ELECTRIC SHOCKS

Injury from electric shock is truly an interdisciplinary topic. It involves field theory, electrochemistry, cell biology, and organismal physiology, as well as electrical engineering, human factors, and other disciplines. As the technological sophistication of generation and distribution of electrical energy has grown, so has the general concern about the effects of electric fields on human health.

Historically, death from contact with man-made power-frequency electric shock emerged as a significant public health concern just before the beginning of this century (1). The development of the electric lamp and the explosive growth of the use of electric power, which occurred between 1880 and 1900, corresponded with the rise in incidence of electrical injury and the rise in medical science interest. In the same era, harmful effects of another man-made source of electric energy, ionizing irradiation, was recognized.

Today, people of industrialized nations are bathed in natural and man-made electromagnetic energy which spans a frequency bandwidth from zero (i.e., batteries) to more than 10^{15} cycles/s (ionizing radiation) which can be separated into bioeffect ranges (Table 1). This reality has triggered more research efforts to understand field interactions with biological systems. If exposure to high-energy fields occurs, harm can result. It is clear that man-made electric power is capable of dreadfully destructive bioeffects. Electric shock can be fatal. Survivors of electric shock injury may experience serious long-term effects from their trauma. Reported neuropsychiatric sequelae can vary from vague complaints, seemingly unrelated to the injury event because of the time elapse, or apparent severity, to sequelae consistent with traumatic brain injury.

Data from France suggest that the experience of electric shock is cross-cultural. Cabanes (2) reported 10 years of data for 120,000 electric utility workers. They noted an accident rate of about 125 events/year, with 1231 events over the 10-year period of their study (2). Fatalities were noted in 2.4% of cases. In 77% of their cases, the French authors noted the occurrence of an electric arc event. In reviewing the experience of survivors, 21% of workers were noted to have permanent disability.

Electric shock injuries are typically classified as low or high voltage, although engineering analysis suggests that a description of electric field strength, type of tissue injured, anatomic location of injury, and percentage of body area involved is a better approach to medical classification of the injury consequences of electric shock. Electric field strength refers to the spatial gradient of voltage (i.e., volts per unit length.) As an example of the use of this terminology, when an average-height field electrician is in contact with a 20,000 V power line, the magnitude of the electric field is expected to be very nonuniform, but roughly on the order of 10,000 V/m. Knowledge of the total voltage is useful in understanding the circumstances of the injury; however, the local estimated electric field strength is more helpful for understanding the potential tissue destruction.

EXPOSURE TO ELECTRIC SHOCK

Among electrical utility workers in the United States, the majority of shock victims experience hand-to-hand or hand-to-foot contacts between 6000 V and 10,000 V. Electric shock simulations by computer suggest that with perfect electrical contacts such circumstances can produce electric field strengths in upper-extremity tissues ranging between 60 V/cm and 160 V/cm (3). Fields of this magnitude can produce skeletal muscle and peripheral nerve membrane damage through mechanisms including electroporation (4), Joule heating (4,5), electroconformational protein change (6), or a combination of these mechanisms.

The relative contributions of these different mechanisms to electric shock injury depend in part on the duration of current flow. If the contact time is brief, cell damage due to nonthermal electric breakdown mechanisms will be most important; but when the contact time is longer, heat damage predominates (6).

Table 1. Important Frequency Ranges of Electrical Injury

Frequency	Regime	Applications	Harmful Effects
Direct current to 10 kHz	Low frequency	Commercial electrical power; soft-tissue healing; transcutaneous electrical stimulation	Joule heating; destructive cell membrane potentials
100 kHz to 100 MHz	Radio frequency	Diathermy; electrocautery	Joule heating; dielectric heating of proteins
100 MHz to 100 GHz	Microwave	Microwave ovens	Dielectric heating of water
10^{13}–10^{14} Hz	Infrared	Heating; CO_2 lasers	Dielectric heating of water
10^{14}–10^{15} Hz	Visible light	Optical lasers	Retinal injury; photochemical reactions
10^{15} Hz and higher	Ionizing	Radiotherapy, X-ray imaging, ultraviolet therapy	Generation of free radicals

Note: 1 hertz (Hz) = 1 cycle/s, 1 kilohertz (kHz) = 1000 cycles/s, 1 megahertz (MHz) = 10^6 cycles/s, 1 gigahertz (GHz) = 10^9 cycles/s.

During electric shock, the victim forms a link in a closed electric circuit. The electric current distribution across the tissues in the body depends on the electric conductivity of the various tissues and the electric field intensity variation. In electric shock injury, by Faraday's law, once the current travels away from the contact points to the subcutaneous tissues, it can be expected to distribute like a Laplacian process (7). The current distributes across the extremity tissues so that the electric field strength is nearly constant in any cross-sectional plane perpendicular to the axis of the extremity. Consequently, at a distance from the contact points, the tissues with the least resistance (i.e., muscle, nerve and blood vessels) will carry the largest current density (3,6).

In addition, since the electric field strength is the product of the tissue conductivity and the current density (Ohm's law in continuous systems), other factors such as variation in the area fraction of different tissues along the current path also influence the field strength (7). For example, when current flows through the lower extremity, the electric field strength will be higher in the region of the knee and ankle than in the midcalf or midthigh because the current density is highest near the joints and because the area fraction of bone and skin to muscle is highest, causing the average conductivity over the cross section to decrease.

Since both Joule heating and electric breakdown of cell membranes depend on tissue electric field strength, the extent of tissue injury predictably varies along the current path (4). Particularly when very high voltages are involved, current will be initiated through the victim *before* actual physical contact with the conductor is made. Dielectric breakdown or "arcing" is the mechanism. The breakdown strength of air is approximately 2×10^6 V/m. However, no matter how small the air gap, dielectric breakdown generally will not occur unless there is at least a 300 V potential difference across the gap. The exact arc initiation voltage depends on the temperature and the geometry of the two charged surfaces. After an arc is initiated, on average 2×10^3 V/m is required to maintain the arc (6).

Injury and Death

There are over 540,000 electrical workers in the United States. In 1992, data from the US National Institute of Occupational Safety and Health (NIOSH) on the age-adjusted proportionate mortality ratios for construction electricians showed elevated proportionate mortality from traumatic injury due to electrocution (PMR = 1145) (8). In 1994, US data showed reported days away from work due to illness and injury were recorded for 1915 employee cases of electrical burns, 4190 employee cases of electrocution/electric shock injuries (9), and 5048 employee cases of fires and explosions (10). Often explosions have an electrical ignition source. While electrical injury, fires, and explosions are not frequent events in the workplace (for example, in comparison to contact dermatitis, musculoskeletal injuries, or eye injuries), their occurrence is commonly accompanied by fatal or near-fatal consequences requiring extensive surgical and medical evaluation, treatment, and rehabilitation. In over 90% of the cases of electric shock, the affected workers typically have 4 years to 8 years of job experience. This reflects the demographics of industrial employment. Generally, electric shock events occur while the victims are doing a task (Fig. 1). Almost all of these injuries affect at least one upper extremity.

Arc

As noted above, mechanical contact with a power source is not necessary for direct current or low-frequency alternating current to pass. Electrical contact can be mediated by arcing. Arc contact, or "dielectric breakdown," happens when the electric field strength in a conducting material reaches such a great magnitude that the atoms of the material are pulled apart, creating a hot ionized gas which is a very good conductor of electricity. In effect, an arc can form a direct electrical connection and allow current to pass along its length without mechanical contact or touching by the victim.

Electric shock victims are generally categorized as "true" electrical injuries if there are focal contact wounds providing evidence of a current path into the body. They may be referred to as "flash" victims if there are skin burns on exposed surfaces and no focal contact wounds. Again, it must be kept in mind that the electric arc plasma which burns the skin of "flash" survivors is an excellent electrical conductor. Victims in the "flash" group can experience electrical effects just as victims of lightning contacts can. Even though temperatures for arcs range between 7000°C and 22,000°C, a brief (<10 ms) arc exposure of the body transmits only enough heat energy to cause a partial-thickness skin burn. This result occurs because water (a main constituent of the body) has a density

Figure 1. Staged electric shock incident. Physical contact by a screwdriver held in the mannequin worker hand (top) resulted in an electric shock and arc blast event with a characteristic flash of ionized highly conducting arc plasma.

1000 times greater than that of air, the medium through which the arc is conducting.

Lightning

Apart from current generated by domestic energy production, the natural phenomenon of lighting creates an environmental exposure risk for electrical injury. In the United States there are about 20 million individual flashes hitting the ground each year (11), with lightning-related injuries affecting about 2000 annually. About 200 to 300 people in the United States die each year from lightning. In the United States, the National Lightning Data Network run by Global Atmospherics in Tucson, Arizona monitors lightning activity. The return stroke of a lightning sequence can carry currents ranging from a few kiloamperes to around 300 kA and be driven by a voltage potential of hundreds of millions of volts traveling at a speed close to half the speed of light (11).

BIOLOGICAL EFFECTS OF ELECTRIC SHOCK

Manifestations of Exposure

Heart and Lung. When low-frequency electric current passes through the chest, both cardiac and respiratory functions can be arrested. Respiratory muscle spasm in response to transthoracic currents are well documented (1,12). As little as 20 mA will produce respiratory arrest (12). Atrial or ventricular fibrillation can result from exposure to this nonphysiological electric current if the electric field possesses sufficient magnitude during the repolarization of cardiac cycle. The so-called "vulnerable period" for ventricular rhythm interference is in the stage of (end) ventricular contraction systole which coincides with the most rapid recovery in membrane excitability. The term, vulnerable period, which corresponds to the early T-wave component of the electrocardiogram (EKG), was introduced by Wiggers and Wegria (13). It has been proposed that if the excitable regions of myocardium are stimulated to contract again before other areas have regenerated, then the contraction will propagate through the excitable paths and return through adjacent areas of myocardium that are slower to regenerate. This leads to very abnormal circular conduction paths, ineffective discordant myocardial contraction, and ineffective blood pumping.

On the basis of animal studies, estimates of human cardiac responses have been made. In an accident scenario, if the current is increased to levels of approximately 60 mA hand-to-hand, 50% of victims will experience cardiac rhythm disturbances within 30 s (13). The probability per unit time that fibrillation will occur in a time-varying field is a function of the amplitude of the applied current. For ventricular fibrillation to occur, the magnitude of the electric field must be sufficiently strong during the vulnerable period. The greater the amplitude of the sinusoidal current, the higher the percentage time that the field will be of sufficient amplitude to trigger fibrillation during the vulnerable period. Thus, if hand-to-hand current is increased, the average time before fibrillation is induced decreases in a nonlinear manner. This functionality reaches a plateau beyond which further increases have no effect. While victims of relatively low-voltage electrical shock (e.g., household power) show a higher rate of death by cardiac arrest, this does not imply that strong fields cannot cause the same. However, typically victims of high-voltage shock break contact quickly as a result of the blast force or muscle contractions, so their time of "at-risk" for cardiac arrest is less.

There is very little data regarding the current thresholds for cardiac and pulmonary arrest that exist for different possible paths through the body. If an electric shock passes from one hand to the other, the total current required to induce disturbances in respiratory muscle and cardiac rhythm will be higher than that required to produce excitation of skeletal muscle in the upper extremity. There are two explanations for this phenomenon. The first reason is that in the thorax, the current density, and therefore the electric field strength, will be substantially less than those that exist in the upper extremity. Secondly, although electrically coupled in a synchysis, cardiac muscle cells are substantially smaller in size than skeletal muscle cells in the upper extremity.

The risk of inducing cardiac arrhythmia from electric shock is frequency-dependent. The well-known studies of Dalziel (12,14,15) demonstrated that the heart and skeletal muscle are most susceptible to frequencies in the 50 Hz to 100 Hz range. Both the let-go and fibrillation thresholds are lowest in this range (12,14).

Epidermal Breakdown. When current from an external direct current (dc) source passes through the human body, the epidermal component of the skin contributes 95% to 99% of the resistance to current passage. In most areas, the epidermis is a 100 μm- to 500 μm-thick layer of fused squamous epithelial cells, forming a thin molecular transport barrier. This barrier serves also to limit transport of ions. Depending on its state of hydration, the resistance of one square centimeter of epidermis may range from $5 \times 10^4\ \Omega$ to $5 \times 10^5\ \Omega$. On the palms of the hands and the soles of the feet, the epidermis can build up to double or triple thickness than in other areas, resulting in two to three times greater resistance. With the epidermis intact, the resistance (R) to the dc passage is typically around 100,000 Ω. The resistance of the epidermis is frequency-dependent because alternating current can capacitatively couple across it.

The epidermal dc impedance also depends on degree of hydration, and the voltage drop imposed across it. According to Freiberger, it is reasonable to think of the epidermis with less than 10 dc volts as a highly insulating barrier (16). With greater applied voltage, the epidermis suffers structural breakdown. When sufficiently large transcutaneous potentials are applied, the skin undergoes microscopic structural breakdowns or perforations which allow formation of hydrated electrically conducting channels. The cell layers are torn apart explosively; consequently, portions of the epidermis with better insulating properties are often also destroyed (17). Freiberger (16) has postulated that hair follicles and other skin appendages are the initial sites of electrical breakdown. Below the breakdown voltage, the skin resistance varies only slightly as applied voltages change. Near the skin breakdown voltage, the total resistance drops quickly with increasing voltage (16–19). Recent studies have confirmed these results (16,17). This breakdown voltage is near 150 V in most areas and approximately 400 V on the palms and soles.

Complete epidermal destruction can occur in the epidermis at contact points when the contact voltage is more than 200 V. Since nearly all the voltage drop exists initially across the insulating epidermis, a 200 V contact can produce an intraepidermal field of 10^6 V/m, resulting in instant boiling and charring. As a result, the epidermal layer instantly vaporizes, and subsequently permeabilizes, allowing dc passage to the dermis and deeper tissues. A similar effect is responsible for the skin "kissing burns" frequently seen across joints. As the current passes through the joint, large electric potential (i.e., voltage) drops can build up across the joint. If skin-to-skin contact occurs on either side of a joint, then breakdown level transcutaneous potentials are established.

Electrical Interation in Subcutaneous Tissue. Because various tissues have different electrical properties, the body is a nonhomogeneous electrically conducting material. During current passage through the body the electrical current distribution in the tissues depends on the relative electrical conductivity of various tissues and the frequency of the current. In general, for low-frequency (i.e., below radio frequency) current delivered by contact covering a small surface of the skin, the current density is greatest at the contact points. Once the current travels away from the contact points into subcutaneous tissues, it spreads across the extremity tissues so that the electric field strength is nearly the same within any cross-sectional plane perpendicular to the current path (20,21). Consequently, at a distance from the contact points, the body behaves like a volume conductor with the conductivity of normal saline. Although the tissues with the least resistance (i.e., nerve, muscle, nerve, and blood vessels) carry the highest current density, the difference in conductivity of most tissues is small.

As a basic conceptual framework, it is important to appreciate that the organization of the tissues will influence electric field and current in adjacent tissues. If current passes parallel to the major tissue planes, then the field strength in adjacent tissue will be roughly the same. If current passes perpendicular to the tissue planes, then the current will pass through each tissue in sequence, and the tissues are electrically approximately in series. In series configuration, the current is the same in each tissue but the electric field is not. The electric field will be strongest in the most resistive material if arranged in series (4).

With low-frequency exposure, the microscopic current distribution in subcutaneous tissue is affected by the density, orientation, shape, and size of cells. Because cell membranes are good insulators, electrical current tends to pass between cells. The presence of cells diminishes the area available for ion flux and, as a consequence, makes tissues less conductive than physiologic saline. In effect, resistance to current flow increases with cell density. For connective and other tissue where the volume fraction of tissue occupied by cells is low, this is not an important consideration. In muscle and nerve, the effects of the cells on the electric field distribution is of more importance.

Field distribution is also governed by the geometry and size of the cells. As cell size increases, the membrane has less influence on cellular electrical properties, because the fractional volume of the cell occupied by the membrane decreases (22). For this reason, the conductivity of muscle that is measured parallel to the long axis of the muscle cells is greater than the conductivity of muscle that is measured perpendicular to the major axis. Water content is important too. In cortical bone and epidermis, the resistance is higher because water content is lower.

THE BIOPHYSICS OF ELECTRIC SHOCK

Dosimetry

Table 1 lists a general categorization of electrical fields as relates to frequency and biological effect. Alternating-current electrical power can pass through the body by both direct and indirect electrical transmission. Electrical conduction results from electrical charge interactions—that is, the force experienced by an electrically charged particle in the presence of a spatial gradient in electrical potential (i.e., voltage gradient). The rate of change in voltage across a distance is the electric field strength. The force applied to the charged particle is the charge times the electrical field strength. Field strength will change over time if the amount of charge changes. The rate of change in field strength with time is described by the field frequency. Conventional electrical jargon uses the abbreviation "dc" (i.e., direct current) to indicate a field frequency of zero (i.e., constant voltage gradient) and "ac" (i.e., alternating current) to indicate that the field is changing direction (i.e., alternating polarity) with time.

Radiation is another important mechanism of transmitting electrical power into the body. Electrical energy can be transmitted by radiation when it is very rapidly changing. The radiating field alternates polarity long before the field can reach the affected charge. The time required for one electrical charge to feel the appearance of another charge is the distance between the charges divided by the speed of light.

Electric force is propagated at the speed of light. Or more intuitively stated, light travels at the speed of electrical force. Over distances of meters comparable to the human body, microwaves, light, and X-ray and radioactive radiation can transmit electrical power by radiation.

The effect of electricity on the body depends on the strength and frequency of the electric field, the path of the field, and the histoarchitecture of the tissues. As a result, the story is complex. During mechanical contact with a dc electrical power source, such as a transit system third rail, dc electrical power passes through the body when the circuit between the person and source is closed upon direct electrical contact, which allows charges to move across the interface.

In metals, the mobile charges are weakly held outer atomic shell electrons. In aqueous electrolyte solutions, the mobile charges are the salt ions in solution. Pure water is approximately 10,000 times more resistive to electric current than physiologic saline. The conductivity of physiologic saline is approximately 1 S/m [*Note:* 1 siemens (S) = $1/\Omega$, 1 V = 1 A $\times$ 1 Ω]. By comparison, copper's conductivity is 10^5 S.

Alternating current in the body can be established by alternating magnetic fields. Magnetic forces are more difficult to envision because these are a vectorial force acting between moving charges. The body is readily penetrated by magnetic fields. If the magnetic field changes with time, electrical current will be driven to flow in a circular motion around the magnetic field lines.

According to Ohm's law, electrical current and electric field are inexorably linked. In effect, time-varying magnetic fields act on electrically conductive materials to produce an electric field.

Total Body Resistance

Because the relative volume fraction of various tissue types depends on axial position, so does the current distribution. As a consequence the total resistance depends on axial position. Earlier studies by Freiberger have explained how the high resistance of the joints is due to a large portion of the cross section being occupied by skin and bone. Freiberger has shown that about 25% of the total hand-to-foot resistance is in the wrist, and about 30% is in the ankle (16). Comparison of the resistance of different bodies has shown that people with large joints and strong muscles generally have a lower body resistance than thin, less muscular individuals. Freiberger also has suggested that 60% of the internal body resistance is in the wrist and ankle in hand–foot measurements. This information can be used to make sample models of the internal body resistance. The model shown is valid across the frequency range of dc to microwave. Again, in the radio-frequency (RF) and higher range, there are additional tissue interactions that take place that make the electrical injury picture more complex.

During the 1960s, Kouwenhoven (23) measured limb-to-limb body resistance at various voltages. With intact epidermis, the resistance between any two points on the body surface was observed in excess of 100,000 Ω. This was primarily due to the transport barrier function of the epidermis. Above 200 V the epidermis broke down to levels similar to normal saline. After breakdown, the limb-to-limb body impedance from one hand to one foot was about 1000 Ω, while the internal body impedance from two hands to two feet was about 500 Ω. Additional measurements indicated that the resistance associated with each arm or leg was about 500 Ω, and Kouwenhoven suggested that the resistance associated with the torso was about 100 Ω. These measured resistance values are noted to be consistent with calculated resistance values, when assuming that the body is made up of half-normal saline solution.

Results from computational simulations of the expected tissue field strength within the upper extremity during 60 Hz electrical shock have been reported (3,4). These simulations were based on average adult male body morphology with 1 kV to 20 kV potential drop from one hand to the other. At frequencies above 10,000 Hz, the impedance of the intact epidermis drops to half-normal saline levels. This is because in this frequency region, it is possible to pass significant current through an unaltered epidermis. For example, for hyperthermia therapy RF current can be delivered to subcutaneous tissues without burning the skin.

Central Role of the Cell Membrane

An important feature that electrical force mediated injuries have in common is structural damage to the cell's plasma membrane followed by loss of its ionic barrier function (5). Ionic compartmentalization, as permitted by the cell membrane, is essential for the chemical processes of life (24). The most basic function of the cell membrane is to provide a barrier to restrict ionic transport. The energy required to move a monovalent ion across a pure planar phospholipid bilayer approaches 100 times the thermal energy at room temperature, causing the measured resistance per square centimeter to approach 10^{11} Ω (25). Because cell membranes are typically 30% protein, the energy barrier of the membrane is somewhat less (26). Most (<90%) of the basal metabolic energy expenditure is for driving the membrane pumps which maintain transmembrane ion concentration differences. Nonetheless, structural integrity of the delicate lipid bilayer component is vital for maintaining the transmembrane physiological ionic concentration gradients at a metabolic energy cost that is affordable.

Direct Electric Effects

Recently, it has become well known that passage of low-frequency electrical current through tissue can produce damaging effects due to the direct action of electrical forces on the electrically charged or electrically polarized components of cells (5,18,27,28). In contrast to thermal forces, which are random and, over time, average to zero, electric forces denature macromolecules and macromolecular assemblies by direct vectorial action.

An important distinction between electrical effects and heating effects is that tissue structures with dimensions much larger than that of a macromolecule are influential in electrical effects. For example, the vulnerability of a cell to electrical damage is related to its length in the direction of the field (Fig. 2), whereas its vulnerability to supraphysiologic temperatures is not. If the cell membrane is porated, allowing intracellular current to increase, the induced transmembrane potential elsewhere along the membrane drops. In thermal injury, damage to one component of the cell is not affected by the others, rendering the cell heat-injury-insensitive to cell size and orientation.

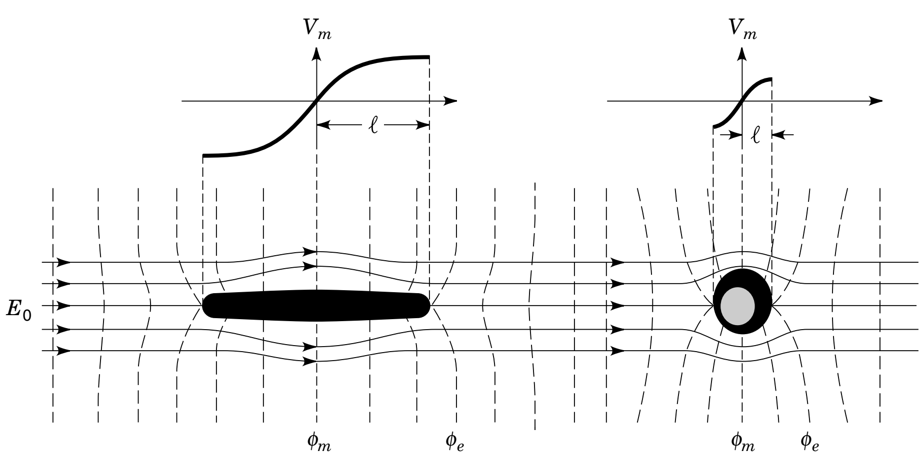

Figure 2. Cell in electrical field. A plot of electrical current lines (solid) and constant voltage (dotted) lines around two different size cells in the same electric field. Note that the larger cell experiences a longer induced transmembrane potential. Because of their relative length, this explains why nerve and muscle cells are at greater risk of electric shock. Electric field lines (E_0) are the same as current lines.

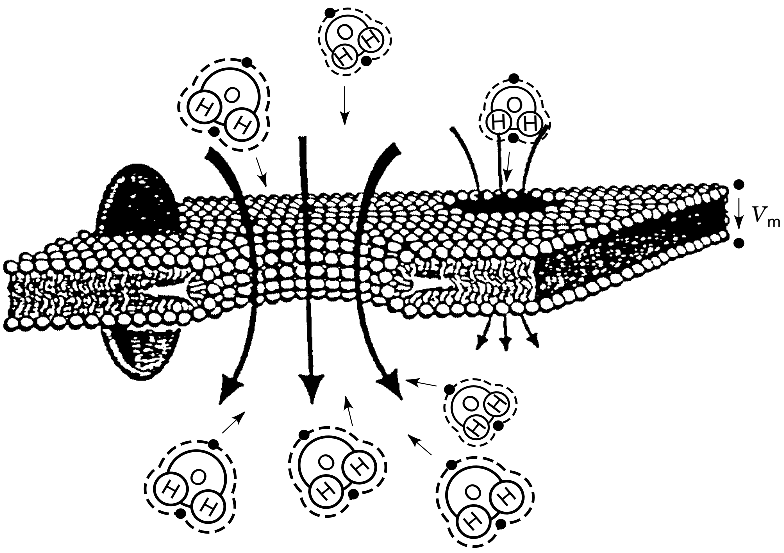

Figure 3. Schematic of cell membrane poration. A schematic illustration of electroporation of a shocked cell.

The pathophysiologic significance of these differences is substantial. First, the cellular injury pattern is different. This is a consequence of the structure of cells, as illustrated in Fig. 2. Since the cytoplasm and extracellular fluids have similar ionic strength, they both are good electrical conductors relative to pure water or oils. The electrical conductivity of the cell membrane, however, is characteristically 1 million times less than the surrounding media. Consequently, electrical current established in the extracellular space is largely shielded from the cytoplasm by the electrically insulating cell membrane. This current shielding limits the voltage drop within the cytoplasm. If there is no current in a conductor, there can be no voltage drop. However, with current passing outside of the cell, there is a voltage gradient along the external surface of the cell changes. This sets up an "induced" transmembrane potential which varies according to position on the cell membrane. This induced transmembrane potential will range from zero at the axis of symmetry to a maximum at the extreme projections of the cell in the direction of the current passage (6,27,29,30). The maximum induced transmembrane potential will scale with the total voltage drop along the outer surface of the cell. Therefore, the magnitude of the induced transmembrane potential depends on the size, geometry, and orientation of the cell with respect to the field.

In most major electrical shocks, the upper extremity is within the current path. Usually the current passes along the extremity axis. Under this circumstance, the long axes of most skeletal muscle cells and nerve axons are oriented approximately parallel to the direction of the field lines. It has been postulated that, given the typical dimensions of human muscle cells, destructive levels of electrical force are imposed on skeletal muscle and nerve cells (27). Two well-described consequences result: (a) disruption of the cell membrane lipid bilayer structure by a process called electroporation and (b) denaturation of membrane proteins by direct vectorial field action on the protein.

Electroporation. Bilayer lipid membranes cannot maintain their structure when the transmembrane potential magnitude is too large. Structural defects or "pores" (31) are formed in the membrane which effectively permeabilize the membrane to ions and molecules as large as DNA (32,33). This electrically driven pore formation process, termed electroporation (Fig. 3), typically occurs with submillisecond kinetics (31–34). The molecular physics responsible for electroporation is still debated, but in general it involves the transport of water into molecular scale pores in the cell membrane until the pore exceeds a critical size (35) (beyond which it is energetically favorable for expansion rather than pore closure). Supraphysiologic transmembrane potentials of greater than 300 mV to 400 mV lead to electroporation. The growth of pores in the bilayer lipid component of mammalian cell membranes are thought to be restricted by membrane proteins, which comprise approximately 30% of the total membrane mass.

When a 1 cm long skeletal muscle cell is placed in a saline conducting medium with a 150 V/cm applied electric field, the membrane is rapidly electroporated, and its electrical conductivity greatly increases. As the conductivity of the membrane increases toward that of the cytoplasm, the membrane electric field decreases and the cytoplasm field strength increases, both approaching that of the externally applied field. The drop in the membrane field strength limits further membrane permeabilization. Although the intracellular field strength reaches that of the extracellular field, the intracellular membranes are not electroporated because of the relatively small size of intracellular organelles. Unlike thermal injury in which all membranes and macromolecules are affected, damage resulting from electrical forces is typically restricted to the plasma membrane.

Protein Electroconformational Change. Thirty percent of mammalian cell membranes is protein. Many types of integral membrane proteins span the entire thickness of the membrane. Generally, these proteins are composed of amino acids with acidic and basic side groups which can be acted upon directly by an intense intramembrane electric field. In addition, amino acids of these proteins are electrical dipoles which can align along the length of the transmembrane protein to create a large electric dipole that can also be acted upon by the field. Typically each amino acid unit contributes an electric dipole moment to the entire protein (36,37). In the

α-helical structure of protein, many small peptide dipoles are aligned almost perfectly to effectively form a larger dipole. In general, a molecule under a strong electric field will tend to shift to a greater dipole moment in the direction of the field. Therefore, membrane proteins will change their conformation in the presence of a strong electric field in a direction to make the effective dipole strength larger. Potassium channels are very sensitive to electroconformational damage. Voltage-gated ion channels are the most likely target for this effect because they are designed to be sensitive to transmembrane voltage differences. The consequences of this effect may underlie the transient nerve and muscle paralysis following shock.

Joule Heating. The passage of current through a resistive material leads to heating. Here "resistive" implies a material that has an electrical conductivity between zero and infinity. The rate of temperature rise depends on the square of the electric field strength and the material's electrical properties. With respect to human tissue, if the temperature exposure from the electric field becomes large enough, then a thermal burn can result. At supraphysiologic temperatures, macromolecules alter optical properties, and grossly visible changes in tissue follow. To understand burns, we must first remind ourselves that temperature, as discovered by Boltzmann, is proportional to the kinetic energy of molecules (38). The relationship is defined by

$$\text{Temperature (K)} \propto \frac{\text{kinetic energy}}{k_B} \tag{1}$$

where T is the absolute temperature (K) of the object. Restated more precisely, the time-average speed, $|s|$, of a monatomic molecule in free solution at temperature, T, is defined by the relationship

$$k_B T \approx mvs^2 \tag{2}$$

where T is the absolute temperature (K) of the object, k_B is Boltzmann's constant, and m is the mass of the molecule with a speed s. Equations (1) and (2) are equivalent statements. As temperature rises, both the molecular momentum transfer between colliding molecules and the frequency of intermolecular collisions escalate. When sufficient momentum is transmitted to folded proteins, bonds maintaining conformation break, and molecular denaturation can take place. As a consequence, at supraphysiologic temperature the probability per unit of time that proteins and other macromolecules denature increases.

The passage of electrical current produces heat because the collisions between the moving charges and other molecules cause a general increase in molecular speed. Joule heating refers to the heat rise from ionic current. Dielectric heating refers to the heat rise from rotating molecular dipoles (e.g., water) in a high-frequency ac electric field. For example, microwave and radio-frequency (e.g., diathermy) heating are forms of dielectric heating. Victims of electrical shock from contact with commercial 60 Hz powerlines experience almost pure Joule heating in the extracellular space. Rotation of the water molecules at 60 Hz adds negligible energy to tissue water. Stated mathematically, the power dissipation density in the form of Joule and dielectric heating can be expressed:

$$\langle \text{Joule heating} \rangle \propto \sigma E^2 \tag{3}$$

$$\langle \text{Dielectric heating} \rangle \propto \omega\epsilon E^2 \tag{4}$$

where σ is the electrical conductivity, and ω is the frequency in cycles/s and ϵ is the volume density of dipoles in the material (i.e., dielectric permittivity). For biological tissue, ϵ is high because of water. Water molecules are small electric dipoles that have a concentration near 55 molar. No other substance is present at such high concentrations. E is the magnitude of the electric field strength. The exact forms of these expressions can be used to calculate the rate of temperature rise in tissues when the electric field or current density distribution is known. If the duration of current passage and the thermal properties of the tissue are also known, the temperature rise itself can be calculated.

The biological significance of Joule heating in electrical trauma can be estimated by first determining the tissue temperature as a function of time. The tissue temperature responses predicted by these simulations can be used to predict tissue damage accumulation based on a chemical reaction rate theory. The kinetics of tissue damage accumulation in response to a given temperature history can be estimated by taking advantage of the fact that the speed of the transition from natural to denatured states is governed by the Arrhenius rate equation (39,40) which states that when the kinetic energy of the molecule exceeds a threshold magnitude E, transition to the denatured state will occur. For a large number of molecules or cells at temperature T the fraction with a kinetic energy above the E is governed by the Maxwell–Boltzmann relation (41):

$$\Gamma(t) = \exp\left(\frac{-E}{k_B T(t)}\right) \tag{5}$$

where k_B is Boltzmann's constant. Since the strength of bonds retaining the folded conformation of macromolecules is very dependent on the nature of the chemical bonds, the value of E is dependent on molecular structure.

Other Harmful Effects of Electric Shock

Surface Heating From Electric Arcs. In addition to electric shock exposure, thermal injury may occur from the surface heating from an electrical arc. Writing in 1986 to 1987, R. H. Lee developed the Drouet–Nadeau empiric relationship into a family of curves relating the distance from the arc center to effective pressure for arc currents ranging from 500 A to 100 kA rms (42,43). This graphical analysis served as a conceptual and practical basis for work practice guidelines regarding safe distances for employees doing tasks with the risk of arc generation.

Blast. Electrical shock is often accompanied by an explosion at the time of an electrical arc. However, blast trauma may not be readily appreciated in survivor triage because of the subsecond time course of these scenarios and the absence of significant external wounds. Barotrauma leading to brain injury, tissue damage at air–fluid boundaries internally (e.g., lungs, ears, bowel), and concussions from explosion shrapnel may not be accompanied by electrical contact sites or burns. For example, a staged electrical arc fault in a 480 V/22,600 A equipment setup (Fig. 1) showed a measured pressure wave of 2160 lb/ft^2 and 141.5 dB sound (29).

Ultraviolet and Infrared Radiation. Intense light, ultraviolet, and infrared radiation can accompany an electric shock when an arc occurs. The exposure to these energies can lead to eye cataracts, corneal burns, and optic nerve injury from weeks to months after the shock. The time course of the ultraviolet and infrared exposure may be subsecond and not widely appreciated by witnesses.

PROGRESS IN PREVENTING ELECTRIC SHOCK

Avoidance of electric shock by the public and workers requires multiple prevention strategies. Hazard awareness, personal protection, and an appreciation of the human factors are individual aspects to electric shock prevention. Engineering in safety in design and specification of equipment and installations as well as administrative controls are preventive strategies that organizations and institutions can adopt.

Electrical safety education presents unique challenges. First, as a hazard, electricity is silent, odorless, and invisible even though the equipment that conducts it may be huge, located in difficult environments, and itself potentially hazardous, especially when an explosion occurs. Second, electricity is commonly experienced as safe: Every time a light switch is thrown in a bathroom or a mouse points a cursor on a video display, electrons flow and injury or damage rarely, if ever, occurs. In other words, there are numerous common experiences in each person's daily life where electricity is essential and yet not noticeable to task completion. For electrical safety educators, the obligation is to raise awareness of electrical risk even though truly no risk may be perceived. After raising risk awareness, the challenge is to modify its acceptability by how individuals and organizations respond to electric shock hazards.

BIBLIOGRAPHY

1. T. Bernstein, Electrical Burns: A Historical Review, in R. C. Lee, E. G. Cravalho, and J. F. Burke (eds.), *Electrical Trauma: The Pathophysiology, Manifestations, and Clinical Management,* Cambridge: Cambridge Univ. Press, 1992, pp. 3–14.
2. J. Cabanes, Industrial Electrical Accidents and Their Complications Observed by Electricite de France, in R. C. Lee, E. G. Cravalho, and J. F. Burke (eds.), *Electrical Trauma: The Pathophysiology, Manifestations, and Clinical Management,* Cambridge: Cambridge Univ. Press, 1992, pp. 15–32.
3. B. J. Tropea and R. C. Lee, Thermal injury kinetics in electrical trauma, *ASME J. Biomed. Eng.,* **114** (2): 241–250, 1992.
4. R. C. Lee and M. S. Kolodney, Electrical injury mechanisms: Dynamics of the thermal response, *Plast. Reconstr. Surg.,* **80**: 663–671, 1987.
5. R. C. Lee et al., Role of cell membrane rupture in the pathogenesis of electrical trauma, *J. Surg. Res.,* **44** (6): 709–719, 1988.
6. R. C. Lee, Tissue injury from exposure to power frequency fields, in J. C. Lin (ed.), *Advances in Electromagnetic Fields in Living Systems,* vol. 1, New York: Plenum, 1994.
7. H. A. Haus and J. R. Melcher, *Electromagnetic Fields and Energy,* Englewood Cliffs, NJ: Prentice-Hall, 1989.
8. C. Robinson, M. Peterson, and S. Talu, NIOSH poster presentation, *2nd World Cong. Electr. Electromag. Biology,* Bologna, 1997.
9. US Bureau of Labor Statistics (BLS) and Occupational Health and Safety Administration (OSHA) data, 1994 [Online]. Available ftp: ftp://stats.bls.gov/pub/special.requests/ocwc/osh/c_d_data, tables R13.
10. Bureau of Labor Statistics (BLS) Occupational Health and Safety Administration (OSHA) data, 1994 [Online]. Available ftp: ftp://stats.bls.gov/pub/special.requests/ocwc/osh/c_d_data, tables R12
11. J-P. Diels et al., Lightning control with lasers, *Sci. Amer.,* **August**: 50–55, 1997.
12. C. F. Dalziel and W. R. Lee, Lethal electric currents, *IEEE Spectrum,* **6** (2): 44–50, 1969.
13. C. J. Wiggers and R. Wegria, Ventricular fibrillation due to single, localized induction and condenser shocks applied during vulnerable phase of ventricular systole, *Am. J. Physiol.,* **128**: 500–505, 1939.
14. C. F. Dalziel, Effects of frequency on let-go currents, *Trans. Amer. Inst. Electr. Eng.,* **62**: 745–750, 1943.
15. C. F. Dalziel, Threshold 60-cycle fibrillating currents. *AIEE Trans. III Power App. Syst.,* **79**: 667–673, 1960.
16. H. Frieberger, *The Electrical Resistance of the Human Body to Commercial Direct and Alternating Currents (Der elektrische widerstand des menschlichen Korpers gegen technischen gleich und wechselstrom),* Z. VDI Bd. 78 D. 1413, Berlin: Springer-Verlag, 1934. Translated from German by Allen Translation Service, Maplewood, NJ, Item No. 9005, 1934.
17. M. R. Prausnitz et al., Electroporation of mammalian skin: A mechanism to enhance transdermal drug delivery, *Proc. Natl. Acad. Sci. USA,* **90**: 10504–10508, 1993.
18. K. T. Powell, A. W. Morgenthaler, and J. C. Weaver, Tissue electroporation: Observation of reversible breakdown in viable frog skin, *Biophysics J.,* **56** (6): 1163–1171, 1989.
19. U. F. Pliquett et al., Imaging of fluorescent molecule and small ion transport through human stratum corneum during high voltage pulsing: Localized transport regions are involved, *Biophys. Chem.,* **58**: 185–205, 1996.
20. A. Sances et al., Experimental electrical injury studies, *J. Trauma,* **21** (8): 589–597, 1981.
21. R. K. Daniel et al., High-voltage electrical injury, *J. Hand. Surg.,* **13A**: 44–49, 1988.
22. D. G. Gaylor, A. Prakah-Asante, and Raphael C. Lee, Significance of cell size and tissue structure in electrical trauma, *J. Theor. Biol.,* **133**: 223, 1988.
23. W. B. Kouwenhoven, Human safety and electric shock, Wilmington: Wilmington ISA Electrical Safety Course, 1968.
24. J. Horgan, In the beginning . . ., *Sci. Amer.,* **264**: 116–125, 1991.
25. A. Parsegian, Energy of an ion crossing a low dielectric membrane: Solutions to four relevant problems, *Nature,* **221** (5183): 844–846, 1969.
26. O. F. Schanne and E. P. R. Ceretti, *Impedance Measurements in Biological Cells,* New York: Wiley, 1978.
27. R. C. Lee and M. S. Kolodney, Electrical injury mechanisms: Electrical breakdown of cell membranes, *Plast. Reconstr. Surg.,* **80**: 672–679, 1987.
28. L. Tung et al., Effects of strong electrical shock on cardiac muscle tissue, *Ann. NY Acad. Sci.,* **720**: 160–175, 1994.
29. R. A. Jones et al., Staged tests increase awareness of arc-flash hazards in electrical equipment, in the *Conf. Proc. IEEE IAS PCIC Annu. Meet.,* Banff, CA, September 1997.
30. J. P. Reilly, Scales of reaction to electric shock: thresholds and biophysical mechanisms, *Ann. NY Acad. Sci.,* **720**: 21–37, 1994.
31. J. D. Lister, Stability of lipid bilayers and red blood cell membranes, *Physics Lett.,* **53** (A): 193–194, 1975.
32. K. T. Powell and J. C. Weaver, Transient electric pore in bilayer membranes: A statistical theory, *Bioelectrochem. Bioenerg.,* **15**: 211–227, 1986.

33. T. Y. Tsong, Electroporation of cell membranes, *Biophys. J.,* **60**: 2977–3306, 1991.

34. U. Zimmerman, Electrical breakdown: electropermeabilization and electrofusion, *Rev. Physiol. Biochem. Pharmacol.,* **105**: 175–256.

35. G. I. Taylor and D. H. Michael, On making holes in a sheet of fluid, *J. Fluid Mech.,* **58**: 625–640, 1973.

36. T. Y. Tsong and R. D. Astumian, Electroconformational coupling and membrane protein function, *Prog. Biophys. Mol. Biol.,* **50**: 1–45, 1987.

37. T. Y. Tsong and R. D. Astumian, Electroconformational coupling: How membrane-bound ATPase transduces energy from dynamic electric fields, *Annu. Rev. Physiol.,* **50**: 273–290, 1988.

38. J. Blackmore, *Ludwig Boltzmann, His Later Life and Philosophy, 1900–1906, Book One: A Documentary History,* Boston: Kluwer, 1995.

39. K. R. Diller, The mechanisms and kinetics of heat injury accumulation, in R. C. Lee, M. Capelli-Schellpfeffer, and K. M. Kelley (eds.), *Electrical Injury: A multidisciplinary approach to therapy, prevention, and rehabilitation,* New York: New York Academy Sciences, 1994, pp. 38–55.

40. E. G. Cravalho et al., Response of cells to supraphysiologic temperatures: Experimental measurements and kinetic models, *Ann. NY Acad. Sci.,* **720**: 281–300, 1994.

41. C. Tanford, *Physical Chemistry of Macromolecules,* New York: Wiley, 1961.

42. R. H. Lee, The shattering effect of lightning—pressure from heating of air by stroke current, *IEEE Trans. Industry Appl.,* **IA-22**: 416–419, 1986.

43. R. H. Lee, Pressures developed by arcs. *IEEE Trans. Ind. Appl.,* **IA-23**: 760–764, 1987.

M. Capelli-Schellpfeffer
R. C. Lee
University of Chicago