the human brain associated with aggressiveness and impulse control. Abnormalities in the frontal cortex are associated with an increased risk of suicide.

FRONTAL LOBE DEMENTIA. Dementia caused by a disorder, usually genetic, that affects the front portion of the brain.

FRONTAL LOBES. A region of the brain that influences higher mental functions often associated with intelligence, such as the ability to foresee the consequences of actions, planning, comprehension, and mood.

FROTTAGE. The act of touching or rubbing against the body or genitals of a non-consenting individual.

FUGUE. A dissociative experience during which a person travels away from home, has amnesia for their past, and may be confused about their identity but otherwise appears normal.

FUGUE STATE. A form of amnesia in which the person appears to be conscious and to make rational decisions, but upon recovery, the period is not remembered. Fugue states represent one type of reaction to traumatic experiences.

G

GABA. Gamma-aminobutyric acid, an inhibitory neurotransmitter in the brain.

GADOLINIUM. A very rare metallic element useful for its sensitivity to electromagnetic resonance, among other things. Traces of it can be injected into the body to enhance MRI pictures.

GALACTORRHEA. Lactation occurring in the absence of pregnancy.

GAMMA RAY. A high-energy photon emitted by radioactive substances.

GAMMA-AMINOBUTYRIC ACID (GABA). A neurotransmitter that helps to lower or reduce the level of excitement in the nerves, leading to muscle relaxation, calmness, sleep, and prevention of seizures.

GANSER SYNDROME. A rare subtype of factitious disorder accompanied by dissociative symptoms. It is most often seen in male patients under severe stress in prison or courtroom settings.

GANTRY. A name for the couch or table used in a CT scan. The patient lies on the gantry while it slides into the x-ray scanner.

GAS CHROMATOGRAPHY/MASS SPECTROMETRY (GC/MS). A definitive method of testing for specific drugs, used to confirm immunoassay results indicating drug use. GC/MS separates the substances present in the urine sample, then breaks them into unique molecular fragments, which are matched against a database of known substances.

GASTRITIS. Inflammation of the lining of the stomach.

GATEWAY DRUG. A mood-altering drug or substance, typically used by younger or new drug users, that may lead to the use of more dangerous drugs.

GENDER DYSPHORIA. A state of persistent discomfort or depression associated with one's gender role or biological sex.

GENDER ROLE CONFLICT OR STRESS. A negative psychological state resulting from a discrepancy between gender role expectations and how one actually thinks, feels, or behaves.

GENDER ROLES. Stereotypical expectations regarding how one should think, behave, and feel depending on whether one is male or female.

GENERALIZATION. A person's ongoing use of new behaviors that were previously modeled for him or her. Generalization is also called transfer of training or maintenance.

GENERALIZED ANXIETY DISORDER. A general form of fear that can dominate a person's life.

GENERIC. A term that refers to a medication that is not protected by a registered trademark.

GENETIC POOL. The genetic material of an entire population.

GENOGRAM. A family tree diagram that represents the names, birth order, sex, and relationships of the members of a family. Therapists use genograms to detect recurrent patterns in the family history and to help the members understand their problem(s).

GENOME. The total genetic makeup of a cell or organism. The human genome is the complete genetic constitution of a human being.

GENOMIC IMPRINTING. The process in which specific genes or DNA segments are modified during the development of sperm or egg cells in a parent-specific fashion. The modification is reversible and appears to include the addition or removal of methyl groups to specific areas within the DNA sequence.

GENOTYPE. The genetic makeup of an organism or a set of organisms.

GESTALT. A German word that means "form" or "structure." The Gestalt Closure subtest on the K-SNAP measures a person's ability to identify a whole object from a partially completed drawing of its form.

GESTALT THERAPY. A therapeutic approach that focuses on increasing awareness of feelings and impulses in the present.

GHB. GHB, or gamma hydroxybutyrate, is a central nervous system depressant that has been abused in the United States for euphoric, sedative, body-building, and date-rape purposes.

GILLBERG'S CRITERIA. A six-item checklist for AS developed by Christopher Gillberg, a Swedish researcher. It is widely used in Europe as a diagnostic tool.

GINGKO BILOBA. A shade tree native to China with fan-shaped leaves and fleshy seeds with edible kernels. Gingko extract is being studied as a possible complementary or adjunctive treatment for Alzheimer's.

GINSENG ABUSE SYNDROME. A group of symptoms recognized by Chinese physicians as the result of excessive use of ginseng. The symptoms include dizziness, high blood pressure, restlessness, nausea, possible bleeding from the digestive tract, and skin rashes.

GLANS. The tip of the penis.

GLAUCOMA. A group of eye diseases characterized by increased pressure within the eye significant enough to damage eye tissue and structures. If untreated, glaucoma results in blindness.

GRAND MAL SEIZURE. A seizure characterized by a sudden loss of consciousness that is immediately followed by generalized convulsions. Such a seizure is usually preceded by a sensory experience, called an aura, which provides a warning as to an impending convulsion.

GRANDIOSE. Having an exaggerated belief in one's importance or status. In some people, grandiosity may be so extreme as to be delusional.

GRANDIOSE DELUSIONS. Grandiose delusions magnify the person's importance; the delusional person may believe himself or herself to be a famous person, to have magical superpowers, or to be someone in a position of enormous power (such as being the Prime Minister or President).

GRANDIOSITY. Exaggerated and unrealistic self-importance; inflated self-assessment. Grandiosity is considered one of the core characteristics of persons diagnosed with narcissistic personality disorder.

GRIDIRON ABDOMEN. An abdomen with a network of parallel scars from repeated surgical operations.

GROUP COHESIVENESS. The degree to which a group functions well in its assigned task.

GROUP PSYCHOTHERAPY; GROUP THERAPY. A form of therapy in which a small, carefully selected group of individuals meets regularly with a therapist to assist each individual in emotional growth and personal problem-solving. The group provides support and correction through feedback, constructive criticism, and a forum for consultation and reference.

GUANETHIDINE. A medication used to treat high blood pressure.

GUIDED IMAGERY. Techniques in which individuals actively imagine themselves in a scene (usually a different location, such as a relaxing beach, or a trigger situation where one handles the situation successfully), typically guided by another person describing the scene.

H

HABITUATION. The reduction of a person's emotional or behavioral reaction to a cue by repeated or prolonged exposure.

HALF-LIFE. The time required for half of the atoms in a radioactive substance to disintegrate.

HALLUCINATIONS. False sensory perceptions. A person experiencing a hallucination may "hear" sounds or "see" people or objects that are not really present.

Hallucinations can also affect the senses of smell, touch, and taste.

HALLUCINOGENS. Substances that cause hallucinations.

HASHISH. The dark, blackish resinous material that exudes from the leaves of the Indian hemp plant.

HATHA YOGA. The form of yoga most familiar to Westerners; often practiced as a form of physical therapy.

HEALTH MAINTENANCE ORGANIZATION (HMO). A type of managed care system that involves payment contracts with a group or panel of health care providers.

HEALTH MAINTENANCE ORGANIZATION ACT OF 1973. Federal legislation that provided aid to develop HMOs.

HEBEPHRENIC SCHIZOPHRENIA. An older term for what is now known as the disorganized subtype of schizophrenia.

HEMATEMESIS. Vomiting blood. Hematemesis is a symptom that sometimes occurs with gastrointestinal ulcers made worse by high levels of caffeine consumption.

HEMATOMA. An accumulation of blood, often clotted, in a body tissue or organ, usually caused by a break or tear in a blood vessel.

HEMISPHERE. One side of the brain, right or left.

HEPATITIS. An inflammation of the liver that can be caused by a variety of factors.

HIB DISEASE. An infection caused by *Haemophilus influenza*, type b (Hib). This disease mainly affects children under the age of five. In that age group, it is the leading cause of bacterial meningitis, pneumonia, joint and bone infections, and throat inflammations.

HIERARCHY. In exposure therapy, a list of feared items or situations, ranked from least fearsome to most fearsome.

HIGH-DENSITY SEX OFFENSES. Several offenses within a short period of time.

HIGH-FUNCTIONING AUTISM (HFA). A subcategory of autistic disorder consisting of children diagnosed with IQs of 70 or higher.

HIPPOCAMPUS. A part of the brain that is involved in memory formation and learning. The hippocampus is shaped like a curved ridge and belongs to an organ system called the limbic system.

HISTAMINE. Substance released during allergic reactions.

HISTRIONIC. Theatrical.

HOLDING THERAPY. A controversial treatment for autism, reactive attachment disorder, and other problems of children in which an adult holds a child despite any resistance from the child until the child submits and experiences an emotional release.

HOLISTIC. An approach to health care that emphasizes the totality of an individual's spiritual, psychological, and physical, well-being, and that situates a disease or disorder within that totality.

HOMEOSTASIS. The tendency of a family system to maintain internal stability and resist change.

HOMOCYSTEINE. A chemical that builds up in the blood when methionine is not properly processed. High blood levels of homocysteine increase risk of heart disease and stroke.

HOST. The dominant or main alter in a person with dissociative identity disorder.

HUMAN POTENTIAL MOVEMENT. A movement dating back to the beginning of the 1900s that reflected an altered perspective of human nature from inherently corrupt to inherently good.

HUMANISTIC AND EXISTENTIAL THERAPIES. Therapies that focus on achieving one's full potential, guided by subjective experience.

HUMOR. In ancient medicine, one of four body fluids (blood, phlegm, yellow bile, and black bile) that were thought to determine a person's basic constitution and personality.

HUMORAL. A term describing a hormonal substance secreted by an endocrine gland (such as the thyroid).

HUNTINGTON'S DISEASE. A hereditary disorder that appears in middle age and is characterized by gradual brain deterioration, progressive dementia, and loss of voluntary movement. It is sometimes called Huntington's chorea.

HYDRATED. Combining a substance with water.

HYDROCEPHALUS. The accumulation of cerebrospinal fluid (CSF) in the ventricles of the brain.

HYDROGEN. The simplest, most common element known in the universe. It is composed of a single electron (negatively charged particle). It is the nuclear proton of hydrogen that makes MRI possible by reacting resonantly to radio waves while aligned in a magnetic field.

HYDROMORPHONE. A prescribed opiate (Dilaudid) used to treat severe pain; also abused illegally.

HYPERACTIVE. Behavior disturbances, usually in children and adolescents, that involves impulsiveness, low levels of concentration, and distractibility.

HYPERAROUSAL. A symptom of traumatic stress characterized by abnormally intense reactions to stimuli. A heightened startle response is one sign of hyperarousal.

HYPEREMESIS GRAVIDARUM. Uncontrollable nausea and vomiting associated with pregnancy. Acupuncture appears to be an effective treatment for this condition.

HYPERPHAGIA. An abnormally large appetite for food. Hyperphagia is one of the symptoms of Prader-Willi syndrome.

HYPERSENSITIVE INTERNAL SUFFOCATION ALARM. A sensitive alarm goes off and the affected person's brain sends the body false signals that not enough oxygen is being received, which causes an increase in their breathing rate.

HYPERTENSION. High blood pressure, often brought on by smoking, obesity, or other causes; one of the major causes of strokes.

HYPERTHERMIA. Elevated body temperature resulting from ingestion of amphetamines.

HYPERTHYROIDISM. Condition resulting from the thyroid glands secreting excessive thyroid hormone, causing increased basal metabolic rate, and causing an increased need for food to meet the demand of the metabolic activity; generally, however, weight loss results.

HYPERVENTILATION. A pattern of rapid, shallow breathing that is frequently found in patients with Rett's disorder.

HYPERVIGILANCE. A state of abnormally intense wariness or watchfulness that is found in survivors of trauma or long-term abuse. Hypervigilance is sometimes described as "being on red alert all the time."

HYPERVIGILANT. Extreme attention to both internal and external stimuli.

HYPNAGOGIC HALLUCINATIONS. Dream-like auditory or visual hallucinations that occur while a person is falling asleep.

HYPNOSIS. The means by which a state of extreme relaxation and suggestibility is induced; used to treat amnesia and identity disturbances that occur in dissociative disorders.

HYPNOTHERAPY. The use of an induced trance state, or hypnosis, as a therapy.

HYPNOTIC. A type of medication that induces sleep.

HYPOCHONDRIASIS. A mental condition in which the affected person perceives illness or symptoms of illness when none exist.

HYPOGONADISM. Abnormally decreased gonad function with retardation of sexual development.

HYPOKALEMIA. Abnormally low levels of potassium in the blood. Hypokalemia is a potential medical emergency, as it can lead to disturbances of the heart rhythm. Muscle cramps and pain are a common symptom of hypokalemia in bulimic patients.

HYPOKINESIA. A condition of abnormally diminished motor activity.

HYPOMANIA. A milder form of mania which is characteristic of bipolar II disorder.

HYPONATREMIA. A condition characterized by an abnormally low concentration of sodium in the blood.

HYPOPNEA. Breathing that is too shallow to maintain adequate levels of oxygen in the blood.

HYPOTENSION. Low blood pressure.

HYPOTHALAMIC-PITUITARY-ADRENAL (HPA) SYSTEM. A part of the brain involved in the human stress response. The HPA system releases cortisol, the primary human stress hormone, and neurotransmitters that activate other brain structures associated with the "fight-or-flight" reaction. The HPA system appears to function in abnormal ways in patients diagnosed with depersonalization disorder. It is sometimes called the HPA axis.

HYPOTHALAMUS. A part of the forebrain that controls heartbeat, body temperature, thirst, hunger, blood pressure, blood sugar levels, and other functions.

HYPOTHESIS. An assumption, proposition, or educated guess that can be tested empirically.

HYPOTHYROIDISM. Thyroid gland that is abnormally low-functioning. A lowered metabolic rate results.

HYPOVENTILATION. An abnormally low level of blood oxygenation in the lungs.

HYSTERIA. In nineteenth-century psychiatric use, a neurotic disorder characterized by violent emotional outbursts and disturbances of the sensory and motor (movement-related) functions. The term "hysterical neurosis" is still used by some psychiatrists as a synonym for conversion disorder.

I

ID. A construct in Freudian psycho dynamic theory that represents the irrational, self-centered aspects of human thought.

IDEAL WEIGHT. A range of body weights recommended for generally healthy adults.

IDENTIFIED PATIENT (IP). The family member in whom the family's symptom has emerged or is most obvious.

IDENTITY DIFFUSION. A character formation that is scattered or spread around rather than an identity that becomes solidified or consolidated.

IDIOGRAPHIC. An approach to interpreting the results of a projective test within the context of the individual subject's record.

IEP. *See* Individualized Education Plan

ILLUSION. A misperception or misinterpretation in the presence of a real external stimulus.

IMITATIVE BEHAVIOR. Behaviors of a therapist or group member that are imitated, consciously or unconsciously, by other group members.

IMMUNOASSAY. The method used in routine or preliminary urine drug screening.

IMMUNOSUPPRESSANT. Medications that suppress or lower the body's immune system, primarily used to help the body accept a transplanted organ.

IMPLICIT. Implied or suggested without being clearly stated. Some critics of *DSM-IV-TR* maintain that its contributors based the criteria sets for certain disorders on an implicit notion of a mentally healthy human being.

IMPLICIT MEMORY. Unconsciously recalled memory for skills, procedures, or associations.

IMPULSE CONTROL DISORDERS. Group of disorders characterized by impulsive behavior, such as stealing.

IN VIVO. A Latin phrase that means "in life." In modeling and exposure therapies, it refers to practicing new behaviors in a real setting, as distinct from using imagery or imagined settings.

INBORN ERROR OF METABOLISM. A rare enzyme deficiency; children with inborn errors of metabolism do not have certain enzymes that the body requires to maintain organ functions. Inborn errors of metabolism can cause brain damage and mental retardation if left untreated. Phenylketonuria is an inborn error of metabolism.

INCEST. Unlawful sexual contact between persons who are biologically related. Many therapists, however, use the term to refer to inappropriate sexual contact between any members of a family, including stepparents and stepsiblings.

INCISORS. The four teeth in the front of each jaw in humans. The incisors of patients with bulimia frequently show signs of erosion from stomach acid.

INCONTINENCE. Inability to control the release of urine or feces.

INDEMNITY INSURANCE. Insurance plans that pay on a fee-for-service basis in the event of illness or injury.

INDICES. Scores based on performance in more than one area. On the WAIS, there are four index scores, each based on an individual's performance in more than one subtest.

INDIVIDUALIZED EDUCATION PLAN (IEP). A plan of instruction drawn up for an individual student who is having specific difficulties with mathematics, reading, or other skills necessary to progress beyond elementary school.

INDIVIDUAL PSYCHOTHERAPY. A relationship between therapist and patient designed to foster the patient's emotional growth and personal problem-solving skills.

INFORMATION GIVING. Imparting of information about a disease or condition as part of the therapeutic process.

INFORMED CONSENT. A person's agreement to undergo a medical or surgical procedure, or to participate in a clinical study, after being properly advised of the medical facts related to the procedure or study and the risks involved.

INFUSION. The most potent form of extraction of an herb into water. Infusions are steeped for a longer period of time than teas.

INHALANTS. A class of drugs that are inhaled in order for the user to experience a temporary "high." These chemicals include volatile solvents (liquids that vaporize at room temperature) and aerosols (sprays that contain solvents and propellants), and include glue, gasoline, paint thinner, hair spray, and others. They are dangerous because they can cause hallucinations, delusions, difficulty breathing, headache, nausea, vomiting, and even "sudden sniffing death." Inhalants can also cause permanent damage to the brain, lung, kidney, muscle, and heart.

INSIDIOUS. Proceeding gradually and inconspicuously but with serious effect.

INSOMNIA. A chronic inability to sleep or to remain asleep throughout the night.

INSULIN RESISTANCE. The body's inability to utilize blood sugar, at times leading to diabetes.

INSULT. In medicine, an injury or trauma to the brain or other part of the body.

INTEGRATED SETTING. Placing individuals in typical employment situations rather than making placements into sheltered workshops or other segregated settings.

INTELLIGENCE QUOTIENT (IQ). A measurement of intelligence obtained by dividing a person's mental age (determined by level of performance on an age-graded test) by his or her chronological age and multiplying by 100. For example, a ten-year-old with a mental age of thirteen would have an IQ of 130.

INTER-RATER RELIABILITY. The degree to which judgments about a person are consistent among raters or diagnosticians.

INTERMEDIATE CARE FACILITY. An inpatient facility that provides periodic nursing care.

INTERNALIZING DISORDERS. Mental disorders with primary symptoms that involve inner emotions as opposed to outward behavior.

INTEROCEPTIVE. Referring to stimuli or sensations that arise inside the body. In interoceptive exposure treatment, the patient is asked to exercise or perform other actions that produce feared internal physical sensations.

INTERPERSONAL LEARNING. Learning that takes place via feedback from others.

INTERPERSONAL THERAPY. An approach that includes psychoeducation about the sick role, and emphasis on the present and improving interpersonal dynamics and relationships. Interpersonal therapy is effective in treating adjustment disorders related to physical illness.

INTOXICATION. The condition of being drunk.

INTRAMUSCULAR. An injection that is given into a muscle.

INTRAPSYCHIC. Occurring inside a person's mind or psyche.

INTRON. A segment of DNA that interrupts an exon and that does not encode any information about the protein sequence.

IONIZING RADIATION. Electromagnetic radiation that can damage living tissue by disrupting and destroying individual cells. All types of nuclear decay radiation (including x rays) are potentially ionizing. Radio waves do not damage organic tissues they pass through.

IPECAC. The dried root of *Caephalis ipecacuanha*, a South American plant. Given in syrup form, ipecac is most commonly used to induce vomiting in cases of accidental poisoning.

IRRITABLE BOWEL SYNDROME (IBS). A condition affecting the small and large intestine, usually associated with emotional stress. There may be complaints of diarrhea and pain in the lower abdomen.

ISCHEMIA. Localized anemia of tissues due to obstructed inflow of blood.

J

JAUNDICE. A yellowing of the skin caused by excess bilirubin in the blood; a liver disorder.

JOURNALING. Involves writing out thoughts and feelings in an unstructured format. A "stream of consciousness" approach (writing whatever comes to mind) is suggested for greatest effectiveness.

K

KAVALACTONES. Medically active compounds in kava root that act as local anesthetics in the mouth and as minor tranquilizers.

KAVAPYRONES. Compounds in kava root that act as muscle relaxants and anticonvulsants.

KETAMINE. An anesthetic, used predominantly by veterinarians to treat animals, that can be used as a date-rape drug.

KI. The Japanese spelling of qi, the traditional Chinese term for vital energy or the life force.

KILOGRAM. A metric unit of weight. It equals 2.2 lb.

KLEPTOMANIA. A disorder of impulse control characterized by repeated stealing or shoplifting of items that the person does not need.

KLINEFELTER'S SYNDROME. A genetic disorder in males characterized by the presence of an extra X chromosome in addition to the normal XY. Most men with Klinefelter's syndrome have learning problems, are sterile, and have a shortened life expectancy.

KORSAKOFF'S SYNDROME. A disorder of the central nervous system resulting from long-term thiamine deficiency. It is characterized by amnesia, confusion, con-

fabulation, and unsteady gait, and is most commonly seen in alcoholics.

KUNDALINI. In Indian yoga, a vital force or energy at the base of the spine that is activated or released by certain yoga postures or breathing techniques. This release is called the "awakening" of the kundalini. Some Westerners have had kundalini experiences that were diagnosed as psychotic episodes or symptoms of schizophrenia.

L

(LA) BELLE INDIFFÉRENCE. A psychiatric symptom sometimes found in patients with conversion disorder, in which the patient shows a surprising lack of concern about the nature or implications of his/her physical symptom.

LABIA. The outside folds of tissue that surround the clitoris and the opening of the urethra in women.

LABILE. Subject to frequent change, particularly in reference to mood.

LANUGO. Downy hair, usually associated with infants, that sometimes develops on the face and back of people affected by anorexia nervosa.

LAPSE. A single, isolated occurrence of a symptom or negative behavior.

LARYNGOSPASM. Spasms that close the vocal apparatus of the larynx (the organ of voice production).

LATERALIZATION. The control of specific neurological functions by one side of the brain or the other; for example, in most right-handed people, language functions are controlled by the left side of the brain and spatial and visual functions are controlled by the right side of the brain.

LAXATIVE. Substance or medication that encourages a bowel movement.

LEAST RESTRICTIVE ENVIRONMENT. Refers to care options that involve the least amount of restraint and the greatest degree of independence possible, while still meeting the individual's needs and maintaining safety.

LEUCOTOMY OR LEUKOTOMY. White matter cutting—severing the white matter of the frontal lobe of the brain.

LEUKODYSTROPHY. A disturbance of the white matter of the brain.

LEWY BODIES. Areas of injury found on damaged nerve cells in certain parts of the brain associated with dementia. Lewy body dementia was first ecognized in the 1980s and is now distinguished from Alzheimer's disease.

LEWY-BODY DISEASE. A type of dementia that resembles Alzheimer's disease, but progresses more rapidly. Common symptoms include fluctuations in confusion and recurring visual hallucinations. In this disease, abnormal brain cells are distributed throughout the brain.

LIBIDO. Psychic energy or instinctual drive associated with sexual desire, pleasure, or creativity.

LIMBIC SYSTEM. A group of structures in the brain that includes the amygdala, hippocampus, olfactory bulbs, and hypothalamus. The limbic system is associated with homeostasis and the regulation and arousal of emotions.

LIMITED SUPPORT. A predetermined period of assistance required to deal with a specific event, such as training for a new job.

LOBOTOMY. A surgical procedure involving the cutting of nerve fiber bundles in the brain.

LOCALIZATION. The control of specific neurological functions by specific areas in the brain.

LOCUS CERULEUS. A part of the brain where the neurotransmitter causes excitation.

LOFEXIDINE. A medication approved for use in England to aid the opioid detoxification process.

LOW AFFECT. Severe lack of interest and emotions; emotional numbness.

LUX. The International System unit for measuring illumination, equal to one lumen per square meter.

M

MACERATION. A technique for extracting essential oils from plant leaves and stems by crushing the plant parts and soaking them in warm vegetable oil.

MACHISMO. The exaggerated image of extreme masculinity that includes such qualities as concern for personal honor, virility, physical strength, heavy drinking, toughness, aggression, risk taking, authoritarianism, and self-centeredness.

MACROSOCIAL. Pertaining to the wider society, as distinct from such smaller social groupings as families, neighborhoods, etc.

MAGNETIC FIELD. The three-dimensional area surrounding a magnet, in which its force is active. During

MRI, the patient's body is permeated by the force field of a superconducting magnet.

MAINTENANCE TREATMENT. The period of treatment beginning after the initial introduction of the treatment medication. During this period, the dose of medication can be either increased or decreased, depending on the program and needs of the patient.

MALADROITNESS. Another word for awkwardness or clumsiness.

MALAISE. The medical term for a general condition of unease, discomfort, or weakness.

MALINGERING. Knowingly pretending to be physically or mentally ill to avoid some unpleasant duty or responsibility, or for economic benefit.

MANIA. An elevated or euphoric mood or irritable state that is characteristic of bipolar I disorder. This state is characterized by mental and physical hyperactivity, disorganization of behavior, and inappropriate elevation of mood.

MANIC. Referring to mania.

MANTRA. Originally, a sacred word or phrase repeated over and over to help focus the mind during meditation; in the Western world, this may refer to any repeated syllable, word, or phrase used to meditate.

MAO INHIBITORS. A group of antidepressant drugs that decrease the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

MARIJUANA. The dried and shredded or chopped leaves of the Indian hemp plant.

MASCULINITY. Prescribed behavior for males, characterized by independence, strength, control, and avoidance of emotional expressiveness.

MASOCHISM. A mental disorder in which a person obtains sexual satisfaction through pain or humiliation inflicted by the self or by another person. The term is sometimes used more generally to refer to a tendency to find pleasure in submissiveness or self-denial.

MASOCHISTIC TENDENCIES. Tendencies to direct harm or hatred toward oneself.

MAZINDOL (SANOREX, MAZANOR). A prescription medication for weight loss currently on the market.

MEAN. The mathematical average of all scores in a set of scores.

MEDICAID. A program jointly funded by state and federal governments that reimburses hospitals and physicians for the care of individuals who cannot pay for their own medical expenses. These individuals may be in low-income households or may have chronic disabilities.

MEDICAID HOME AND COMMUNITY-BASED WAIVER. Legislation regarding the use of Medicaid funds for care services; allows certain federal requirements to be bypassed so that states can use the funds more flexibly for accessing home- and community-based services rather than using hospitals or intermediate-care facilities.

MEDICAL MODEL. The basic conceptual framework in the West since the nineteenth century for understanding, researching, and classifying mental disorders.

MEDICARE. A federally funded health insurance program for individuals age 65 and older or certain categories of younger persons with disabilities.

MEDROXYPROGESTERONE. A progestin, a female hormone.

MEDROXYPROGESTERONE ACETATE (MPA). A female hormone that may be prescribed for male patients with sexual sadism or other paraphilias. MPA helps to control sexual urges in men by speeding up the clearance of testosterone from the bloodstream.

MELANCHOLIA. A form of severe depression characterized by weight loss, insomnia, and an inability to experience pleasure.

MENINGES. A membrane covering the brain and spinal cord that consists of three layers: the *pia mater,* the innermost layer; the *arachnoid,* in the middle; and the *dura mater,* the outermost layer.

MENOPAUSE. A period of decreasing hormonal activity in women, when ovulation stops and conception is no longer possible.

MERIDIANS. In traditional Chinese medicine, a network of pathways or channels that convey qi (also sometimes spelled "ki"), or vital energy, through the body.

MERYCISM. Another name for rumination disorder.

META-ANALYSIS. The statistical analysis of a large collection of analyses from individual studies for the purpose of integrating the findings.

METABOLISM. The group of biochemical processes within the body that release energy in support of life.

METHADONE. A drug often prescribed legally as a replacement for heroin. It induces a slight high but blocks heroin from producing a more powerful euphoric effect. It may be used in heroin detoxification to ease the process, or it may be used daily after detoxification as maintenance therapy. Methadone maintenance therapy is controversial.

METHAMPHETAMINE. The most common illegally produced amphetamine.

METHIONINE. An amino acid that, when not metabolized properly, allows homocysteine to build up in the blood. Folic acid aids methionine metabolism.

METHYLPHENIDATE. A mild central nervous system stimulant that is used to treat hyperactivity.

MIDDLE NOTE. A term used in perfumery and aromatherapy to designate essential oils whose odors emerge later than top notes but evaporate more rapidly than bottom notes. Chamomile is considered a middle note in aromatherapy.

MILD COGNITIVE IMPAIRMENT (MCI). A transitional phase of memory loss in older people that precedes dementia or Alzheimer's disease.

MILLIGRAM (MG). One-thousandth of a gram. A gram is the metric measure that equals about 0.035 ounces.

MILLON CLINICAL MULTIAXIAL INVENTORY (MCMI-II). A self-report instrument designed to help the clinician assess DSM-IV-related personality disorders and clinical syndromes. It provides insight into 14 personality disorders and 10 clinical syndromes.

MINNESOTA MULTIPHASIC PERSONALITY INVENTORY (MMPI-2). A comprehensive assessment tool widely used to diagnosed personality disorders.

MIXED MANIA/MIXED STATE. A mental state in which symptoms of both depression and mania occur simultaneously.

MODALITY. One of the primary forms of sensation, such as vision, touch, or hearing.

MODELING. A type of teaching method used in social skills training. Therapists who use this method may offer positive and negative examples of the behaviors that make up a social skill.

MONOAMINE OXIDASE INHIBITORS (MAOIS). A group of antidepressant drugs that decrease the activity of monoamine oxidase, a neurotransmitter found in the brain that affects mood.

MONOGENIC. Determined or controlled by a single gene. Huntington's disease is one of the few psychiatric disorders that is monogenic.

MONOMANIA. A nineteenth-century term for a pathological obsession with one idea or one social cause. Nineteenth-century psychiatrists often associated explosive behavior with monomania. The word is no longer used as a technical term.

MONOZYGOTIC. Developed from a single fertilized ovum. Monozygotic twins are sometimes called identical twins.

MORBIDITY. The unhealthiness or disease characteristics associated with a mental disorder.

MOSAICISM. A genetic condition in which some cells in an organism have one set of chromosomes and other cells have a different set.

MOTIVATIONAL ENHANCEMENT THERAPY. Therapy that focuses on increasing motivation for change by empathically comparing and contrasting the consequences and benefits of changing or not changing.

MOTOR. Involving muscle movement.

MOTOR SKILLS. Skills pertaining to or involving muscular movement.

MOVEMENT EDUCATION. A term that refers to the active phase of bodywork, in which clients learn to move with greater freedom and to maintain the proper alignment of their bodies.

MOXIBUSTION. A technique in traditional Chinese medicine that involves burning a *Moxa*, or cone of dried wormwood leaves, close to the skin to relieve pain. When used with acupuncture, the cone is placed on top of the needle at an acupuncture point and burned.

MRI. Magnetic resonance imaging. A special imaging technique used to image internal parts of the body, especially soft tissues.

MULTI-INFARCT DEMENTIA. Dementia caused by damage to brain tissue resulting from a series of blood clots or clogs in the blood vessels. It is also called vascular dementia.

MULTIAXIAL. Refers to a type of classification system that involves numeric measurement along more than one dimension and is not based on assignment to mutually exclusive categories.

MULTIMODAL. Involving several types of therapeutic interventions such as heat or ice packs, electrical stimulation, and ultrasound; sometimes refers to a mix of physical and psychological therapies.

MULTIPLE PERSONALITY DISORDER (MPD). An older term for dissociative identity disorder (DID).

MULTIPLE SCLEROSIS. A disease characterized by patches of hardened tissue in the brain or spinal cord, paralysis, and/or muscle tremors.

MUSCLE DYSMORPHIA. A subtype of BDD, described as excessive preoccupation with muscularity

and bodybuilding to the point of interference with social, educational, or occupational functioning.

MUTATION. A spontaneous change in the sequence of nucleotides in a chromosome or gene. Mutations may affect the number and structure of chromosomes or cause deletions of part of a chromosome. Rett's disorder is caused by a mutation on the long arm of the X chromosome.

MUTISM. Inability to speak due to conscious refusal or psychological inhibition.

MYASTHENIA GRAVIS. A disease characterized by weakness of the muscles caused by an autoimmune reaction.

MYELIN SHEATHS. A fatty layer around nerve cells that aids the transmission of nerve impulses.

MYOCARDIAL DISEASE. Disease of the muscular layer of the heart wall.

MYOCLONUS. An abrupt spasm or twitching in a muscle or group of muscles. It is more common in early-onset AD than in late-onset Alzheimer's.

N

NARCISSISTIC PERSONALITY DISORDER. Personality characterized by continually exaggerating one's own positive qualities and refusing to recognize personal defects or flaws.

NARCISSISTIC PERSONALITY INVENTORY (NPI). The most widely used English-language diagnostic instrument for narcissistic personality disorder. Based on the *DSM-III* criteria for NPD, the NPI is frequently used in research studies as well as patient assessment.

NARCOLEPSY. A disorder characterized by frequent and uncontrollable attacks of deep sleep.

NARCOTHERAPY. A form of psychotherapy that involves the administration of a drug that makes the patient drowsy.

NALOXONE. A drug that combines competitively with opiate receptors on the nerve cells and blocks or reverses the action of narcotic analgesics.

NARROW-ANGLE GLAUCOMA. An eye disorder caused by a buildup of fluid pressure inside the eyeball due to an abnormally small angle between the iris (the colored portion of the eye) and the cornea (the transparent front part of the eye).

NATURAL SUPPORTS. Using a person's already existing support network to help the person reach a goal, such as the employment of their choice.

NEGATIVE SYMPTOMS. Symptoms of schizophrenia that represent a loss or reduction of normal functioning.

NEPHRITIS. Inflammation of the kidney.

NERVOUS TIC. A repetitive, involuntary action, such as the twitching of a muscle or repeated blinking.

NEUROFIBRILLARY TANGLES. Accumulations of twisted protein fragments found inside the nerve cells in the brains of Alzheimer's patients.

NEUROLEPTIC. Another name for the older antipsychotic medications, such as haloperidol (Haldol) and chlorpromazine (Thorazine).

NEUROLEPTIC MALIGNANT SYNDROME (NMS). An unusual but potentially serious complication that develops in some patients who have been treated with antipsychotic medications. NMS is characterized by changes in blood pressure, altered states of consciousness, rigid muscles, and fever. Untreated NMS can result in coma and death.

NEUROLEPTIC-INDUCED ACUTE DYSTONIA. A severe form of dystonia, a neurological movement disorder, caused by the use of neuroleptic drugs.

NEUROLEPTIC-INDUCED AKATHISIA. Refers to the disorder characterized by a physical restlessness (the inability to sit still, for example), and excessive voluntary movements, as a result of the use of neuroleptic drugs; research indicates it is likely the most common of neuroleptic-induced movement disorders.

NEUROLEPTIC-INDUCED PARKINSONISM. Symptoms similar to Parkinson's disease that may appear in people taking neuroleptic (antipsychotic) medications. These symptoms include tremors in muscles and a shuffling gait.

NEUROLEPTIC-INDUCED TARDIVE DYSKINESIA. A potentially irreversible neurological disorder caused by the use of antipsychotic/neuroleptic medications, with symptoms involving uncontrollable movement of various body parts. Some of these movements include involuntary movements of the tongue and mouth, grimacing, and lip-smacking.

NEUROLOGIC. Pertaining to the nervous system (brain and nerve cells).

NEURONS. Nerve cells in the brain that produce nerve impulses.

NEUROPATHIC. Relating to neural damage.

NEUROPSYCHOLOGICAL FUNCTIONING. The ability of the nervous system and brain to process and interpret information received through the senses.

NEUROTRANMISSION. The conduction of a nerve impulse along a chain of nerve cells, which occurs when a cell in the chain secretes a chemical substance, called a neurotransmitter, onto a subsequent cell.

NEUROTRANSMITTER. A chemical in the brain that transmits messages between neurons, or nerve cells.

NEUROTRANSMITTERS. Chemicals that carry nerve impulses from one nerve cell to another. Alzheimer's disease causes a drop in the production of several important neurotransmitters.

NIMBY PHENOMENON. Acronym for Not In My Backyard, describing the common opposition displayed by citizens toward the placement of group homes or other social service facilities in their neighborhoods.

NOMOTHETIC. An approach to interpreting the results of a projective test in which the subject's answers are measured against a normative comparison sample.

NON-AMBULATORY. Unable to walk.

NON-DOMINANT HAND. The hand that one does not typically use when performing various tasks, such as writing or throwing an object.

NONDIRECTIVE THERAPY. An approach to therapy in which the therapist actively attempts to avoid giving advice, making interpretations, or otherwise influencing the focus of the individual's thoughts or statements.

NONENDOGENOUS. A factor that arises or is produced outside of the organism.

NORADRENERGIC. Acts similarly to norepinephrine or noradrenaline.

NOREPINEPHRINE. A neurotransmitter in the brain that acts to constrict blood vessels and raise blood pressure. It works in combination with serotonin.

NOREPINEPHRINE (NORADRENALINE). A hormone with similar stimulatory effects to epinephrine but, in contrast to epinephrine, has little effect on cardiac (heart) output and in relaxing smooth muscles.

NORMAL CURVE EQUIVALENTS. Standard scores with an average of 100. The normal curve equivalents divide the normal or bell-shaped curve into 100 equal parts. As a result, those scores can be used for statistical analysis because they can be added, subtracted, multiplied, and divided.

NORMED. Describes a process used in the developmental stages of a test instrument. The new test is first given to a cross-section of a population for which it is designed. The scores, placements, rankings, etc., of these persons then become the source for all future comparisons (norm group). When a new subject takes the test, his/her score, placement, ranking, etc., is determined based upon comparison with or deviation from the norm group.

NOSOLOGY. The branch of medicine that deals with the systematic classification of diseases and disorders.

NUCLEAR FAMILY. The basic family unit, consisting of father, mother, and their biological children.

NUCLEOTIDE. One of the molecules that form the building blocks of DNA or RNA. The nucleotides of DNA include a phosphate group, four chemical bases (adenine, cytosine, guanine, and thymine), and a sugar containing five carbon atoms. In RNA, the thymine base is replaced by uracil.

NYSTAGMUS. A persistent involuntary movement of the eyes from side to side. It is one of the symptoms of inhalant intoxication syndrome.

O

OBJECT RELATIONS. In psychology, a phrase that refers to the way in which a subject describes relationships with other people in their environment, and the ways in which he or she has internalized interpersonal relationships.

OBJECT-RELATIONS THEORY. An approach to psychological development that includes Nancy Chodorow's stating that children develop according to interactions with their primary caregivers.

OBSESSION. A persistent image, idea, or desire that dominates a person's thoughts or feelings.

OBSESSIVE-COMPULSIVE. Characterized by obsessive and compulsive behaviors.

OBSESSIVE-COMPULSIVE DISORDER. Disorder in which the affected individual has an obsession (such as a fear of contamination, or thoughts he or she doesn't like to have and can't control) and feels compelled to perform a certain act to neutralize the obsession (such as repeated handwashing).

OCCIPITAL. The occipital bone forms the back part of the skull.

OEDIPAL CONFLICT. A developmental conflict that emerges during the third or oedipal stage of Freud's psychosexual development stages. During this stage, a conflict emerges with regard to the triad of father, mother, and child. The conflict concerns the sexual impulses that the child has toward the parent of the opposite gender and the hostile impulses that the child has towards the

parent of the same gender. During this stage, the developmental conflict concerns a resolution of oedipal issues.

OLFACTORY NERVE. The cranial nerve that regulates the sense of smell.

ONSET. The point in time at which the symptoms of a disorder first became apparent.

OPERANT. Conditioning in which the desired response is reinforced by an introduced stimulus.

OPIATES. A class of drugs that is either derived from opium (i.e. morphine, hydromorphone, oxymorphone, heroin, codeine, hydrocodone, oxycodone) or resembles these opium derivatives (such as meperidine) and is commonly referred to as narcotics.

OPIOIDS. Substances that reduce pain and may induce sleep. Some opioids are endogenous, which means that they are produced within the human body. Other opioids are produced by plants or formulated synthetically in the laboratory.

OPPOSITIONAL DEFIANT DISORDER. An emotional and behavioral problem of children and adolescents characterized by defiant, hostile, or disobedient behavior that has lasted for longer than six months.

ORAL PHASE. The first of Freud's psychosexual stages of development in which satisfaction is focused on the mouth and lips. During this stage sucking and eating are the primary means of gratification.

ORGANIC BRAIN SYNDROME. A class of disorders characterized by progressive deterioration of mental processes caused by temporary brain dysfunction or permanent brain damage. Symptoms include delusions, dementia, amnesia, and delirium that are not caused by drugs, alcohol, or as a side effect of medication.

ORGASM. Another word for sexual climax.

ORIENTATION. In psychiatry, the ability to locate oneself in one's environment with respect to time, place and people.

ORLISTAT (XENICAL). A prescription medication for weight loss currently on the market.

ORTHOSTATIC HYPOTENSION. A sudden decrease in blood pressure due to a change in body position, as when moving from a sitting to standing position.

OSTEOPOROSIS. A loss of bone minerals.

OUTCOME EXPECTANCIES. What one believes will happen as a result of engaging in a certain behavior.

OVERCOMPENSATION. An attempt to overcome or correct a behavior by going too far in the opposite direction.

OVERVALUED IDEA. An unreasonable, sustained belief that is held with less than delusional intensity (i.e., the person can acknowledge, to some degree, that the belief may be false). The belief is not accounted for by the individual's cultural or religious background.

OXIMETRY. The measurement of blood oxygen levels.

P

PAIN DISORDER. One of several somatoform disorders described in the revised, fourth edition of the mental health professional's handbook, the *Diagnostic and Statistical Manual of Mental Disorders*. The term "somatoform" means that symptoms are physical but are not entirely understood as a consequence of a general medical condition or as a direct effect of a substance, such as a drug.

PAINSTATES. Refers to the four-way classification of pain disorder as being (1) acute with psychological factors, (2) acute with psychological factors and a general medical condition, (3) chronic with psychological factors, and (4) chronic with psychological factors and a general medical condition.

PANACEA. A medicine or other substance regarded as a cure for all ills.

PANIC ATTACK. Specific periods of time when a person has a feeling that s/he is dying or having a heart attack with chest pain, a feeling as though s/he could pass out, and fear that s/he is going insane.

PANIC DISORDER. An anxiety disorder in which an individual experiences sudden, debilitating attacks of intense fear.

PANIC DISORDER WITH AGORAPHOBIA. Repeated panic attacks in which the patient is worried about the attacks enough that the worry restricts their activity.

PANIC DISORDER WITHOUT AGORAPHOBIA. Repeated panic attacks without symptoms of agoraphobia.

PARAMETER. A characteristic or factor that is measured during a test of a complex process or activity like sleep.

PARANOIA. A mental disorder characterized by baseless suspicions or distrust of others, often delusional in intensity.

PARANOID. A mental attitude characterized by unjustified or excessive distrust of other people, usually combined with anger.

PARANOID PERSONALITY. A personality disorder characterized by unwarranted suspicion, jealousy, hypersensitivity, social isolation and a tendency to detect malicious intent in the words and actions of others.

PARAPHILIAS. A group of mental disorders that is characterized by recurrent intense sexual urges and sexually arousing fantasies generally involving (1) nonhuman objects, (2) the suffering or humiliation of oneself or one's partner (not merely simulated), or (3) children or other non-consenting persons.

PARASOMNIA. A type of sleep disorder characterized by abnormal changes in behavior or body functions during sleep, specific stages of sleep, or the transition between sleeping and waking.

PARESTHESIAS. Abnormal sensations of tingling or "pins and needles." Paresthesias are a common panic-like symptom associated with agoraphobia.

PARKINSON'S DISEASE. A disease of the nervous system most common in people over 60, characterized by a shuffling gait, trembling of the fingers and hands, and muscle stiffness. It may be related in some way to Lewy body dementia.

PARKINSONIAN. Related to symptoms associated with Parkinson's disease. These symptoms may be induced by certain medications, and, in these cases, the person does not have Parkinson's disease—they have Parkinson-like or Parkinsonian symptoms.

PASSIVE-AGGRESSIVE BEHAVIORS. Behaviors that represent covert expressions of hostile or negative feelings that the person is unable or unwilling to express directly.

PATHOGNOMONIC. Describing symptoms characteristic of a particular disease.

PATIENT CARE EPISODES. A specific measure of the volume of care provided by an organization or system. It begins with a treatment visit to a health care facility (a hospital or residential treatment center, for example) and ends when a person leaves the facility, so it may vary by patient and visit. Over time, the volume of patient care episodes indicates the degree to which a population uses certain health care capacities. Other measures that may be used to measure volume of care include number of beds or bed-days, total number of patients served, and also more specific measures like patient-contact hours.

PAVOR NOCTURNUS. The Latin medical term for sleep terror disorder.

PELVIS. The basin-like cavity in the human body below the abdomen, enclosed by a framework of four bones.

PENETRANCE. In genetics, the frequency with which a specific gene produces its effects in a group of people or other organisms. Penetrance is expressed as a percentage.

PENIS. The external male sex organ.

PERCENTILE RANK. The point at which a given percentage of people fall at or below the individual's test score being calculated. For example, if a person's test score was at the 60th percentile, 40% of other test takers received a higher score, while 60% received a score that was at or below that of the test taker.

PERFORMANCE ANXIETY. A subcategory of circumscribed social phobia in which the patient's fear is limited to performing certain activities or tasks in public. Common areas of performance anxiety include public speaking, acting on stage, solo singing, and playing instrumental solos.

PERINEAL. An anatomical area located between the external genitals and the anus.

PERIPHERAL NERVE. A nerve in a distant location from the brain that receives information in the form of an impulse from the brain and spinal cord.

PERSECUTORY DELUSIONS. Unrealistic conviction of being harassed, tormented, and persecuted.

PERSON-CENTERED PLANNING. A technique in which a plan for a person's future is developed by a team consisting of the person, family members, service providers and friends (natural supports). The team develops a practical plan based on the person's wishes and dreams. Each team member agrees to perform certain tasks identified in the plan to help the person reach goals.

PERSON-CENTERED THERAPY. A therapeutic approach that believes the client's own drive towards growth and development is the most important factor in healing.

PERSONALITY DISORDER. A personality disorder is a maladaptive pattern of behavior, affect, and/or cognitive style displayed in a broad range of settings. The pattern deviates from the accepted norms of the individual's culture and can occur over a lifetime.

PERSONALITY INVENTORY. A type of psychological test that is designed to assess a client's major personality traits, behavioral patterns, coping styles, and similar characteristics. The Minnesota Multiphasic Personality Inventory is an example of a personality inventory.

PERSONALIZATION. The tendency to refer large-scale events or general patterns of events to the self in inappro-

priate ways. For example, a person who regards the loss of a friend or relative in an accident as punishment for having quarreled with them before the accident is said to be personalizing the event. Personalization increases a person's risk of developing acute stress disorder or post-traumatic stress disorder after a traumatic event.

PERVASIVE DEVELOPMENTAL DISORDERS (PDDS). A category of childhood disorders that includes Asperger's syndrome and Rett's disorder. The PDDs are sometimes referred to collectively as autistic spectrum disorders.

PET. Abbreviation for positron emission tomography, a highly specialized imaging technique using radioactive substances to identify active tumors, as well as nuerological disease progression.

PETECHIAE. Pinpoint-sized hemorrhages in the skin or a mucous membrane. In bulimia, petechiae may appear in the skin around the eyes as a result of increased pressure in the capillaries caused by vomiting.

PHENCYCLIDINE. The full name of the drug commonly called PCP that is often abused to induce hallucinations.

PHENOL. A white crystalline water-soluble substance used chiefly as an antiseptic and disinfectant.

PHENOMENOLOGICAL THERAPY. A therapeutic approach that focuses on the interpretations individuals place on their experiences.

PHENOTHIAZINES. A class of drugs widely used in the treatment of psychosis.

PHENOTYPE. The observable signs, symptoms, and other aspects of the makeup of an organism. The term is also used sometimes to refer to the appearance of an organism resulting from the interaction between its genotype and its environment.

PHENYLKETONURIA. (PKU) An inherited disease in which the body cannot metabolize the amino acid phenylalanine properly. If untreated, phenylketonuria can cause mental retardation.

PHOBIA. Irrational fear of places, things, or situations that leads to avoidance.

PHONICS. A method of teaching reading and spelling based on the phonetic interpretation of ordinary spelling.

PHONOLOGICAL DISORDER. A developmental disorder of childhood in which the child fails to use speech sounds that are appropriate for his or her age level and native language or dialect.

PHOTON. A light particle.

PHOTOTHERAPY. Another name for light therapy in mainstream medical practice.

PHYSICAL DEPENDENCE. A maladaptive behavior that over a three-month period has caused the individual to experience tolerance and withdrawal symptoms.

PHYSIOLOGY. The branch of medicine concerned with biological processes or functions in the human body or any of its parts.

PHYSOSTIGMINE. A short-acting drug that enhances levels of a substance (acetylcholine) between neurons in the brain.

PICK'S DISEASE. A rare type of primary dementia that affects the frontal lobes of the brain. It is characterized by a progressive loss of social skills, language, and memory, leading to personality changes and sometimes loss of moral judgment.

PLACEBO. An inactive substance or preparation used as a control in experiments with human subjects to test the effectiveness of a drug or herbal preparation. Some patients may experience a medicinal response or experience side effects to a placebo simply because they have faith in its powers even though it contains no medicine.

PLAQUE. A sticky cholesterol-containing substance that builds up on the walls of blood vessels, reducing or blocking blood flow.

PLAQUES. Clumps or clusters of beta amyloid fragments, dead or dying nerve cells, and other cellular debris, found in the brains of patients with Alzheimer's disease.

PLAY THERAPY. A type of psychotherapy for young children involving the use of toys and games to build a therapeutic relationship and encourage the child's self-expression.

PNEUMOTHORAX. A condition in which air or gas is present in the chest cavity.

POLARITY THERAPY. A form of energy therapy influenced by Ayurvedic medicine that integrates bodywork with diet, home exercises, and self-awareness techniques. It is sometimes called polarity balancing.

POLYGENIC. A trait or disorder that is determined by a group of genes acting together. Most human characteristics, including height, weight, and general body build, are polygenic. Schizophrenia and late-onset Alzheimer's disease are considered polygenic disorders.

POLYSOMNOGRAM. A machine that is used to diagnose sleep disorders by measuring and recording a variety of body functions related to sleep, including heart rate, eye movements, brain waves, muscle activity,

breathing, changes in blood oxygen concentration, and body position.

PORPHYRIA. A group of disorders that arise from changes in the metabolism of porphyrin, a naturally occurring compound in the body. The disorders are characterized by acute abdominal pain and neurological problems.

PORPHYRIN. Any iron- or magnesium-free pyrrole derivative occurring in many plant or animal tissues.

POSITIVE AFFIRMATION STATEMENTS. Statements repeated to oneself, either aloud or mentally, that reflect attitudes of self-worth.

POSITIVE REINFORCEMENT. A procedure or response that rewards a desired behavior.

POSITIVE SYMPTOMS. Symptoms of schizphrenia that represent excesses or distortions of normal mental functions.

POSITRON. One of the small particles that make up an atom. A positron has the same mass and amount of charge as an electron, but the positron has a positive charge.

POST MORTEM. After death. The definitive diagnosis of Alzheimer's disease can be made only after the patient's death.

POST-TRAUMATIC STRESS DISORDER. A disorder caused by an extremely stressful or traumatic event (such as rape, act of war, or natural disaster) in which the trauma victim is haunted by flashbacks. In the flashbacks, the event is re-experienced in the present. Other symptoms include nightmares and feelings of anxiety.

POSTURAL TREMOR. A continuous quiver that affects body posture and movement.

PRADER-WILLI SYNDROME. A developmental disorder of childhood characterized by mental retardation, poor muscle tone, delayed growth and sexual maturation, and childhood onset of an abnormally large appetite for food.

PRANA. The Sanskrit word for vital energy, roughly equivalent to qi in traditional Chinese medicine.

PRANAYAMA. The breathing exercises that accompany the asanas in hatha yoga.

PRE-MENSTRUAL SYNDROME. A severe change in mood that occurs in women immediately prior to, and during, their menstrual period.

PREECLAMPSIA. A complication of pregnancy characterized by high blood pressure, fluid retention, and protein in the urine. If the patient develops convulsions, the condition is called eclampsia.

PREFERRED PROVIDER ORGANIZATION (PPO). A type of managed care system involving payment contracts with a group or panel of health care providers.

PREMIUM. The cost of enrollment in a health insurance plan. Premiums are usually paid on a monthly basis.

PRESENILE DEMENTIA. An older name for Alzheimer's disease.

PRESSURE ULCERS. Also known as pressure sores or bed sores, these can develop in stroke patients who are unable to move. If not treated properly, they can become infected.

PREVALENCE. Occurence in a population.

PRIAPISM. Persistent abnormal erection of the penis, usually without sexual desire, and accompanied by pain and tenderness.

PRIMARY ENURESIS. Bed-wetting in a child who has not yet developed bladder control.

PRIMARY GAIN. In psychiatry, the principal psychological reason for the development of a patient's symptoms. In conversion disorder, the primary gain from the symptom is the reduction of anxiety and the exclusion of an inner conflict from conscious awareness.

PRIMARY NARCISSISM. Sigmund Freud's term for a normal phase in early childhood development in which the infant has not yet learned to distinguish between itself and its world, and sees other people and things in its environment as extensions of itself.

PRIMARY PERSONALITY. The core personality of a patient with dissociative identity disorder. In women, the primary personality is often timid and passive, and may be diagnosed as depressed.

PRIMARY PULMONARY HYPERTENSION (PPH). A rare but potentially fatal disorder that affects the blood vessels in the lungs.

PRION. A protein particle that lacks nucleic acid.

PROCESS ADDICTION. An addiction to a mood-altering behavior or series of behaviors rather than a substance.

PROCESS-EXPERIENTIAL THERAPIES. A group of therapies based on a person-centered approach that incorporates elements of cognitive and Gestalt therapies.

PRODROMAL. Premonitory; having the character of a warning. The first psychotic episode in schizophrenia is often preceded by a prodromal phase.

PROGRESSIVE RELAXATION. A technique for managing stress in which the person relaxes major muscle groups in a fixed sequence, often beginning with the feet and moving towards the head.

PROJECTION. A psychological process in which a person unconsciously attributes unacceptable feelings to someone else. Narcissists often project their envy onto other people, claiming that the person in question is envious of them.

PROJECTIVE TEST. A psychological test in which the test taker responds to or provides ambiguous, abstract, or unstructured stimuli, often in the form of pictures or drawings. A projective test may assess a person's thinking patterns, observational ability, feelings, and attitudes.

PROLACTIN. A hormone that stimulates milk production and breast development.

PROSTAGLANDINS. A group of unsaturated fatty acids involved in the contraction of smooth muscle, control of inflammation, and many other body processes.

PROSTATE GLAND. The gland at the base of a male's urethra that produces a component of semen.

PROTOCOL. A plan for carrying out a scientific study or a patient's course of treatment.

PSEUDODEMENTIA. A term for a depression with symptoms resembling those of dementia. The term "dementia of depression" is now preferred.

PSEUDOSEIZURE. An attack that resembles an epileptic seizure but is not associated with abnormal electrical discharges in the patient's brain.

PSYCHIATRIC EPIDEMIOLOGY. A field of research for establishing the incidence, distribution or prevalence, and control of mental disorders in a population, including the sum of the factors controlling the presence of mental disorders.

PSYCHIC NUMBING. An inability to respond emotionally with normal intensity to people or situations; this affects positive emotions as well as fear or anger.

PSYCHOACTIVE SUBSTANCE. A drug that produces mood changes and distorted perceptions; a mind-altering drug.

PSYCHOANALYTIC THEORY. A psychological theory proposed by Sigmund Freud involving unconscious conflicts and specific stages of development; central themes include sexuality and male superiority.

PSYCHODRAMA. A specific form of role play that focuses on acting out "scripts" of unresolved issues within the family, or helping family members adopt new approaches and understanding of one another.

PSYCHODYNAMIC. Referring to the motivational forces, unconscious as well as conscious, that form human attitudes and behavior.

PSYCHODYNAMIC GROUPS. Psychotherapy groups that utilize the principles of unconscious needs and motivations developed by Sigmund Freud.

PSYCHODYNAMIC THEORISTS. Therapists who believe that the origins of mental problems lie in a person's internal conflicts and complexes.

PSYCHOEDUCATION. An approach to treatment that combines instruction with various therapeutic techniques.

PSYCHOGENIC. Originating in the mind, or in a mental process or condition. The term "psychogenic" is sometimes used as a synonym for "conversion."

PSYCHOLOGICAL ASSESSMENT. A process of gathering and synthesizing information about a person's psychological makeup and history for a specific purpose, which may be educational, diagnostic, or forensic.

PSYCHOMETRIC. Pertaining to testing and measurement of mental or psychological abilities. Psychometric tests convert an individual's psychological traits and attributes into a numerical estimation or evaluation.

PSYCHOMOTOR. Referring to a response or reaction that involves both the brain and muscular movements.

PSYCHOMOTOR RETARDATION. Slowed mental and physical processes characteristic of a bipolar depressive episode.

PSYCHOMOTOR SEIZURE. A seizure characterized by electrical activity that is characterized by variable degrees of loss of consciousness and often accompanied by bizarre behavior.

PSYCHONEUROTIC. Pertaining to a neurosis or disorder of the brain; emotionally unstable.

PSYCHOPATH. A person who ruthlessly preys on others, using charm, deceit, violence or other methods that allows him or her to get what they want. Another word that is sometimes used for psychopath is sociopath.

PSYCHOPATHY. A psychological syndrome that includes lack of a conscience or sense of guilt, lack of empathy, egocentricity, pathological lying, repeated violations of social norms, disregard of the law, shallow emotions, and a history of victimizing others.

PSYCHOSEXUAL CONFLICTS. In Freudian categories, internal conflicts related to problems at a particular stage

of childhood development. Freud associated each developmental stage with a particular part of the human body, such as the mouth or the phallus.

PSYCHOSIS. (Plural: psychoses) Severe state that is characterized by loss of contact with reality and deterioration in normal social functioning; examples are schizophrenia and paranoia. Psychosis is usually one feature of an overarching disorder, not a disorder in itself. Psychotic symptoms include delusions and hallucinations.

PSYCHOSOCIAL. A term that refers to the emotional and social aspects of psychological disorders.

PSYCHOSOMATIC. Physical disorder originating in, or aggravated by, the psychic or emotional processes of the individual.

PSYCHOTIC. Mental disorder characterized by disturbances of personality and a loss of normal association with reality.

PSYCHOTROPIC. Having an effect on the mind, brain, behavior, perceptions, or emotions. Psychotropic medications are used to treat mental illnesses because they affect a patient's moods and perceptions.

PTOSIS. Drooping of the upper eyelid.

PUNISHER. Anything that causes a decrease of a particular behavior.

PUNITIVE. Concerned with, or directed toward, punishment.

PURGING. Inappropriate actions taken to prevent weight gain, often after bingeing, including self-induced vomiting or the misuse of laxatives, diuretics, enemas, or other medications.

Q

QI. The Chinese term for energy, life force, or vital force.

QIGONG. A traditional form of Chinese energy therapy that includes physical exercises, breathing techniques, postures, and mental discipline. Internal qigong refers to exercises practiced to maintain one's own health and vitality; external qigong refers to the transfer of energy from a qigong master to another person for healing purposes. External qigong is also known as medical qigong.

QUICKENING. A term that refers to the movements or other signs of life of a fetus in the womb.

R

RADIO WAVES. Electromagnetic energy of the frequency range corresponding to that used in radio communications, usually 10,000 cycles per second to 300 billion cycles per second. Radio waves are the same as visible light, x rays, and all other types of electromagnetic radiation, but are of a higher frequency.

RADIOLOGIST. A medical doctor specially trained in radiology (x ray) interpretation and its use in the diagnosis of disease and injury.

RAPID EYE MOVEMENT (REM) SLEEP. A type of sleep during which the person's eyes move back and forth rapidly underneath their closed eyelids. REM sleep is associated with dreaming.

RAPPORT. A relation of empathy and trust between a therapist and patient.

RATIONAL EMOTIVE THERAPY. A form of psychotherapy developed by Albert Ellis and other psychotherapists based on the theory that emotional response is based on the subjective interpretation of events, not on the events themselves.

RAYNAUD'S SYNDROME. A disorder of the circulatory or vascular system characterized by abnormally cold hands and feet because of constricted blood vessels in these areas.

REALITY TESTING. A phrase that refers to a person's ability to distinguish between subjective feelings and objective reality. A person who knows that their body is real even though they may be experiencing it as unreal, for example, is said to have intact reality testing.

REBOUND EFFECT. A physical reaction to stopping a medication characterized by the reappearance of the symptom that the medication was given to suppress. For example, people who stop taking a sedative may experience rebound insomnia.

RECIDIVISM. A tendency to return to a previously treated activity, or repeated relapse into criminal or deviant behavior.

REFERENTIAL. A type of delusion in which the person misinterprets items, minor occurrences, or other people's behavior as referring to them. Misinterpretations of this sort that are not as resistant to reality as a delusion are sometimes called ideas of reference.

REFOCUSING TECHNIQUES. Techniques that direct one's attention away from overwhelming, negative thoughts and emotions by focusing on inner peace and managing one issue at a time.

REGIMEN. A regulated course of treatment for a medical or mental disorder.

REGISTERED DIETITIAN. A person who has met certain education and experience standards and is well-qualified to provide nutrition counseling.

REGURGITATION. The return of partly digested food from the stomach to the mouth. Regurgitation may be either an intentional act or an involuntary physical reaction.

REHABILITATAIVE. To restore; to put back into good condition.

REIKI. A form of energy therapy that originated in Japan. Reiki practitioners hold their hands on or slightly above specific points on the patient's body in order to convey universal life energy to that area for healing.

REINFORCEMENT. Praise or criticism (or, in substance use, physical consequences) that make a behavior more or less likely in the future. Positive reinforcement (like praise or rewards) increase the likelihood of the behavior, and negative reinforcement (such as criticism or withholding of rewards) decrease the likelihood of the behavior.

REINFORCEMENT SCHEDULE. The frequency and amount of reinforcers administered.

REINFORCER. Anything that causes an increase of a particular behavior.

RELAPSE. A person experiences a relapse when he or she re-engages in a behavior that is harmful and that he or she was trying to change or eliminate. Relapse is a common occurrence after treatment for many disorders, including addictions and eating disorders.

RELAXATION RESPONSE. The body's inactivation of stress responses and return of stress hormone levels to normal after a threat has passed.

RELIABILITY. The ability of a test to yield consistent, repeatable results.

REMISSION. In the course of an illness or disorder, a period of time when symptoms are absent.

REPETITIVE STRESS INJURY (RSI). A type of injury to the musculoskeletal and nervous systems associated with occupational strain or overuse of a specific part of the body.

RESPIRATORY DEPRESSION. Significant impairment of the respiratory system.

RESPONSE COST. A behavioral technique that involves removing a stimulus from an individual's environment so that the response that directly precedes the removal is weakened. In a token economy system, response cost is a form of punishment involving loss of tokens due to inappropriate behavior, which consequently results in decreased ability to purchase back-up reinforcers.

RESPONSE-CONTINGENT. An approach to treatment in which rewards or punishments are given in response to a particular behavior to be encouraged or corrected.

RESPONSE-CONTINGENT LEARNING. A principle that posits that the consequences of a behavior determine whether it will increase or decrease in frequency. Behaviors that bring about desired responses tend to increase, while those that either remove the chance to obtain a desirable outcome, or those that cause some unpleasant or painful consequence, tend to decrease.

RETROGRADE AMNESIA. Amnesia for events that occurred before a traumatic injury.

RETROPERITONEAL. The anatomical area between the peritoneum (lining of the abdominal cavity) and the muscular and connective tissues of the abdominal wall.

RHIZOME. The fleshy underground horizontal root of certain plants. Valerian preparations are made from dried rhizomes as well as from roots of the valerian plant.

RISK ASSESSMENT. The process of gathering and interpreting data useful in estimating the probability that an individual will demonstrate violence.

RISK MANAGEMENT PLAN. Using the results of a risk assessment to tailor intervention strategies intended to reduce the probability that an individual will demonstrate violence.

ROLE. The set of customary or expected behavior patterns associated with a particular position or function in society. For example, a person's role as mother is associated with one set of expected behaviors, and her role as a worker with a very different set.

ROLE TRANSITION. Life changes that require an alteration in one's social or occupational status or self-image.

ROLE-PLAYING. A technique used in assertiveness training and other forms of therapy in which participants act out roles relevant to real-life situations in order to change their attitudes and behaviors.

RORSCHACH TEST. Also known as the Rorschach Psychodiagnostic Test. A commonly administered projective measure in which subjects are asked to describe a series of black or colored inkblots. The inkblots allow the patient to project his or her interpretations, which can be used to diagnose particular disorders.

RUMINATE. To chew or rechew regurgitated food.

RUMINATION. A tendency to dwell on certain thoughts, particularly negative ones, repeatedly or obsessively.

RUSH. The initial intensely pleasurable sensation experienced from injecting a narcotic or stimulant drug. The term has also been applied to the feeling of excitement experienced from the behaviors involved in process addictions.

S

SADISM. A mental disorder in which sexual arousal and gratification are obtained by inflicting pain or humiliation on another person.

SCALE. A subset of test items from a multi-item test.

SCAPEGOATING. the emergence of behavioral problems in one family member, usually the identified patient, who is often punished for problems within the entire family.

SCHILDER'S DISEASE. A disturbance of the white matter of the brain that causes blindness, deafness, and mental deterioration

SCHIZOAFFECTIVE DISORDER. A mental disorder that shows a combination of symptoms of mania and schizophrenia.

SCHIZOPHRENIA. A severe mental illness in which a person has difficulty distinguishing what is real from what is not real. It is often characterized by hallucinations, delusions, language and communication disturbances, and withdrawal from people and social activities.

SCHNEIDERIAN SYMPTOMS. Another name for first-rank symptoms of schizophrenia.

SCOLIOSIS. An abnormal lateral (sidewise) curvature of the spine.

SCREENING TEST. A test given as a preliminary tool, that helps to later target a more thorough analysis.

SEASONAL AFFECTIVE DISORDER (SAD). A mood disorder characterized by depression, weight gain, and sleepiness during the winter months. An estimated 4–6% of the population of Canada and the northern United States suffers from SAD.

SECONDARY BEHAVIORS. Negative behavioral, emotional, or cognitive reactions to stuttering.

SECONDARY ENURESIS. Bed-wetting in a child who has established bladder control but has begun to wet the bed again, usually as the result of emotional stress.

SECONDARY GAIN. A term that refers to other benefits that a patient obtains from a conversion symptom. For example, a patient's loss of function in an arm might require other family members to do the patient's share of household chores; or they might give the patient more attention and sympathy than he or she usually receives.

SECTION 504. This section of the Rehabilitation Act of 1973 provides that no person may be discriminated against because of a physical disability.

SEDATION. A state of emotional or physical relaxation. The term is usually used to refer to this condition when it is produced by a medication.

SEDATIVE. A medication that induces relaxation and sleep.

SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRIS). Commonly prescribed drugs for treating depression. SSRIs affect the chemicals that nerves in the brain use to send messages to one another. These chemical messengers (neurotransmitters) are released by one nerve cell and taken up by others. Neurotransmitters not taken up by other nerve cells are taken up by the same cells that released them. This process is termed "reuptake." SSRIs work by inhibiting the reuptake of serotonin, an action which allows more serotonin to be taken up by other nerve cells.

SELF-ACTUALIZATION. The belief that all human beings have an inborn tendency toward growth and self-improvement.

SELF-CONCEPT. Attitudes about oneself.

SELF-EFFICACY. One's belief about how well he or she can perform a given task, regardless of that person's actual ability.

SELF-HELP GROUPS. Groups that fall outside the realm of psychotherapy groups, but that offer help to individuals around a particular problem or concern. These groups typically are not professionally led.

SELF-INSTRUCTIONAL TRAINING. Teaches individuals to become aware of their self-statements, evaluate whether these self-statements are helpful or hindering, and replace maladaptive self-statements with adaptive ones.

SELF-RATED. A term in psychological testing that means that the person taking the test is the one who decides whether a question applies to them and records the answer, as distinct from an examiner's evaluating and recording answers.

SEMANTIC-PRAGMATIC DISORDER. A term that refers to the difficulty that some children with pervasive developmental disorders have with pragmatic language

skills. Pragmatic language skills include knowing the proper tone of voice for a given context, using humor appropriately, making eye contact with a conversation partner, maintaining the appropriate volume of one's voice, etc.

SEMEN. A thick whitish fluid containing sperm, produced by the male reproductive organs.

SEMINAL VESICLES. Sac-like structures bordering the male urethra and serving as storage depots for seminal fluid.

SENSITIVITY TRAINING. Training conducted in T-groups to reduce tensions and racial prejudice among the public.

SENSITIZATION. To make sensitive or susceptible.

SENSORY INTEGRATION THERAPY. A treatment that was originally designed for children with autism. Sensory integration therapy is often performed by occupational or physical therapists; its goal is to help the child with autism process information acquired through the senses (hearing, touch, taste, and smell as well as sight) more effectively.

SEQUELA (PLURAL, SEQUELAE). An abnormal condition resulting from a previous disease or disorder. An episode of depression is a common sequela of acute stress disorder.

SEROTONERGIC. Containing, activating, or otherwise involving serotonin, which is a chemical that aids in the transmission of nerve impulses.

SEROTONIN. A widely distributed neurotransmitter that is found in blood platelets, the lining of the digestive tract, and the brain, and that works in combination with norepinephrine. It causes very powerful contractions of smooth muscle, and is associated with mood, attention, emotions, and sleep. Low levels of serotonin are associated with depression.

SEROTONIN SYNDROME. A condition characterized by at least three of the following symptoms: diarrhea, fever, extreme perspiration, mood or behavior changes, overactive reflexes, fast heart rate, restlessness, shivering or shaking. It is a result of too much serotonin in the body.

SEXUAL VIOLENCE. Actual, attempted or threatened sexual contact with a person who is non-consenting or unable to give consent.

SHAMAN. In certain indigenous tribes or groups, a person who acts as an intermediary between the natural and supernatural worlds. Shamans are regarded as having the power or ability to cure illnesses.

SHAPING. A technique used in teaching social skills by prompting and reinforcing behaviors that come close to the desired behavior.

SHIFT. The transition of control from one alter to another in a person with dissociative identity disorder. Usually shifts occur rapidly, within seconds but in some cases a more gradual change over is observed. Also referred to as a switch.

SHORT-CIRCUITING OF CONTINGENCIES. The proper reinforcer or punisher for a given behavior is not administered.

SIBLING RIVALRY. Competition among brothers and sisters in a nuclear family. It is considered to be an important influence in shaping the personalities of children who grow up in middle-class Western societies but less relevant in traditional African and Asian cultures.

SIMPLE PHOBIA. An older term for specific phobia.

SKILLED NURSING FACILITY. An inpatient facility that provides 24-hour nursing services to individuals in need of extended care.

SLE (SYSTEMIC LUPUS ERYTHEMATOSUS). An autoimmune disease that leads to inflammation and damage to various body tissues and parts, including joints, skin, kidneys, heart, lungs, blood vessels, and brain.

SLEEP APNEA. Short periods in which a person stops breathing during sleep. Breathing re-starts spontaneously; however, this condition can lead a lack of oxygen in the body.

SLEEP PARALYSIS. An abnormal episode of sleep in which the patient cannot move for a few minutes, usually occurring while falling asleep or waking up. Sleep paralysis is often found in patients with narcolepsy.

SLEEP TERROR DISORDER. A sleep disorder that is distinguished from nightmare disorder by the intensity of associated anxiety symptoms, the absence of complete wakefulness, and the person's difficulty recalling the episode.

SLOW SUICIDE. A term used to refer to lifestyle behaviors known to shorten life expectancy, such as smoking, drinking heavily, having unsafe sex, etc.

SOCIAL COGNITIVE THEORY. The theory that behavior is determined by an interaction between cognitive, behavioral, and environmental factors.

SOCIAL LEARNING. Learning by observing others' responses and acquiring those responses through imitation of the role model(s).

SOCIAL LEARNING THEORY. A subset of learning theories based on the concept that human behavior originates in and is affected by the interplay among the person's learned experiences, previous behaviors, and environmental influences.

SOCIAL MODELING. A process of learning behavioral and emotional response patterns from observing one's parents or other adults. Some researchers think that social modeling plays a part in the development of conversion disorder in children.

SOCIAL PERSPECTIVE-TAKING. A skill that involves a person's capacity to perceive or recognize other people's thoughts and feelings.

SOCIALIZATION. An ongoing process in which a person learns and internalizes the values and behavior patterns of his or her culture and social group.

SOLUTION-FOCUSED THERAPY. A type of therapy that involves concrete goals and an emphasis on future direction rather than past experiences.

SOMATIC. Relating to the body or to the physical.

SOMATIC CONCERN. Excessive concern about the body, particularly in relation to illness.

SOMATIC EDUCATION. A term used in both Hellerwork and the Feldenkrais method to describe the integration of bodywork with self-awareness, intelligence, and imagination.

SOMATIZATION. When mental or emotional distress is expressed physically in a way that disrupts body function.

SOMATIZATION DISORDER. A type of mental disorder in which the patient suffers from physical complaints that serve as coping strategies for emotional distress.

SOMATOFORM. Referring to physical symptoms with a psychological origin.

SOMATOFORM DISORDERS. A group of psychiatric disorders that are characterized by external physical symptoms or complaints.

SPECIFIC PHOBIA. A type of phobia in which the object or situation that arouses fear is clearly identifiable and limited. An older term for specific phobia is simple phobia.

SPECT. Abbreviation for single photon emission computerized tomography, a highly specialized imaging technique using radioactive substances used in research, and to identify neurological disorder/disease progression.

SPEECH-LANGUAGE PATHOLOGIST. Specialist trained in assessment and diagnosis of communication disorders.

SPIRAL CT. Also referred to as helical CT, this method allows for continuous 360-degree x-ray image capture.

SPLITTING. A psychological process that occurs during the childhood of a person with NPD, in which the child separates aspects of him- or herself that the parents value from those that they disregard.

SPONTANEOUS REMISSION. Recovery from a disease or disorder that cannot be attributed to medical or psychiatric treatments.

STALKING. The intentional pursuit or surveillance of another person, usually with the intent of forcing that person into a dating or marriage relationship. Stalking is now punishable as a crime in all 50 states.

STANDARD DEVIATION. A measure of variability in a set of scores. The standard deviations are based on a comparison to others in the same age group.

STANDARDIZATION. The administration of a test to a sample group of people for the purpose of establishing test norms.

STANDARDIZED TEST. A test that follows a regimented structure, and each individual's scores may be compared with those of groups of people.

STIGMA. A mark or characteristic trait of a disease or defect; by extension, a cause for reproach or a stain on one's reputation. Such sexually transmitted diseases as HIV infection carry a severe social stigma.

STIMULUS. Something that incites or moves a person to thought, emotion, or action. In mainstream psychotherapy, a stimulus can be anything from a certain picture or image to a smell, a sound, or a word or idea. In aversion therapy, the stimulus is typically a mild electric shock or a medication that produces unpleasant results.

STIMULUS FADING. A form of behavior modification used to treat children with selective mutism, in which goals of gradually increasing difficulty are set for the child.

STOOLS. Feces, bowel movements.

STREPTOCOCCUS (PLURAL, STREPTOCOCCI). A type of bacterium that is spherical in shape and occurs in chains or pairs. Some diseases that are caused by streptococci appear to be related to obsessive-compulsive disorder.

STRESS. A physical and psychological response that results from being exposed to a demand or pressure.

STRESS MANAGEMENT. A set of techniques and programs intended to help people cope more effectively with stress in their lives by analyzing the specific stressors and taking positive actions to minimize their effects. Most stress management programs deal with job stress and workplace issues.

STRESSOR. A stimulus or event that provokes a stress response in an organism. Stressors can be categorized as acute or chronic, and as external or internal to the organism.

STRIATUM. A part of the basal ganglia, a deep structure in the cerebral hemisphere of the brain. Abnormally high levels of dopamine in the striatum are thought to be related to the delusions and hallucinations of schizophrenia.

STRUCTURAL INTEGRATION. The term used to describe the method and philosophy of life associated with Rolfing. Its fundamental concept is the vertical line.

STUPOR. A trance-like state that causes a person to appear numb to their environment.

SUBDURAL HEMATOMA. Active bleeding or a blood clot inside the dura (leathery covering of the brain). This bleeding or clot causes swelling of the brain, and, untreated, the condition can cause death.

SUBJECTIVE. Referring to a person's unique internal thoughts and feelings, as distinct from the objects of those thoughts and feelings in the eternal world.

SUBJECTIVE UNITS OF DISTRESS (SUDS) SCALE. A scale used by patients during exposure treatment to rate their levels of fear and anxiety with numbers from zero to 100.

SUBSTANCE ABUSE DISORDER. Disorder that is characterized by: an individual's need for more of a drug or alcohol than intended, an inability to stop using by choice, and an ongoing difficulty in recovering from the effects of the substance.

SUBSTANTIA NIGRA. Dark-colored matter located in a section of the crus cerebri area of the brain.

SUDDEN SNIFFING DEATH. Death resulting from heart failure caused by heavy use of inhalants in a single lengthy session.

SUICIDE GESTURE. Attempted suicide characterized by a low-lethality method, low level of intent or planning, and little physical damage. Pseudocide is another term for a suicide gesture.

SUPEREGO. According to Freud, the part of the mind that represents traditional parental and societal values. The superego is the source of guilt feelings.

SUPPLEMENTAL SECURITY INCOME. A federal program that provides cash to meet basic needs for food, shelter and clothing for aged, blind, and disabled individuals who have little or no income.

SUPPORT GROUP. A group whose primary purpose is the provision of empathy and emotional support for its members. Support groups are less formal and less goal-directed than group therapy.

SUPPORTIVE THERAPY. An approach to psychotherapy that seeks to encourage the patient or offer emotional support to him or her, as distinct from insight-oriented or educational approaches to treatment.

SURVIVOR'S GUILT. A psychological reaction in trauma survivors that takes the form of guilt feelings for having survived or escaped a trauma without serious injury when others did not.

SYDENHAM'S CHOREA. A serious manifestation of acute rheumatic fever that commonly occurs in children ages seven through 14, peaking at age eight. This disease of the central nervous system is characterized by emotional instability, purposeless movements, and muscular weakness. At its peak in the 1950s it occured in nearly 50% of the acute rheumatic fever cases, but by 2002 had subsided to a degree of less than 10% of the acute cases.

SYNAPTIC CLEFT. An area between nerve cells that can contain neurotransmitters.

SYNCOPE. A brief lapse of consciousness caused by a temporarily insufficient flow of blood to the brain.

SYNDROME. A group of symptoms that together characterize a disease or disorder.

SYSTOLIC. Referring to the rhythmic contraction of the heart (systole), when the blood in the chambers of the heart is forced out. Systolic blood pressure is blood pressure measured during this phase.

T

T-GROUPS. Short for "basic skills training groups" that are focused on education and discussion regarding social issues, personal problems experienced outside the group setting, and problems from one's past.

TACHYCARDIA. A pulse rate above 100 beats per minute.

TACTILE/TACTUAL. Perceptible by touch.

TANNIN. An astringent compound found in chamomile, oak bark, and certain other plants. Tannin in large quantities can interfere with iron absorption.

TARDIVE DYSKINESIA. A condition that involves involuntary movements of the tongue, jaw, mouth or face or other groups of skeletal muscles that usually occurs late in antipsychotic therapy or even after the therapy is discontinued. It may be irreversible.

TARGET BEHAVIOR. The specific behavior to be increased or decreased during treatment.

TAU PROTEIN. A protein that is involved in maintaining the internal structure of nerve cells. The tau protein is damaged in Alzheimer's disease and ends up forming the neurofibrillary tangles.

TEMPERAMENT. A person's natural disposition or inborn combination of mental and emotional traits.

TEMPORAL LOBE. Large lobe of each side of the brain that contains a sensory area associated with hearing.

TEMPOROMANDIBULAR JOINT DISORDER (TMJ). Inflammation, irritation, pain, limited range of motion, and clicking sounds in the jaw caused by improper opening and closing of the joint.

TEMPOROMANDIBULAR JOINT DYSFUNCTION. Condition resulting in pain in the head, face, and jaw. Muscle tension or abnormalities of the bones in the area of the hinged joint (the temporomandibular joint) between the lower jaw and the temporal bone are usually the cause.

TERMINATION. The important process of ending a therapy group.

TETRAHYDRACANNABINOL (THC). The active substance in marijuana.

THALAMUS. The middle part of the diencephalon (a part of the human forebrain), responsible for transmitting and integrating information from the senses.

THEMATIC APPERCEPTION TEST (TAT). A projective test using stories and descriptions of pictures to reveal some of the dominant drives, emotions, sentiments, conflicts, and complexes of a personality.

THEMATIC DREAM MATERIAL. Psychoanalysts use the technique of dream interpretation to offer patients insight into their unconscious conflicts. The dreams of patients include themes, notions, or underlying ideas about specific objects, situations, or issues. When patients begin to understand the content or themes of their dreams, they may gain insight into their unconscious motives.

THERAPEUTIC ALLIANCE. The technical term for the cooperative relationship between therapist and patient that is considered essential for successful psychotherapy.

THERAPEUTIC DYAD. A term that refers to the two people involved in a psychotherapeutic relationship, namely the therapist and the person seeking treatment.

THERAPEUTIC LETTER. A letter written to the deceased in order to help the survivors express feelings and thoughts they may not have been able to before the loss.

THERAPEUTIC TOUCH (TT). An American form of energy therapy based on the ancient tradition of the laying-on of hands. TT is thought to work by removing energy blockages or disturbances from the patient's aura.

THERAPEUTIC VALUE. The potential benefit of an object or situation, in terms of its ability to enhance functioning (social, emotional, intellectual, occupational, etc.) in an individual.

THERAPEUTIC WRITING. A treatment technique in which patients are asked to set down in writing an account of the traumatic event and their emotional responses to it.

THERMISTOR. An electrical device whose resistance decreases with rises in temperature.

THIAMIN. A B-vitamin that is essential to normal metabolism and nerve function, and whose absorption is affected by alcoholism.

THORACIC. Refers to the chest area. The thorax runs between the abdomen and neck and is encased in the ribs.

THOUGHT INSERTION/WITHDRAWAL. The notion that an outside force (space aliens, evil people, etc.) can put thoughts or ideas into one's mind or remove them. It is considered one of the first-rank symptoms of schizophrenia.

THROMBOCYTOPENIA. A condition involving abnormally low numbers of platelets (blood-clotting agents) in the blood; usually associated with hemorrhaging (bleeding).

THYROID. A gland in the neck that produces the hormone thyroxin, which is responsible for regulating metabolic activity in the body. Supplemental synthetic thyroid hormone is available as pills taken daily when the thyroid fails to produce enough hormone.

THYROID HORMONE. A complex hormone that regulates metabolic rate of all cells.

THYROTOXICOSIS. A disease characterized by an enlarged thyroid gland and speeded-up body metabolism

caused by excessive thyroid secretion. It is also known as Graves' disease.

TIC. A sudden involuntary behavior that is difficult or impossible for the person to suppress. Tics may be either motor (related to movement) or vocal, and may become more pronounced under stress.

TINCTURE. An alcohol-based herbal extract prepared by soaking parts of the plant in a mixture of alcohol and water. Established ratios and dilutions are followed.

TISSUE PLASMINOGEN ACTIVATOR (TPA). A drug that is sometimes given to patients within three hours of a stroke to dissolve blood clots within the brain; also used to treat heart attack victims.

TOKEN. Any item that can be seen and collected (such as stickers or points in a point tally) that has no value of its own, but is used as an immediate reward for desirable behavior that is later exchanged for back-up reinforcers.

TOLERANCE. Progressive decrease in the effectiveness of a drug with long-term use.

TONIC-CLONIC (GRAND MAL) SEIZURE. This is the most common type of seizure and is categorized into several phases beginning with vague symptoms hours or days before an attack. During the seizure, there is abnormal muscle contraction and relaxation and the individual may lose consciousness.

TOPICAL. A type of medication or preparation intended for use on the skin or external surface of the body.

TORPOR. Sluggishness or inactivity.

TOURETTE SYNDROME. Neurological disorder characterized by multiple involuntary movements and uncontrollable vocalizations called tics that come and go over years, usually beginning in childhood and becoming chronic. Sometimes the tics include inappropriate language.

TOURNIQUET. A rubber tube or length of cloth that is used to compress a blood vessel in order to stop bleeding or to shut off circulation in a part of the body. The tourniquet is wrapped around the arm (or other limb) and tightened by twisting.

TOXICOLOGY SCREEN. A blood or urine test that detects the presence of toxic chemicals, alcohol, or drugs in body fluids.

TOXOCARIASIS. Infection with roundworm larvae, commonly transmitted by the feces of dogs and cats.

TOXOPLASMOSIS. A parasitic infection caused by the intracellular protozoan *Toxoplasmosis gondii*. Humans are most commonly infected by swallowing the oocyte form of the parasite in soil (or kitty litter) contaminated by feces from an infected cat; or by swallowing the cyst form of the parasite in raw or undercooked meat.

TRACE MINERAL. An element essential to nutrition or bodily processes that is found in minute quantities.

TRACHEOSTOMY. A surgical procedure in which an artificial opening is made in the patient's windpipe to relieve airway obstruction.

TRAIT ANXIETY. A type of persistent anxiety found in some patients with generalized anxiety disorder. Trait anxiety is regarded as a feature (trait) of a person's temperament.

TRANQUILIZER. A medication that induces a feeling of calm and relaxation.

TRANSCENDENTAL MEDITATION (TM). A meditation technique based on Hindu practices that involves the repetition of a mantra.

TRANSSEXUAL. A person whose gender identity is opposite his or her biologic sex.

TRANSVESTITE. A person who derives sexual pleasure or gratification from dressing in clothing of the opposite sex.

TRAUMA. A disastrous or life-threatening event that can cause severe emotional distress, including dissociative symptoms and disorders.

TREMOR. Involuntary shaking of the hands and arms.

TREPANATION OR TREPANNING. Surgical removal of a piece of the skull to expose the brain.

TRIANGLING. A process in which two family members diminish the tension between them by drawing in a third member.

TRICHOBEZOAR. A hairball that results from a buildup of swallowed hairs becoming lodged in the digestive system.

TRICHOPHAGIA. Eating hair.

TRICHOPHAGY. Biting hair.

TRICHOTILLOMANIA. A disorder marked by repeated pulling and tugging of one's hair, usually resulting in noticeable hair loss on the scalp or elsewhere on the body.

TRICHURIASIS. Infection with the larvae of roundworms. These parasites may live for 10–20 years in humans.

TRICYCLIC ANTIDEPRESSANTS (TCAS). Antidepressant medications that have the common characteristic of a three-ring nucleus in their chemical structure. Imipramine and amitriptyline are examples of tricyclic antidepressants.

TRIGGER. Any situation (people, places, times, events, etc.) that causes one to experience a negative emotional reaction, which is often accompanied by a display of symptoms or problematic behavior.

TRIGLYCERIDES. Fats in the blood.

TRISOMY. An abnormality in chromosomal development. Chromosomes are the structures within a cell that carry its genetic information. They are organized in pairs. Humans have 23 pairs of chromosomes. In a trisomy syndrome, an extra chromosome is present so that the individual has three of a particular chromosome instead of the normal pair. An extra chromosome 18 (trisomy 18) causes mental retardation.

TRYPTOPHAN. An essential amino acid released from proteins during the process of digestion. Tryptophan is an important ingredient in the body's production of serotonin.

TSUBO. In shiatsu, a center of high energy located along one of the body's meridians. Stimulation of the tsubos during a shiatsu treatment is thought to rebalance the flow of vital energy in the body.

TUBERCULOSIS. An infection caused by the bacteria *Mycobacterium tuberculosis* that usually affects the lungs. Individuals with tuberculosis may have nighttime sweating, fever, weight loss, cough, and may spit up blood and mucus.

TYPE II DIABETES. Resistance to the effects of insulin in the presence of normal or elevated insulin levels, resulting in failure of glucose to enter cells and in a cascade of other abnormal physiologic reactions.

TYRAMINE. Intermediate product between the chemicals tyrosine and epinephrine in the body and a substance normally found in many foods. Found especially in protein-rich foods that have been aged or fermented, pickled, or bacterially contaminated, such as cheese, beer, yeast, wine, and chicken liver.

U

ULTRASONOGRAPHY. A process that uses the reflection of high-frequency sound waves to make an image of structures deep within the body. Ultrasonography is routinely used to detect fetal abnormalities. In stroke victims, a cardiac ultrasound, or echocardiogram, allows the beating heart to be examined.

UNCONDITIONAL POSITIVE REGARD. A quality of the client-centered therapist, characterized by the therapist's acceptance of the client without judgment.

UNIVERSALITY. The feeling of being isolated, unique, and separate from others, often experienced by therapy group members.

URETHRA. The tubular passage conducting urine from the bladder to the exterior. In the male, the urethra traverses the penis.

URETHRITIS. Inflammation of the urethra, which is the duct that carries urine and (in males) semen to the outside of the body.

URINARY INCONTINENCE. A term that is sometimes used for enuresis in adults. Urinary incontinence is often found in patients with late-stage Alzheimer's disease or other adult-onset dementias.

URINARY RETENTION. Excessive storage of urine in the body.

URINARY SYSTEM. The kidney, urethra, bladder, and associated organs that process urine and eliminate it from the body.

UTERUS. The hollow muscular sac in which a fetus develops; sometimes called the womb.

UTILIZATION REVIEW. A process used by managed care organizations involving scrutiny of service care delivery to determine whether services are necessary.

V

VAGINA. The part of the female reproductive system that opens to the exterior of the body and into which the penis is inserted during sexual intercourse.

VAGINISMUS. An involuntary tightening of the vaginal muscles that makes sexual intercourse painful, difficult, or impossible.

VALERENIC ACID. The primary medicinal component in valerian preparations.

VALIDITY. The ability of a test to measure accurately what it claims to measure.

VASCULAR. Pertaining to the bloodstream (arteries, veins, and blood vessels).

VETERAN'S ADMINISTRATION HOSPITALS. Medical facilities operated by the federal government explicitly for veterans of the United States military.

VICARIOUS. Acquired through imagined participation in the experience of others. Modeling is a form of vicarious learning.

VIRTUAL REALITY. A realistic simulation of an environment, produced by a computer system using interactive hardware and software.

VOLATILE SOLVENT. A solvent (substance that will dissolve another substance) that evaporates at room temperature.

VOYEUR. A person who engages in the behavior of voyeurism.

VOYEURISM. A paraphilia that involves watching unsuspecting people, usually strangers, undress or engage in sexual activity.

VULVAR VESTIBULITIS SYNDROME (VVS). Vulvar vestibulitis syndrome is thought to be the most frequent cause of dyspareunia in premenopausal women. A chronic, persistent clinical syndrome, vulvar vestibulitis is characterized by severe pain on vestibular touch or attempted vaginal entry.

W

WAXY FLEXIBILITY. A condition in which a person can be molded into a strange position and hold that position for a long period of time.

WERNICKE'S SYNDROME. A group of symptoms that appears in some people who are dependent on alcohol. Due to low levels of thiamin, the syndrome results in disordered eye movements, very poor balance and difficulty walking.

WERNICKE-KORSAKOFF SYNDROME. Group of symptoms that appears in people who are dependent on alcohol. The syndrome is due to a thiamin deficiency, and severely affects one's memory, preventing new learning from taking place.

WITHDRAWAL. Symptoms experienced by a person who has become physically dependent on a drug, experienced when the drug use is discontinued.

WRAPAROUND. A relatively new form of mental health service delivery that strives to accommodate all family members based on self-defined needs, flexibly incorporating both formal and informal community services.

X

XANTHINE. A class of crystalline nitrogenous compounds that includes caffeine.

Y

YIN AND YANG. In traditional Chinese medicine and philosophy, a pair of opposing forces whose harmonious balance in the body is necessary to good health.

YOGA. A system of exercises for achieving bodily or mental control and well-being.

YOGI (FEMININE, YOGINI). A person who is a respected expert in or teacher of yoga.

GENERAL INDEX

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