Surgical Implantation of Cardiac Rhythm Devices

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This book is dedicated to the electrophysiology and surgical faculty, laboratory and surgical staff, and fellows with whom we have worked and learned from throughout our careers.

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Preface

Electrophysiology has evolved into a procedure-based subspecialty. A wealth of literature exists addressing arrhythmia recognition and treatment, including catheter-based ablations and appropriate cardiac rhythm device choice and programming. Few texts provide an in-depth description of surgical techniques. While physicians desiring to learn implant techniques can refer to standard surgical texts for many common principles, we considered that a surgical technique–focused text for electrophysiologists was needed.

Electrophysiologists historically learned their surgical techniques from surgical colleagues. However, as implantation of pacemakers and implantable cardioverter-defibrillators (ICDs) transitioned from the surgeon to the electrophysiologist, younger physicians have been further removed from formal surgical training. With each successive generation of trainees, it may become more confusing to differentiate between fundamental technique and personal style or bias. Consequently, trainees may or may not have had the opportunity to learn proper surgical technique or to adapt the procedure to various challenges that may be encountered in an individual patient. *Surgical Implantation of Cardiac Rhythm Devices* was written with this in mind.

This text begins with a historical overview of pacemaker and ICD development. This is followed by a comprehensive review of surgical anatomy specific to device implantation, including relevant anatomic structures and landmarks. A review of surgical tools and techniques is provided to familiarize the electrophysiology trainee with the tools of the trade and their proper use, suture and needle choices, and hemostasis. A chapter on cardiac anesthesia is provided as a resource for those unfamiliar with the complexities of periprocedural analgesia and anesthesia specific to the cardiac patient. A chapter reviewing radiation safety is included as a reminder of the risks of radiation exposure to the patient, operator, and support staff, and how to mitigate the risks of exposure. We also include a discussion of proper patient preparation, procedural planning, patient positioning, skin preparation, and draping. This is followed by a detailed description of the surgical procedure, including surgical planning, incision choices, pocket formation, vascular access, lead positioning and fixation, securing of the lead(s) within the pocket, generator positioning, and wound closure. An accompanying chapter details transvenous lead placement, including leads for rescynchronization pacing. Two additional chapters discuss considerations for novel and alternative lead placement, including epicardial leads and the totally intracardiac pacemaker, and implantation of the subcutaneous implantable defibrillator. Challenges in pediatric and young adult patients are discussed, followed by a description of the procedural management of patients with special circumstances and obstacles (e.g., obesity, cachexia, breast implants). A chapter on pitfalls and complications addresses mitigation of the risks and management of the complications commonly encountered in device implantation. One chapter is dedicated to the important topic of prevention, evaluation, and management of implantable device infections. A final chapter reviews the postoperative management of patients undergoing device implantation from the immediate postoperative period through discharge and follow-up.

We offer a comprehensive compendium of the information needed to successfully implant pacemakers and ICDs. Collectively, it is our goal to provide trainees, recent graduates, and experienced implanting physicians the resources and knowledge needed to augment their skills in device implantation.

Surgical Implantation of Cardiac Rhythm Devices represents the culmination of 30 years of experience working with, and learning from, the experiences of 15 fellow cardiac electrophysiologists and 26 cardiothoracic surgeons at the University of Washington. While we have asked many of our faculty at the University of Washington to contribute to this effort, we also sought the expertise of several colleagues throughout the United States. We appreciate their contributions. It is our hope that the readers of this text will find the information complementary to their mentored learning.

Finally, we give special thanks to Edward Verrier, MD, Professor, Department of Surgery, University of Washington, for his support and to the many cardiothoracic surgical faculty and staff who have collaborated with the electrophysiologists at the University of Washington.

Jeanne E. Poole, MD
Lyle W. Larson, PA-C, PhD

1

Development of Cardiac Implantable Electrical Devices

RAKESH GOPINATHANNAIR, BRIAN OLSHANSKY

Introduction

A remarkable collaborative and forward-thinking effort led to the development of cardiac implantable electrical devices (CIEDs), which can stimulate the heart to beat, protect against cardiac arrest, monitor physiologic parameters and arrhythmias, and improve and forestall heart failure. Some key events that led to modern CIEDs and their implantation are described here with a look toward the future.

Where Did It Start?

Myths and science converge in the initial attempts at cardiac stimulation. The rather dubious beginnings are hard to establish precisely, considering the naivety regarding mechanisms and lack of documentation and peer review. Initial observations occurred before electricity was understood fully and before wall power. Nevertheless, cardiac stimulation began hundreds of years ago, if not long before that, first mechanically, then electrically.

William Harvey made an arrested pigeon's heart beat with the flick of a finger.[1–3] Physicist Nickolev Abildgaard realized that electrical shocks could cause a hen to collapse but additional shocks brought it "back to life."[4] By the 1900s, electrical currents were seen to start and stop ventricular fibrillation.[5] Rudimentary electrical resuscitative device attempts for ambulances, however, initially met with little success.

Birth of the Pacemaker

Astounding observations lent credence to the concept that electricity affected the heart (Table 1.1). In one instance, a child who drowned was resuscitated by attaching one electrode to the leg while rhythmically tapping the heart with another (Duchenne de Boulogne, 1806–1875). Why this was attempted is not clear. In another instance, electrical cardiac stimulation was seen to affect heart rate directly in a woman who had a chest tumor removed, leaving her heart exposed (H. von Ziemssen, 1829–1902). Unfortunately, stimulation attempts may have resulted in her death.

In 1889, John Alexander McWilliam reported brilliant work that showed electrical impulses could cause ventricular contraction (Fig. 1.1). He stated:

> The heart was inhibited by stimulation of the vagus nerve in the neck, and then a periodic series of induction shocks (regulated by a metronome) was applied to the apex of the ventricles.… A series of single induction shocks excites a corresponding series of cardiac beats; the ventricular contraction precedes the auricular contraction when the exciting shocks are applied to the ventricles. Each systole causes the ejection of a considerable amount of blood into the aorta and pulmonary artery, and a marked rise of the blood-pressure at each beat.[6]

Although early experiences were not uniformly successful, by 1927, Marmrostein had found a way to electrically stimulate a dog's heart.[7] Soon thereafter, human cardiac pacing emerged. Mark Lidwell, an Australian physician, is considered one of the fathers of modern pacing. In 1928, with alternating current and a needle inserted into a ventricle, he used intermittent electrical stimulation to resuscitate a child born with a cardiac arrest.[8] To extend these initial observations, in 1932, Albert Hyman, a cardiologist working at the Beth David Hospital in New York, utilized a needle he called a "pace-maker" to create an injury current in the heart ("intracardiac" therapy) to allow it to beat again. In collaboration with his engineer brother, C. Henry Hyman, he developed a spring-wound, hand-cranked stimulating motor he called an "artificial pacemaker."[2] The mechanism was simple, but it worked and was used 43 times with success in 14 patients (Fig. 1.2). Based on the need for increasing heart rates during hypothermic procedures, Wilfred Bigelow, working with John A. Hopps, an electrical engineer, developed a transvenous catheter electrode that was placed in the heart and activated using an external stimulator.[1]

Further development halted for quite some time when, in 1951, Boston cardiologist Paul Zoll developed an external (transcutaneous) pacemaker that could stimulate the heart but required 50 to 150 V of alternating current delivered via external metal electrodes strapped to the chest (Fig. 1.3). It was painful and caused skin burns. The longest period of pacing

2

Surgical Anatomy for the Implanting Physician

CORINNE L. FLIGNER, JOHN I. CLARK, JUDY M. CLARK, LYLE W. LARSON, JEANNE E. POOLE

Introduction

Anatomy, one of the oldest basic medical sciences, is defined by three-dimensional structures, functions, and relationships of organs and their blood supply, innervation, and lymphatics.[1–3] A comprehensive knowledge of human anatomy and the surrounding structures is fundamental to the planning and execution of a successful surgical procedure. In this regard, implantation of cardiac implantable electronic devices (CIEDs) (pacemakers and implantable cardiac defibrillators) is similar to any other surgical procedure. This chapter provides a review of surgically relevant anatomy to the practitioner implanting these devices and highlights those features which are of clinical importance. Static and dynamic relationships of structures are emphasized, as are their appearances on radiographs when appropriate. Illustrated anatomy and cadaveric dissections are used to emphasize the relationship of anatomy to the procedure.

External Anatomic Landmarks

The implanting physician should be familiar with the external landmarks of the human thorax. The skeletal landmarks on the surface of a patient guide the surgeon to the locations of the important musculature and vasculature providing the basic framework for the surgical space. Fig. 2.1 shows the important superficial anatomic landmarks of the anterior chest wall. The skeletal boundaries of the chest include the clavicles, manubrium, sternum, xiphoid process, and rib cage. Critical anatomic associations include the clavicular and sternal attachments of the pectoralis major muscle and the deltopectoral groove, a depression between the lateral edge of the pectoralis major muscle (clavicular head) and the medial border of the anterior deltoid muscle.

The spinal column, scapulae, and ribs compose the primary bony landmarks of the posterior chest wall (Fig. 2.2). The posterior musculature includes the trapezius and latissimus dorsi. The lateral aspect of the external body wall can be separated into vertical regions: the posterior, mid-, and anterior axillary lines. These are easily observed by having a person extend his or her arm and push down on a fixed object. The contraction of the latissimus dorsi and the pectoralis major raises two folds posterior and anterior to the axilla. The posterior axillary line is the raised lateral boundary of the contracting latissimus dorsi, the anterior axillary line is the raised lateral boundary of the contracting pectoralis major, and the midaxillary line is vertically oriented in the center of the hollow axilla (Fig. 2.3).

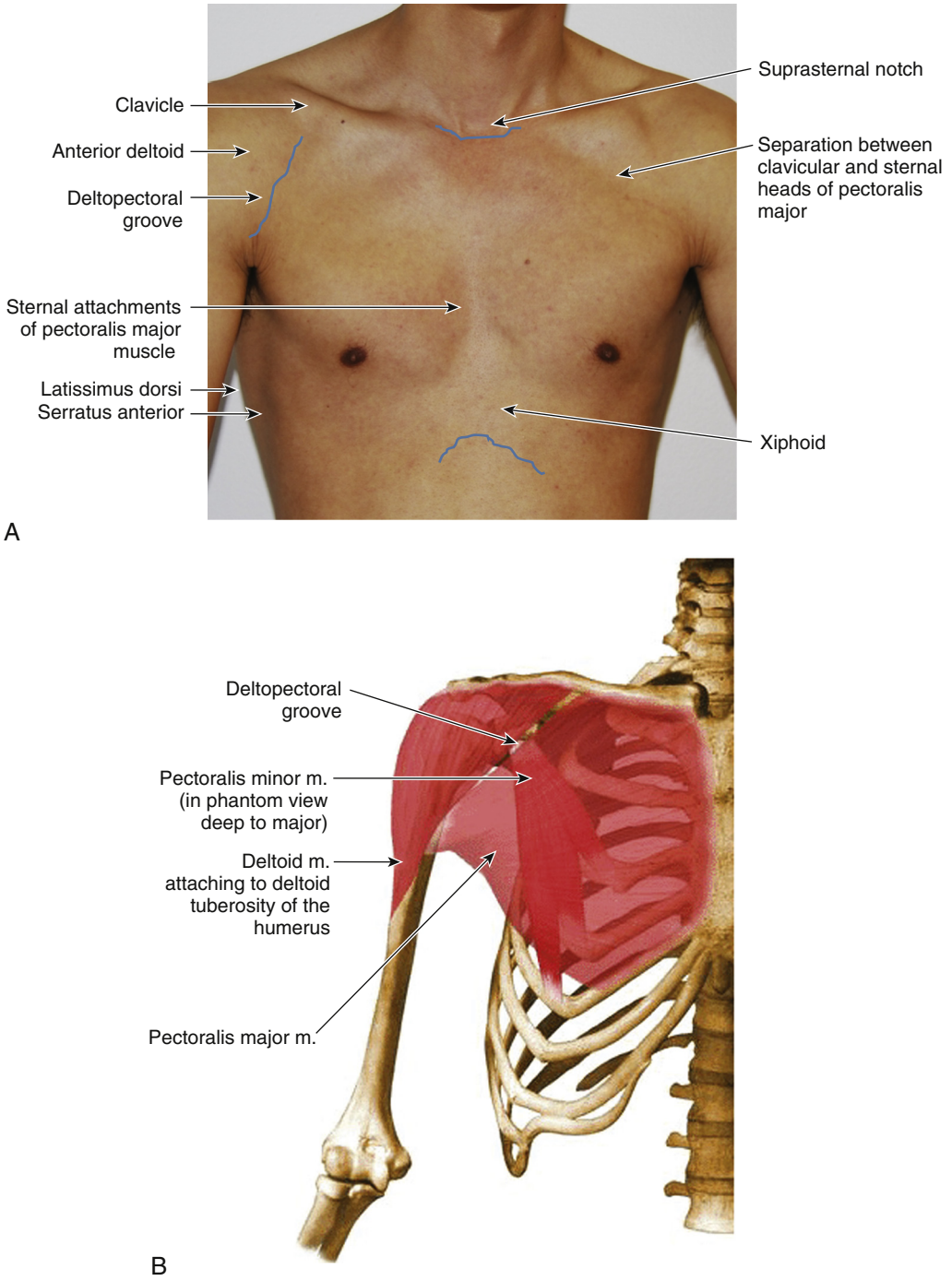
Clinical Correlations

- The chest wall landmarks dictate the boundaries of the surgical sites for device implantation. It is critical to understand the interstructural relationships in both the supine and standing positions.

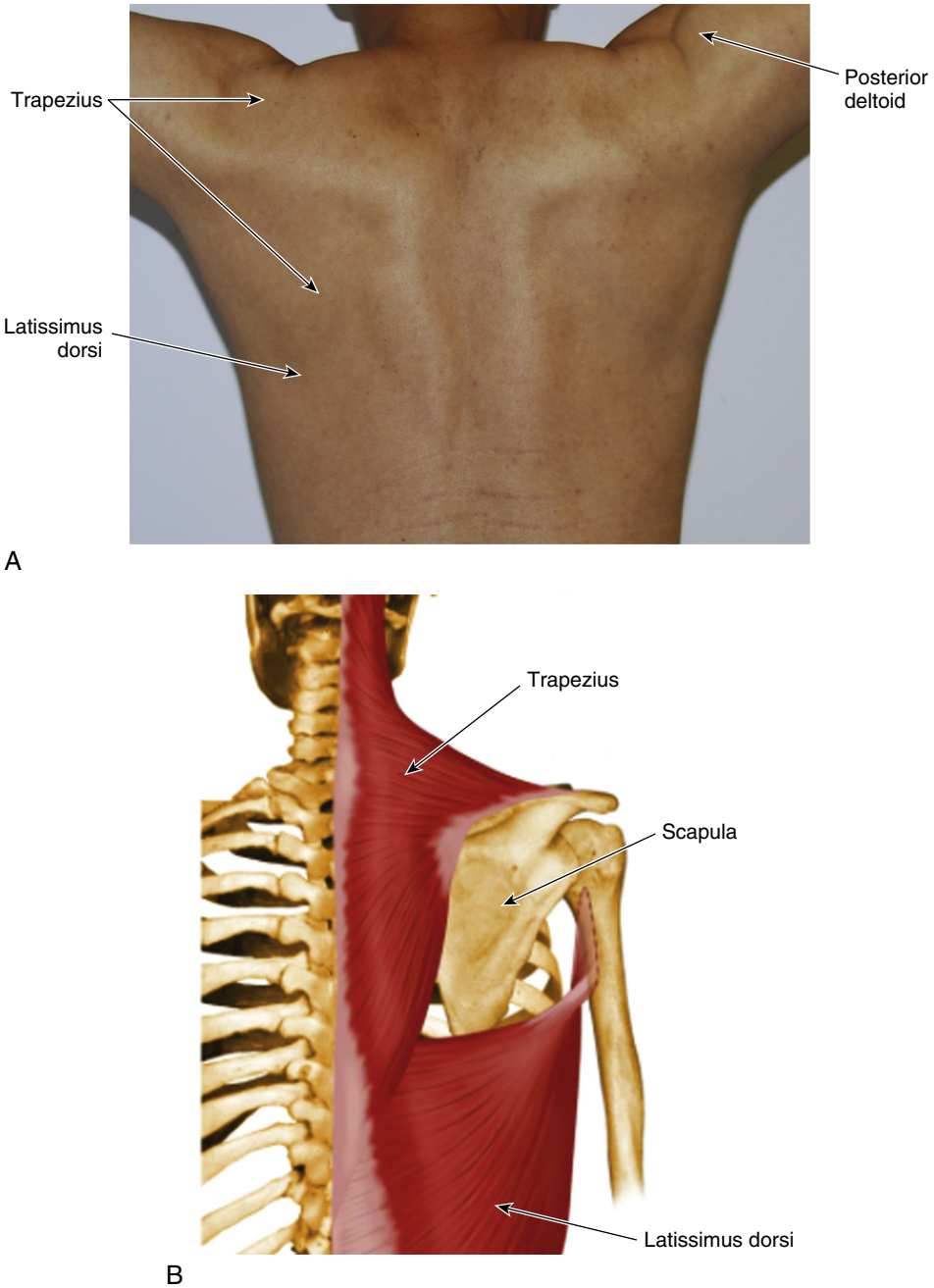
Skin

The surgical procedure starts at the skin, the largest single organ in the body. While seemingly simple, the skin is a complex protective barrier for the body with a rich network of vasculature, lymphatics, and nerves that supply sweat glands, sebaceous glands, hair follicles, and the sequential sensory dermatomes of the body wall (Fig. 2.4). The surface layer of the skin is the epidermis, consisting of the stratum corneum, melanocytes, and Langerhans cells (which participate in the skin's immune response). The underlying dermis is a thick layer of fibrous and elastic tissue (made up of collagen, elastin, and fibrillin) that gives skin its flexibility and strength. The dermis also contains sensory nerve endings and capillaries. Pain, itch, and temperature are sensed by the unmyelinated nerve endings in the papillary dermis. Low stimulation causes itching, whereas high stimulation causes pain. Scratching converts itching to the more tolerable sensation of pain.

The subcutaneous collagen fibers of the dermis course along specific directional lines, referred to as Langer's lines (Fig. 2.5).[4,5] Other "lines" have been identified and include



• **Fig. 2.1** (A) Superficial landmarks of the anterior chest wall used for planning the surgical procedure and identifying the borders of the surgical field. (B) Location of the corresponding muscular structures. (Courtesy University of Washington School of Medicine, Seattle.)

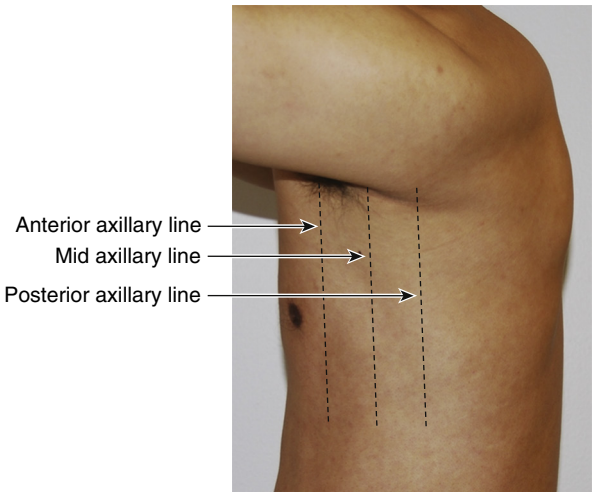


• **Fig. 2.2** (A) Superficial landmarks of the posterior chest wall. (B) Location of the corresponding muscular structures. (Courtesy University of Washington School of Medicine, Seattle.)

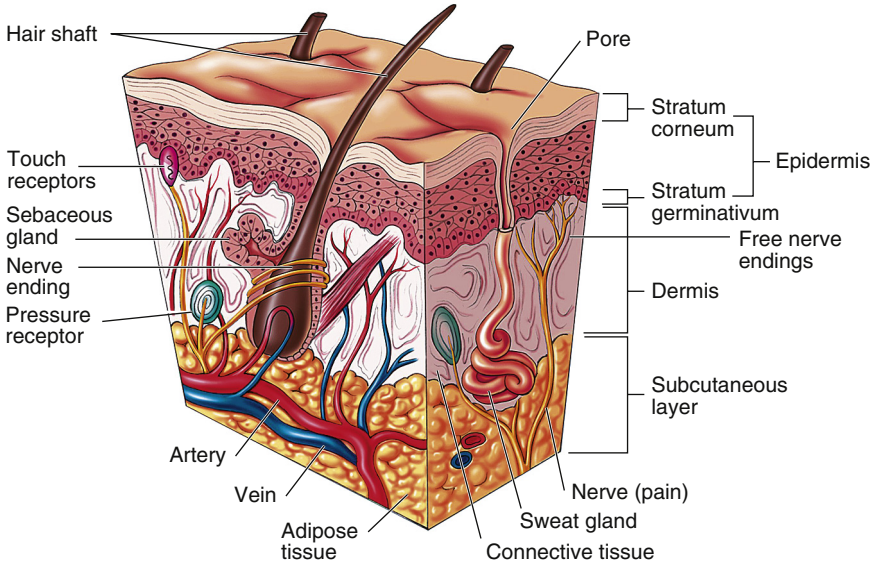
Kraissl lines, which are lines of maximal skin tension oriented perpendicular to the action of underlying muscles, and Borges' lines, which can be identified as relaxed skin tension lines when the skin relaxes after being pinched or after muscular contraction or joint mobilization.[6,7] Below the dermis is the fatty layer of superficial fascia (Camper's fascia) that is supported by a dense membranous layer of fascia, Scarpa's fascia. The subcutaneous fatty layer can be quite thick, even in individuals with normal body mass index. Normally there is a potential space between the membranous layer of superficial fascia and the deep fascia covering the muscles of the body wall. Note that the nomenclature varies, and that it is named the retromammary space in females.

Clinical Correlations

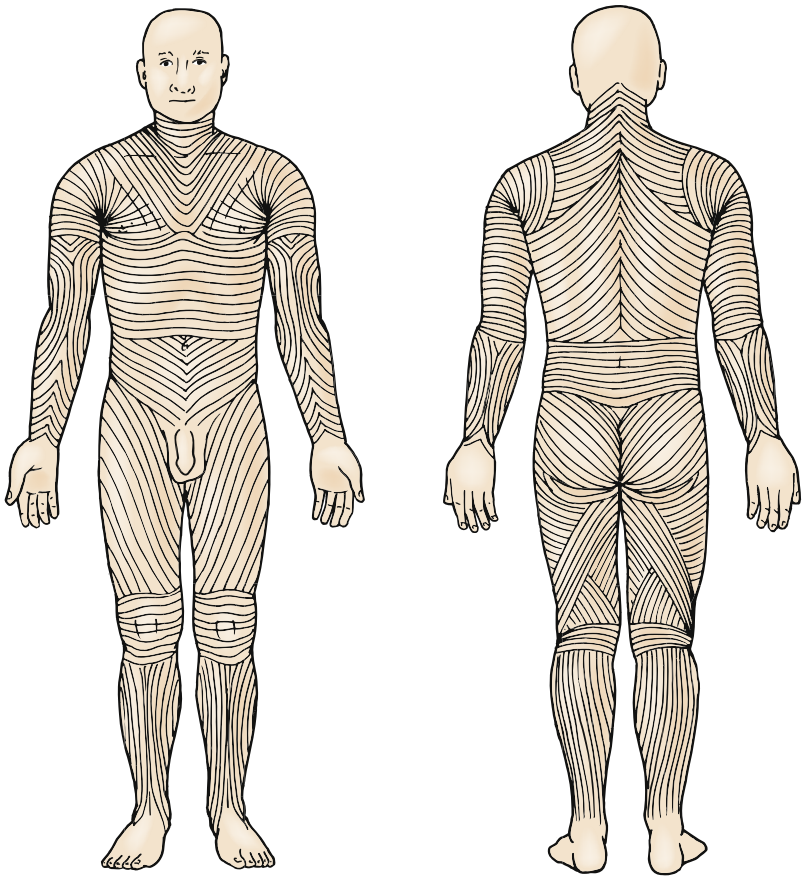
- It is often suggested that skin incisions be oriented in the same direction as Langer's lines, as this results in a thinner, less conspicuous scar, because the healing incision is subjected to less perpendicular force. Practically, however, for the small incisions used in pacemaker and defibrillator placement, an attempt to adhere to Langer's lines is not a major factor in wound healing or wound appearance (see Fig. 2.5).[2]
- Somatic (body wall) nerve endings in the skin are the primary source of pain during the implant procedure, and open skin wounds are a portal for bacteria to penetrate the superficial fascia and enter the muscular layers of the body wall (Fig. 2.6).[7]



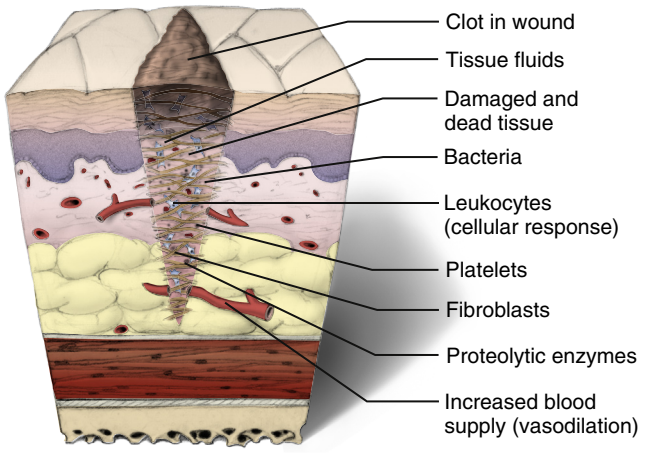
• **Fig. 2.3** Superficial landmarks of the lateral chest wall illustrating the anterior, mid, and posterior axillary lines.



• **Fig. 2.4** Cross-section of the skin illustrating the structural differences between the epidermis, dermis, and subcutaneous layers. (From Pieknik R. *Suture and Surgical Hemostasis: A Pocket Guide*. Philadelphia: Elsevier; 2006.)



• **Fig. 2.5** Orientation of subcutaneous collagen fibers within the dermis (Langer's lines). Incisions in or parallel to these lines are considered to produce a less visible scar. (From Sherris DA, Kern EB. *Essential Surgical Skills.* 2nd ed. Philadelphia: Elsevier.)



• **Fig. 2.6** Cross-section of skin incision illustrating affected structures and response to initial tissue injury. (From Sherris DA, Kern EB. *Essential Surgical Skills.* 2nd ed. Philadelphia: Elsevier.)

Breast Tissue

The breast consists of epithelial glands that develop in the superficial fascia overlying the musculature of the chest wall including the pectoralis major, serratus anterior, external oblique, and rectus abdominis fascia (Fig. 2.7). The breast extends from ribs 2 through 6 in the midclavicular line and from the sternum to the midaxillary line. The blood supply to the breast is from perforating branches of intercostal arteries, as well as branches of the axillary and subclavian arteries. The retromammary space is located between the membranous layer of superficial fascia and the deep fascia covering the muscles of the thorax. The mobility of the female breast on the body wall is largely due to this space; in self-examination for breast cancer, mobility can be decreased. The relative amount of fatty tissue increases as glandular tissue decreases in women following menopause.

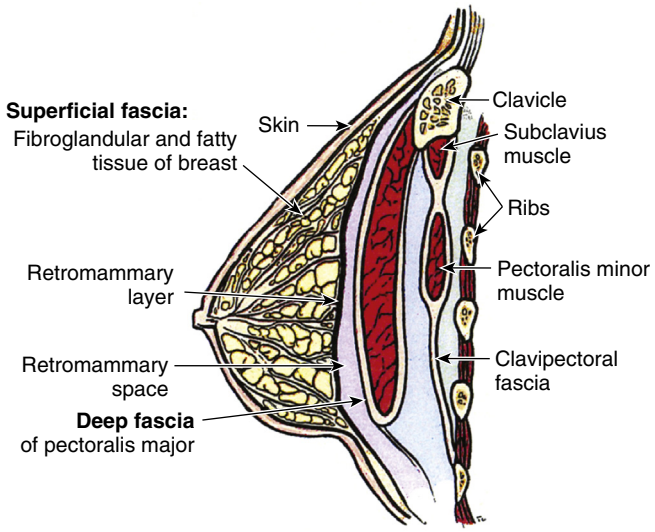
Clinical Correlations

- When implanting CIEDs in women, the dissection of the pocket must be carried down to the deep fascial layer covering the pectoralis major muscle. Dissection is then carried inferior, taking care to stay within the retromammary space. This approach is necessary to avoid implanting the generator within the breast tissue. If the generator is implanted into the breast tissue, the patient may experience chronic pain, mastitis, and/or excessive motion (flipping, or dissection further inferior and lateral) (Fig. 2.8).
- Large breasts can also result in excess weight pulling on the pectoralis major and the device following implantation when the patient assumes an upright posture (Fig. 2.9). The physician can anticipate this circumstance and alternatively

secure the pulse generator on a "loose, hanging" suture placed high on the clavicular head of the pectoralis major muscle (without abutting the clavicle).

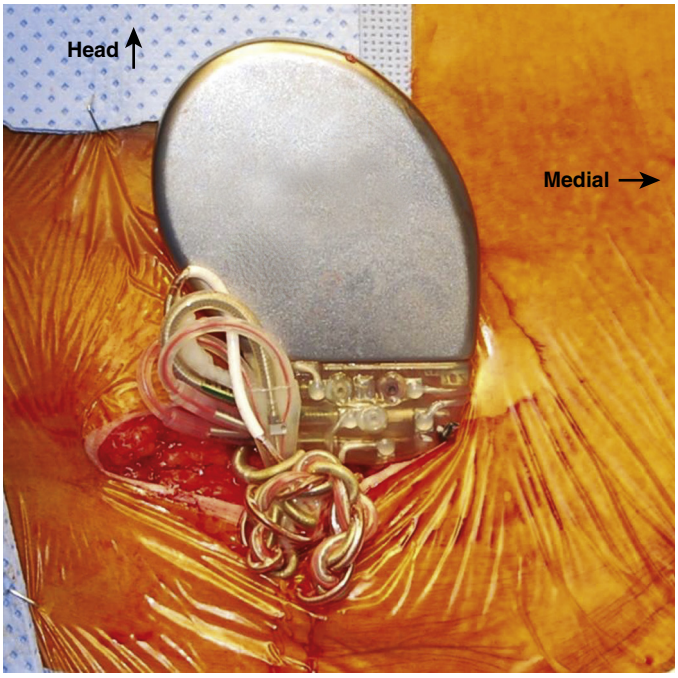
Skeletal Anatomy

The body chest wall is composed of 10 ribs attaching posteriorly to thoracic vertebrae and curving inferior to attach to the sternum anteriorly (Fig. 2.10). The superior thoracic aperture or opening is formed by the T1 vertebrae, first ribs, and their costal cartilages and the superior border of the manubrium, the upper part of the sternum.

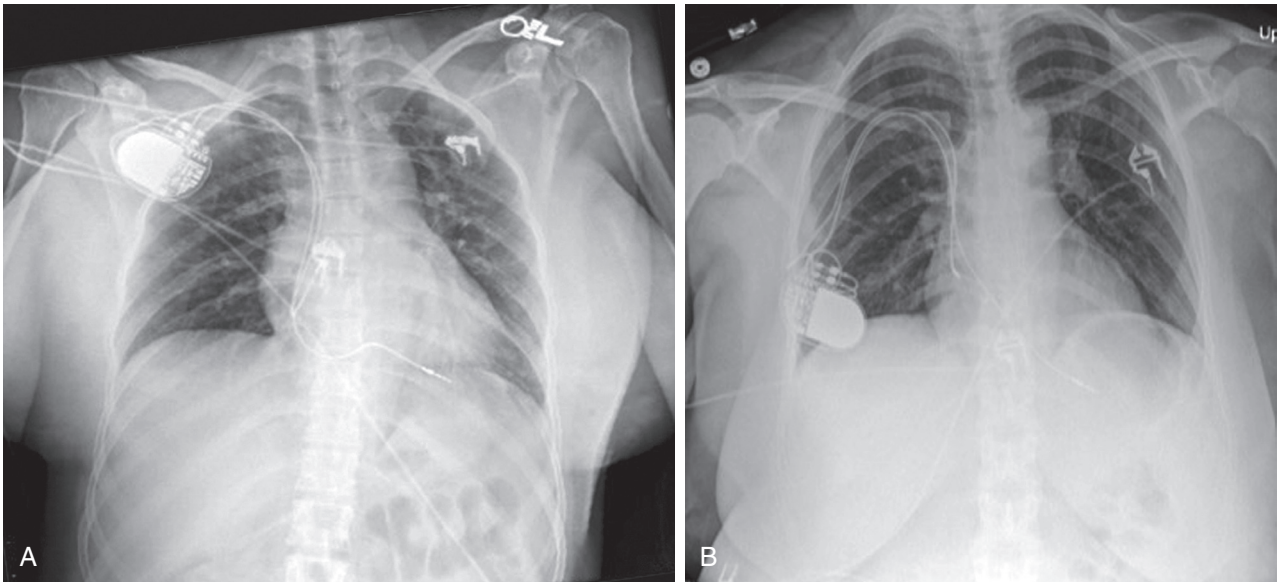


• **Fig. 2.7** Saggital view of the breast and chest wall illustrating the relationship of the various structures. (Drawing by Tyler J.W. Clark.)

The *sternum* is composed of three parts: the manubrium, body, and xiphoid process. The sternum articulates with the cartilages of the first seven ribs and the clavicle. The sternal angle, formed by the junction of the manubrium and body of the sternum, demarcates the level of the second rib, aortic arch, and tracheal bifurcation. The suprasternal or jugular notch is also a consistent



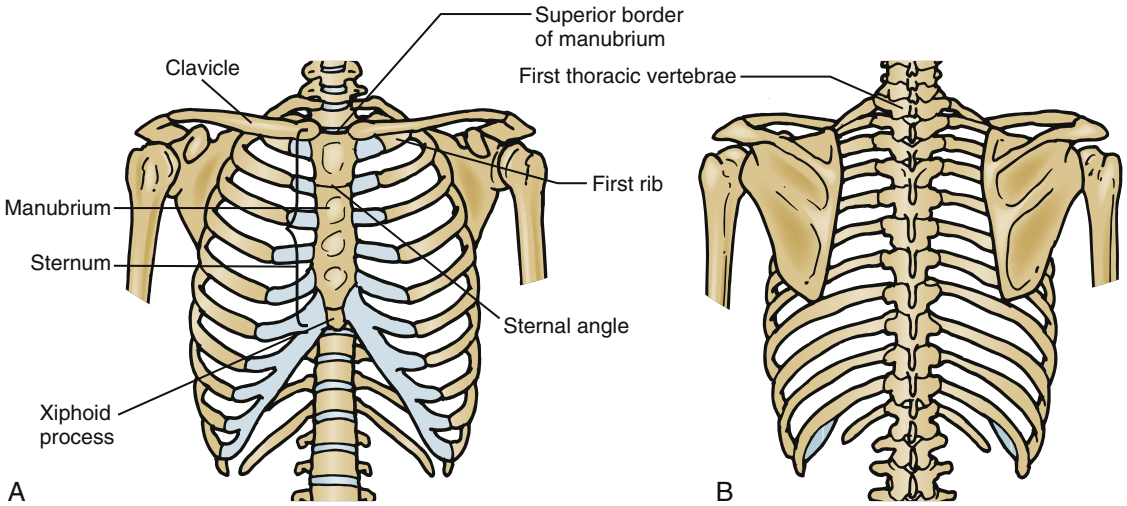
• **Fig. 2.8** A defibrillator generator that had been implanted within breast tissue. Shown are the generator removed from the pocket and the resultant entanglement of the leads ("Twiddler's syndrome"). This can occur due to repeated spontaneous or, less often, patient-initiated rotation of the generator, which twists and entangles the corresponding leads.



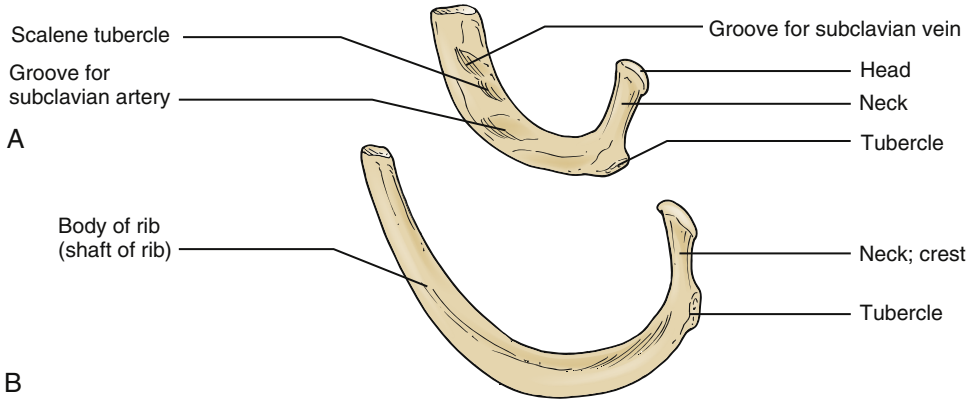
• **Fig. 2.9** Effects of large breasts on a newly implanted pacemaker system. (A) Patient in the supine position with the breasts displaced laterally. Note the location of the pacemaker generator and redundancy of the atrial and ventricular leads within the cardiac silhouette. (B) Patient in an upright position after the pacemaker implant procedure. The breasts are now subject to gravity, pulling the generator inferior with the ipsilateral breast. Note the inferior displacement of the generator and subsequent loss of redundancy of both leads. Anticipating movement of subcutaneous chest wall tissue or well-developed musculature can prompt the implanting physician to be certain that the leads are well secured in the pocket.

landmark for the medial course of the subclavian vein as it joins the jugular vein to become the brachiocephalic vein. Thus, using only bony anatomic landmarks, subclavian venous access is generally successful when a needle is advanced beneath the clavicle, along its inferior third, and toward the suprasternal notch.

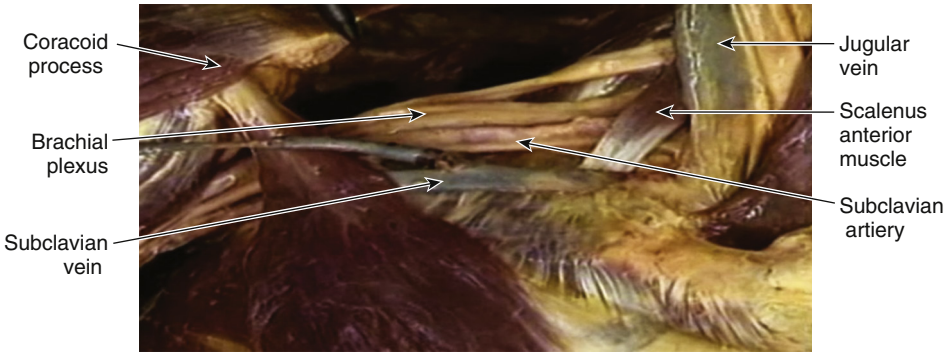
The first rib is broad, flat, and the shortest of the ribs (Fig. 2.11). On its superior surface, a tubercle for the attachment of the scalenus anterior muscle from the neck separates two shallow grooves: for the subclavian vein anteriorly and the subclavian artery posteriorly (Fig. 2.12).



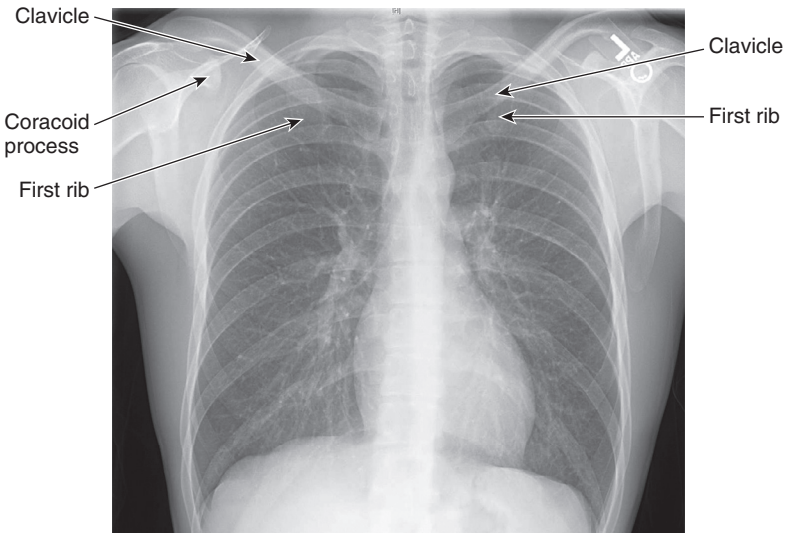
• **Fig. 2.10** Bony structures of the thorax. (A) Anterior chest wall. (B) Posterior chest wall.



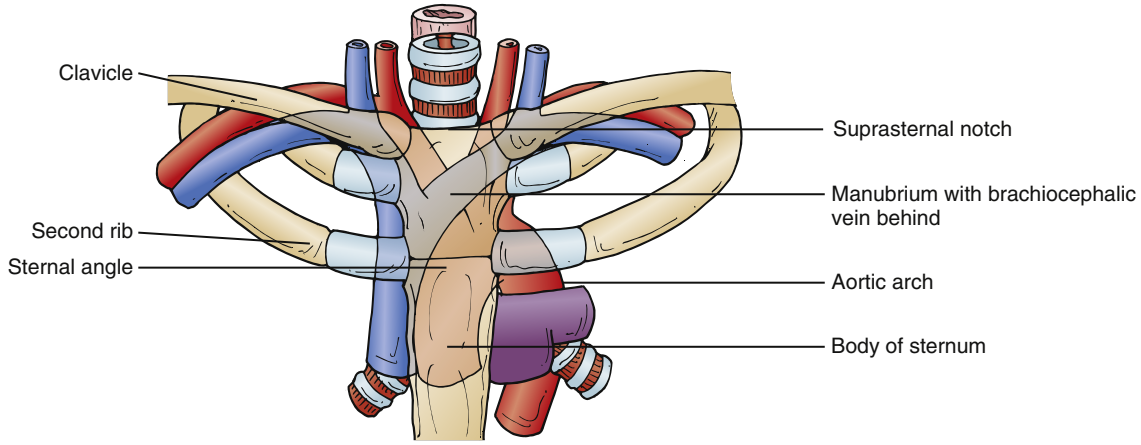
• **Fig. 2.11** Structure of the (A) first and (B) second ribs. (From Waschke J, Paulsen F, eds. *Sobotta Atlas of Human Anatomy,* ed 15. Philadelphia: Elsevier; 2013.)



• **Fig. 2.12** Cadaveric dissection of the right upper chest with clavicle removed to reveal the first rib with insertion of the scalenus anterior muscle. The subclavian vein passes anterior and the subclavian artery passes posterior to the scalenus anterior muscle. Also note that the axillary vein becomes the subclavian vein at the lateral border of the first rib. (From Acland RD. *Acland's Atlas of Human Anatomy, 6 DVD Set.* Philadelphia: Lippincott Williams & Wilkins; 2003.)



• **Fig. 2.13** Chest radiograph illustrating the shadow of the first rib to be used as a landmark for vascular access to the axillary vein.



• **Fig. 2.14** Sternal angle at the level of the aortic arch and tracheal bifurcation. Surrounding structures include the first rib, body of the sternum, clavicles, and subclavian arteries and veins.

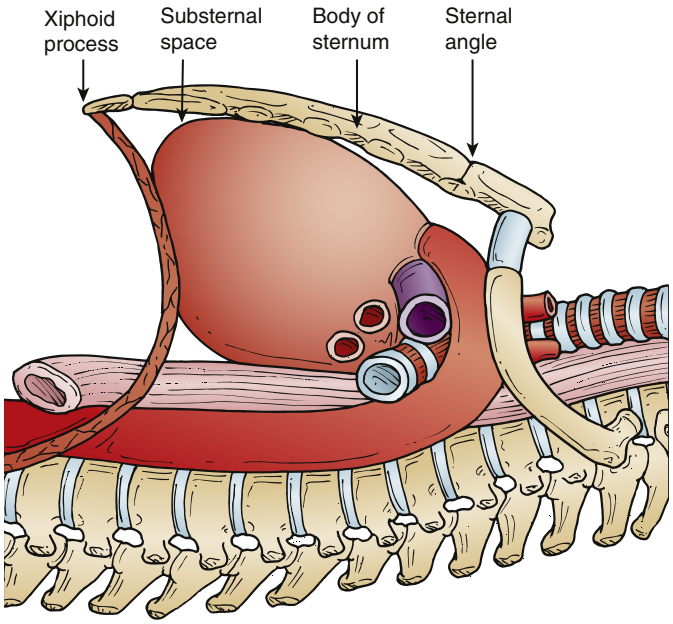
The first rib is a fluoroscopic landmark for the course of the axillary vein where it becomes the subclavian vein at the lateral border of the first rib. The shadow of the first rib is a convenient target to access the axillary vein, as the rib acts as a bony barrier to the thoracic cavity (Fig. 2.13).

The sternal angle is a palpable landmark formed by articulation of the manubrium and the body of the sternum at the level of the aortic arch and tracheal bifurcation (Fig. 2.14). The costal cartilages of the second ribs attach to the sternum in notches at the sternal angle, and ribs 3 through 6 attach to the body of the sternum. Ribs 7 through 10 connect to form a common costochondral cartilage that attaches to the sternum, while ribs 11 and 12 do not attach to the sternum (see Fig. 2.10). Inferiorly, the small and variable xiphoid process attaches to the sternal body at the subcostal angle of the inferior thoracic aperture. It is in the plane of the inferior border of the heart (see Fig. 2.10).

The substernal notch is an access point for the subcostal or pericardial space when a pericardiocentesis or other interventional procedures are performed. The small substernal space that exists between the posterior table of the sternum and the anterior surface of the right ventricle allows access for an alternative placement of a defibrillator or pacemaker lead in the absence of venous access (currently most often used in patients with congenital heart disease) (Fig. 2.15).

The lateral border of the sternum and the xiphoid process are also important landmarks for placement of a tunneled subcutaneous lead for the subcutaneous implantable cardioverter defibrillator (S-ICD). The xiphoid process marks the inferior point for the lead, which is tunneled medially from the midaxillary pocket and then again superiorly along the lateral margin of the sternum (see Chapter 10).

The *clavicle* connects the trunk to the upper limb by extending from the manubrium of the sternum to the acromion of the scapula. It defines the superior border of the chest wall (see Fig. 2.10). It has an S-shaped contour and is palpable along its entire length. The sternocleidomastoid

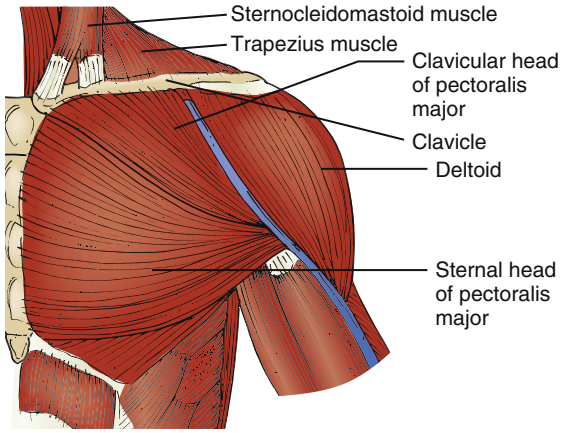


• **Fig. 2.15** A subxiphoid approach can be used as an access point to the anterior mediastinal space for alternative placement of a pacemaker or defibrillator lead. This space is also used for access during pericardiocentesis.

muscle inserts on the medial third of the clavicle from above, and the clavicular head of the pectoralis major originates from the anterior sternal half of the clavicle. The trapezius muscle inserts on the posterior surface and the deltoid originates on the anterior surface of the lateral third of the clavicle (Fig. 2.16).

The clavicle operates like a strut for the upper extremity, allowing significant freedom of motion at the sternoclavicular and acromioclavicular joints when the upper extremity is raised. The sternoclavicular joint stabilizes the clavicle to the sternum by the anterior and posterior sternoclavicular ligaments (Fig. 2.17). A musculotendinous structure, the *subclavius muscle,* originates as a tendon from the junction of the first rib and its costal cartilage, anterior to the costoclavicular ligament, and passes laterally and superiorly to insert onto the inferior surface of the middle third of the clavicle (Figs. 2.17 and 2.18). Contraction of the subclavius muscle moves the clavicle and first rib closer together. The lateral inferior clavicular surface is connected to the coracoid process of the scapula by the conoid and trapezius ligaments.

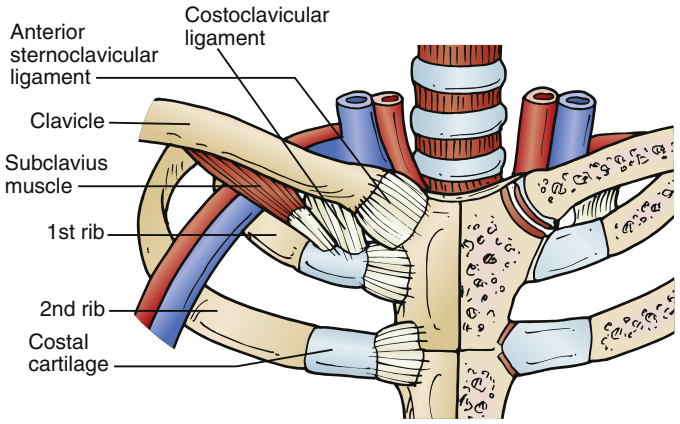
The *coracoid process* of the scapula is an important bony landmark when implanting a transvenous pacemaker or defibrillator device (Fig. 2.19). An anterior projection from the superior surface of the scapula, it is an attachment site for the pectoralis minor, short head of the biceps brachii, and coracobrachialis muscles. In many patients the coracoid process is readily palpated beneath the clavicle in the deltopectoral groove, between the deltoid and the pectoralis major muscles. On chest x-ray, the coracoid process can be seen inferior to the acromioclavicular joint and superiormedial to the glenoid cavity for the head of the humerus (see Fig. 2.13).



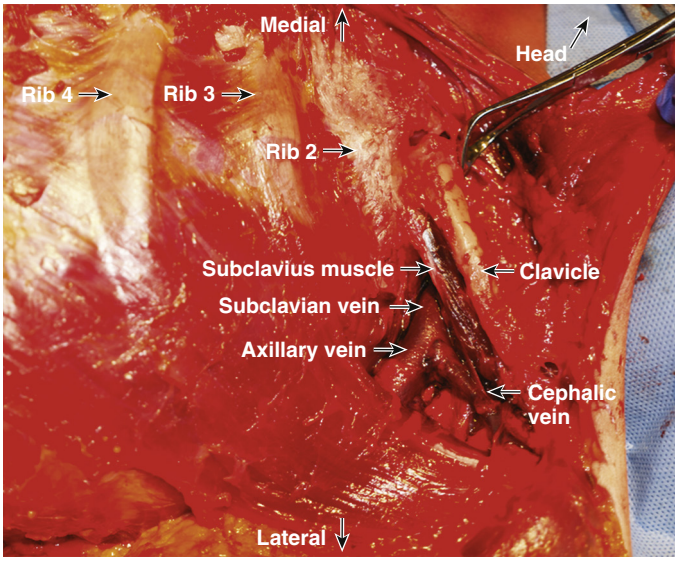
• **Fig. 2.16** Insertion of major chest muscles on the clavicle.

Clinical Correlations

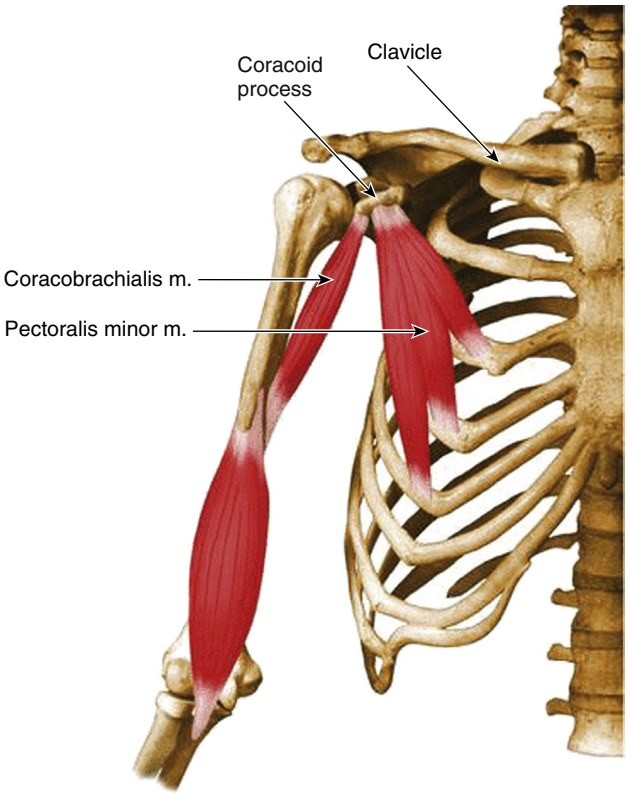
- A pacemaker or ICD placed in its usual position in the upper chest with transvenous leads is in close proximity to the clavicle. A lead coursing in the infraclavicular space will be subject to significant forces acting on it as a result of normal clavicular motion.
- The subclavius muscle can be the primary anatomic structure restricting lead movement and facilitating lead damage. The continuous motion of the clavicle and, especially, the more tendinous portion of the subclavius muscle can result in damage to the lead insulation, commonly referred to as "subclavian-clavicular crush." The subclavius muscle is not present in all patients, which may be one variable in the occurrence of the subclavian crush syndrome. If a subclavian venipuncture is performed too medially, the needle will more likely course through the tendinous portion of this muscular structure where the lead fracture may be greater; however, even coursing through the muscular portion poses a risk (Fig. 2.20).
- The axillary vein as it courses over the first rib has become a popular venous access approach as the risk of pneumothorax is less. This is because the first rib provides a bony barrier to the thoracic cavity. The first rib is angled posteriorly at its superior margin, and thus, the angle of the needle should be perpendicular to the plane of the first rib, which is approximately 30 to 40 degrees angled to the chest. This avoids inadvertent advancement of the needle over the rib edge and into the pleural space (see Chapter 8 and Fig. 8.15B).
- The coracoid process marks the lateral aspect of the chest wall. Positioning the pulse generator medial to this fluoroscopic position will lessen the risk of migration into the axilla or of abutting the anterior deltoid muscle (Fig. 2.21).
- The coracoid process also may be used as a radiologic landmark during subpectoral pacemaker or ICD implants to gauge the location of the thoracoacromial neurovascular bundle as it originates from the axillary vessels and brachial plexus inferior to the coracoid process and deep to the medial border of the superior pectoralis minor muscle to supply adjacent musculature and anterior axillary wall structures[13] (Figs. 2.22 and 2.23).



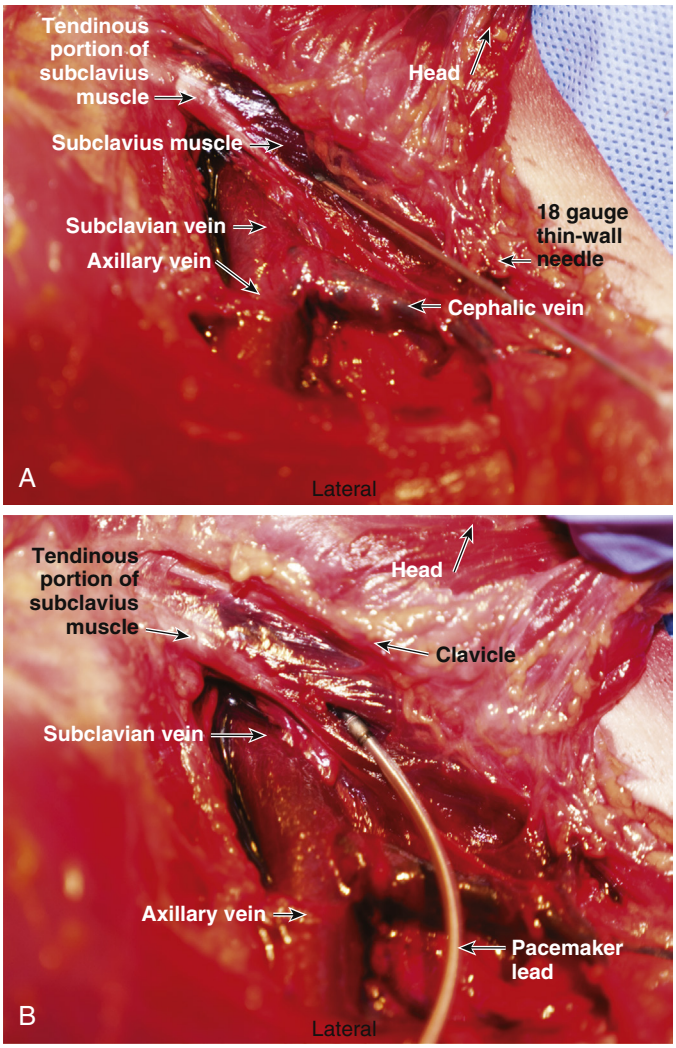
• **Fig. 2.17** Articulation of the clavicle with its corresponding ligaments and the subclavius muscle. The presence of these ligaments permits freedom of motion at the sternoclavicular joint, while the subclavius muscle stabilizes the clavicle and brings the clavicle and first rib closer together.



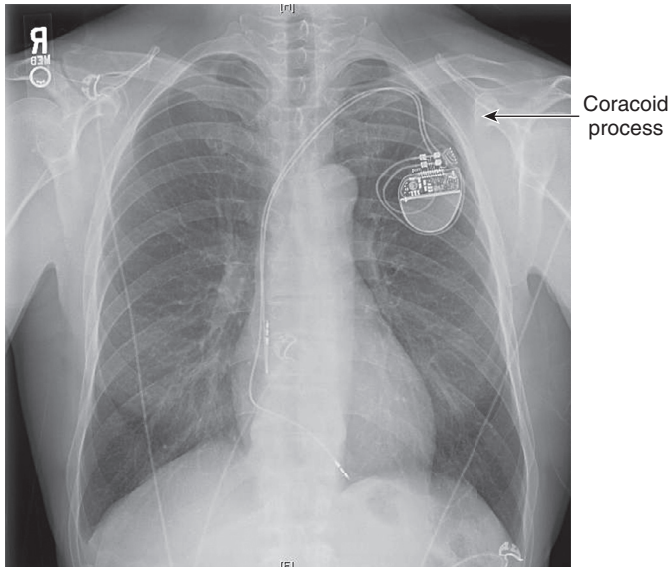
• **Fig. 2.18** Cadaveric dissection of upper left anterior chest wall with clavicle retracted superiorly to reveal the subclavius muscle and sternoclavicular ligaments. The second, third, and fourth ribs are shown, as well as the axillary, cephalic, and subclavian veins.



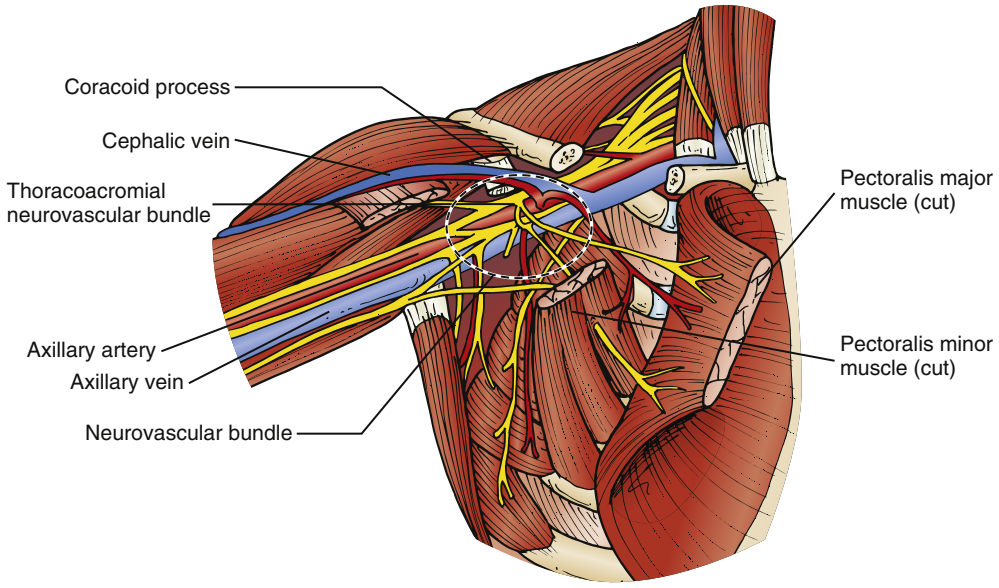
• **Fig. 2.19** Insertion of the pectoralis minor muscle on the coracoid process of the scapula. (Courtesy University of Washington School of Medicine, Seattle.)



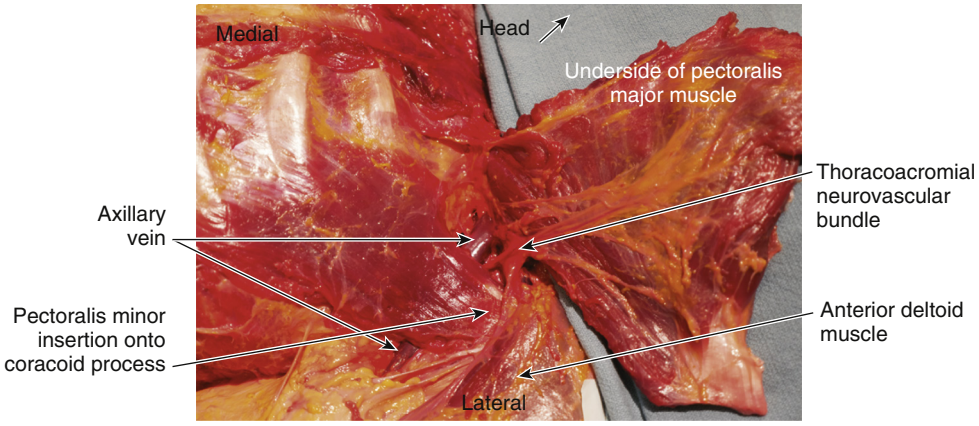
• **Fig. 2.20** Cadaveric dissection of the left anterior chest wall. (A) Direction of subclavian vein vascular access. An 18-gauge needle will pass through the muscular portion of the subclavius muscle if the entry is lateral, whereas it will pass through the tendinous portion if too medial. (B) A lead will then be passed through the same portion of the subclavius muscle, which subjects the lead to repetitive forces from the clavicle, subclavius muscle, and rib motion, increasing the risk of lead fracture.



• **Fig. 2.21** Anterior-posterior chest radiograph illustrating a dual-chamber pacemaker system with the generator ideally located medial to the radiographic location of the coracoid process.



• **Fig. 2.22** The dashed circle illustrates the relationship of the coracoid process and origin of the thoracoacromial neurovascular bundle just posterior to the medial edge of the superior pectoralis minor muscle. Using the coracoid process as a radiographic landmark to identify the location of the neurovascular bundle will help the implanting physician avoid injury to this structure when dissecting beneath the pectoralis major muscle.



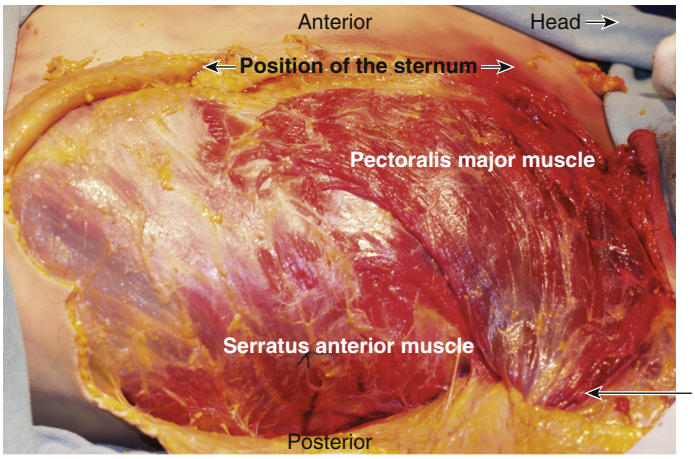
• **Fig. 2.23** Cadaveric dissection of the left chest wall. The pectoralis major muscle is reflected back such that the neurovascular bundle's location on the undersurface of the pectoralis major muscle can be appreciated. The insertion of the pectoralis minor muscle onto the coracoid process can also be noted.

Musculature

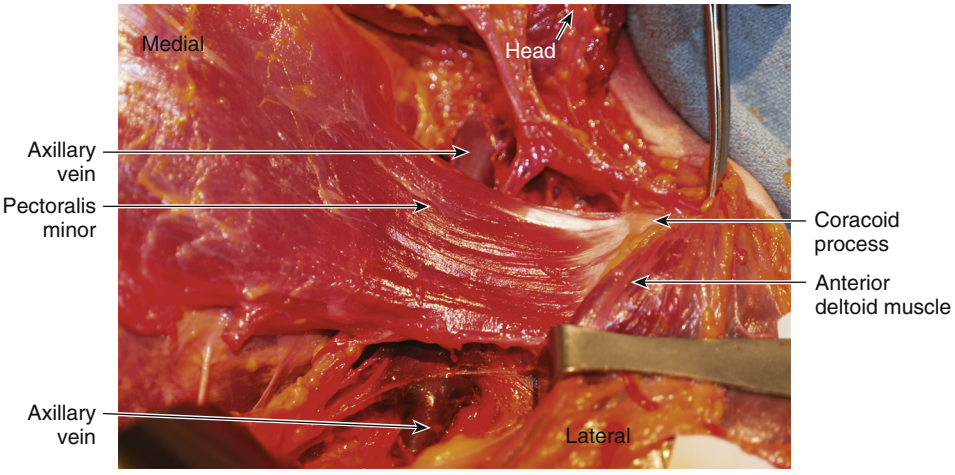
The primary muscle encountered in CIED implantation is the *pectoralis major*. It forms the majority of the upper chest, is covered with the pectoralis fascia (a deep fascial layer), and, in the superior portions, is covered by the platysma muscle extending to the second or third rib. The pectoralis major is composed of three portions: the clavicular head, which arises from the medial third of the clavicle; the sternal head, arising from the anterior manubrium and sternum; and the small abdominal portion, which arises from the sheath of the rectus abdominis (Figs. 2.1B, 2.16, and 2.24).

The sternal and clavicular heads of the pectoralis major muscle fibers converge to insert on the upper end of the humerus, attaching to a lateral lip of the intertubercular groove. The pectoral branch of the thoracoacromial artery supplies the pectoralis major muscle. Innervation arises from the lateral and medial pectoral nerves C5 and C6 for the clavicular head and C7, C8, and T1 for the sternal head. The pectoralis major muscle is a powerful adductor of the humerus and is responsible for internal rotation of the humerus. When working in conjunction with other muscles, it results in internal rotation of the humerus.

The CIED pulse generator is usually placed anterior to the pectoralis major muscle, between the deep (prepectoralis) fascial layer and the subcutaneous tissue with its membranous layer of the superficial fascia (Scarpa's fascia). Alternatively, the CIED generator may be placed posterior to the muscle (subpectoral implant) between the pectoralis major and the pectoralis minor muscles. The lateral border of the pectoralis major forms the medial border of the deltopectoral groove, where the cephalic vein can be isolated for use in transvenous pacemaker or ICD lead placement.



• **Fig. 2.24** Cadaveric dissection of the anterior chest wall illustrating the pectoralis major muscle and its relationship to adjacent structures.



• **Fig. 2.25** Cadaveric dissection of the left chest wall illustrating the pectoralis minor muscle and its relationship to adjacent structures. Note the pectoralis major muscle has been removed.

The *pectoralis minor* is a small, flat muscle lying directly on the chest wall and deep to the pectoralis major (Figs 2.19 and 2.25). The pectoralis minor muscle originates variably from three consecutive ribs 2 to 6 near their costal cartilages and inserts on the medial side of the coracoid process anterior to the thoracoacromial neurovascular bundle. The blood supply to the pectoralis minor muscle is the pectoral branch of the thoracoacromial trunk; it is innervated by the medial pectoral nerve from C7, C8, and T1. The primary action of the pectoralis minor muscle is to stabilize the scapula by drawing it inferiorly and anteriorly against the chest wall.

The *anterior deltoid muscle* originates from the lateral third of the clavicle and the fibers descend vertically to form the lateral margin of the deltopectoral groove on the way to attach onto the deltoid tuberosity of the humerus (see Fig. 2.16). Blood supply is provided by the posterior circumflex artery from the axillary artery and the deltoid artery arising from the pectoral branch of the thoracoacromial trunk. Innervation is provided from the axillary nerve and nerve roots at C5 and C6. The vertical orientation of the fibers is consistent with abduction; the deltoid muscle is a powerful extensor and flexor of the arm at the shoulder.

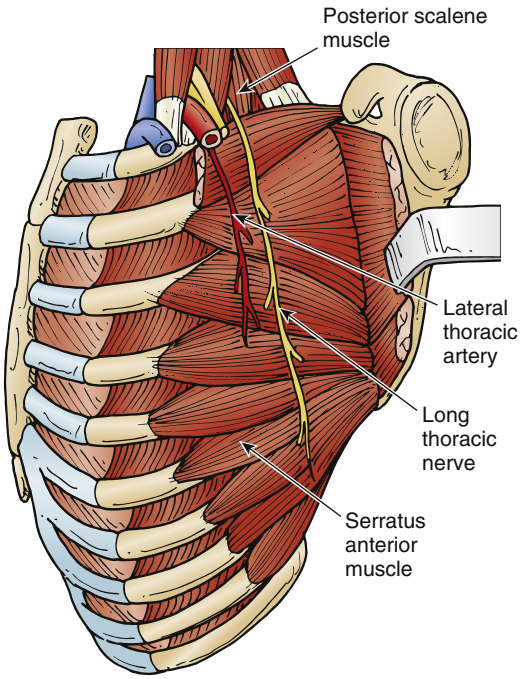
The *deltopectoral groove* defines the lateral border of the clavicular head of the pectoralis major and medial border of the anterior deltoid muscle. The cephalic vein is located beneath the fat pad of the deltopectoral groove and therefore is, in general, easily accessed by direct venous cutdown in this space (see Fig. 2.1). The axillary vein also courses in this space as it crosses the superior aspect of the deltopectoral triangle as a continuation of the brachial vein onto the chest.

The *subclavius muscle,* as discussed earlier, is a short, musculotendinous structure beneath the clavicle and is covered by clavipectoral fascia (fibrous connective tissue high in collagen). The tendinous portion arises from the junction of the first rib and its costal cartilage (see Figs. 2.17 and 2.18). The subclavius passes obliquely lateral and superior to the inferior surface of the middle third of the clavicle, inserting into a groove on the underside of the clavicle. This structure receives its blood supply from the clavicular branch of the thoracoacromial trunk and its nerve innervation from the upper trunk of the brachial plexus (C5, C6). The primary action of the subclavius is to

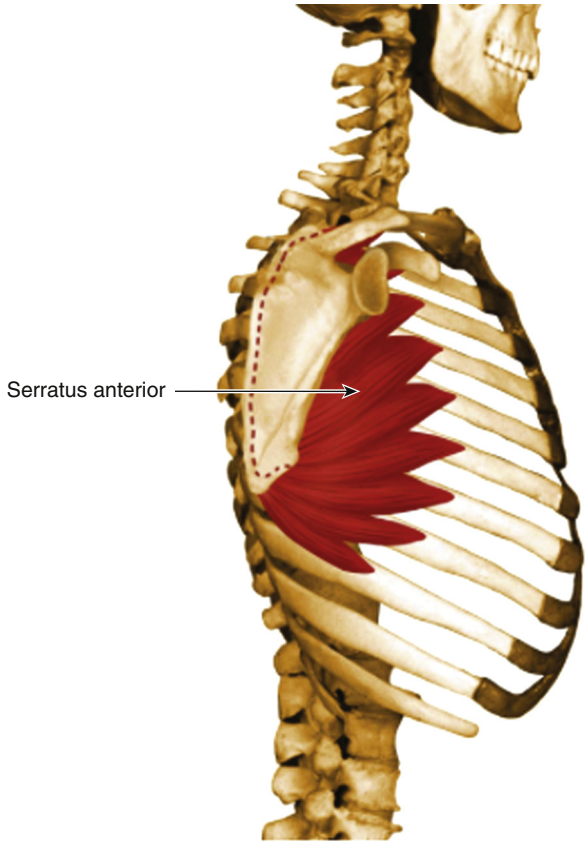
stabilize the clavicle at the sternoclavicular joint and provide traction on the clavicle, thereby drawing the shoulder forward and downward.

The *serratus anterior muscle* is a stabilizing muscle of the lateral chest wall (Fig. 2.26). This muscle originates from the superolateral surfaces of the upper eight or nine ribs and inserts into the vertebral border of the scapula. The blood supply is from the thoracodorsal branch of the subscapular artery and the lateral thoracic branch of the axillary artery, and the nerve innervation is from the long thoracic nerve (C5–C7). The nerve courses anterior to the scalenus posterior muscle and runs inferiorly on the chest wall along the midaxillary line on the outer surface of the serratus anterior giving off penetrating branches to the serratus anterior muscles (Fig. 2.27). The action of the serratus anterior is to draw the scapula forward and upward while abducting and rotating the scapula. The serratus anterior muscle also stabilizes the vertebral border of the scapula and is an important accessory muscle of respiration. Inadvertent injury to the long thoracic nerve in the axilla can result in a winged scapula (Fig. 2.28). It is important to understand these functions as the serratus anterior is the target location for implantation of the subcutaneous (S-ICD) defibrillator generator (see Chapter 10).

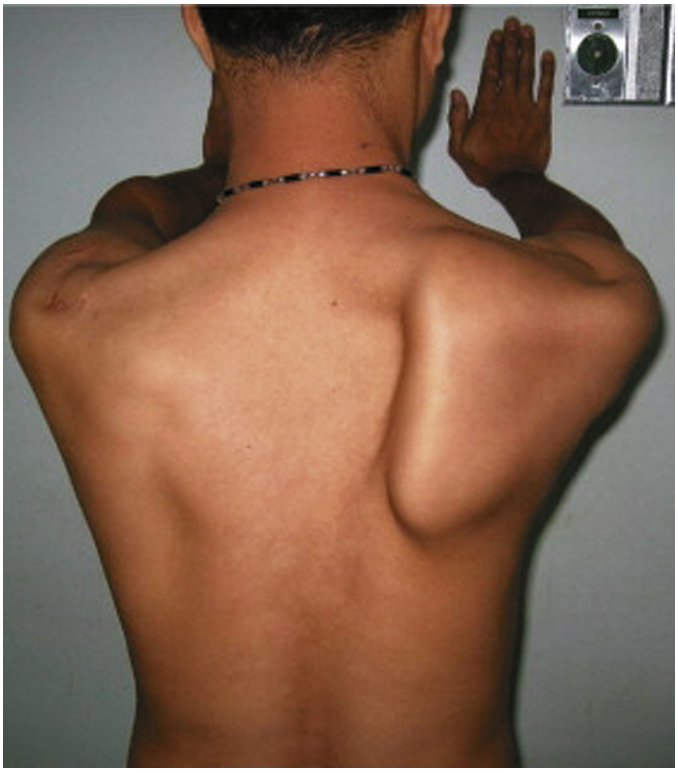
The *latissimus dorsi* is a large powerful muscle that is located in the dorsolateral trunk. It originates on the spinous processes of thoracic vertebrae T7 through T12 as well as ribs 9



• **Fig. 2.27** Course of the long thoracic nerve, which passes anterior to the scalenus posterior muscle and inferiorly along the midaxillary line with multiple penetrating branches into the serratus anterior muscle.



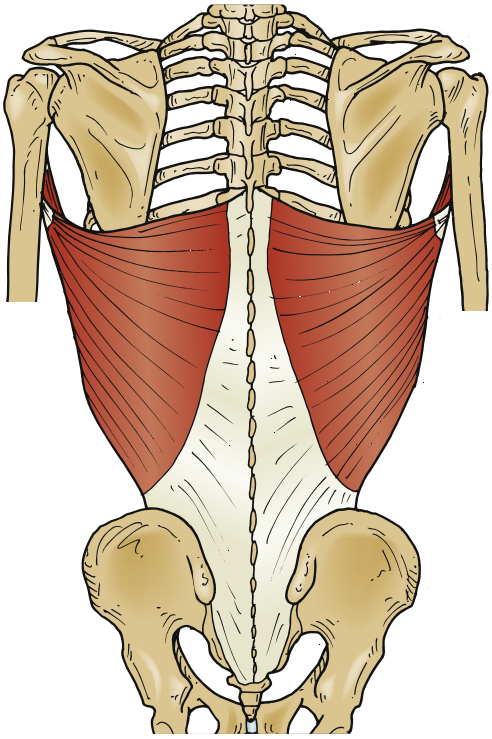
• **Fig. 2.26** Lateral view of the serratus anterior muscle. (Courtesy University of Washington School of Medicine, Seattle.)



• **Fig. 2.28** A patient who experienced a winged scapula as a result of inadvertent injury to the long thoracic nerve. (From Waldman SD. *Atlas of Pain Management Injection Techniques,* ed 3. Philadelphia: Elsevier; 2013.)

through 12 and then swings superiorly and laterally, crossing the inferior border of the scapula, often with fibrous attachments to this bony structure (Fig. 2.29). It inserts into the intertubercular groove of the humerus between the teres major insertion medially and the pectoralis major insertion laterally. This unique flat and broad muscle is responsible for extension, adduction, and horizontal abduction of the torso. It also causes internal rotation of the shoulder and arm and provides flexion of the torso when in an extended position. It participates in extension and flexion of the spine, as well as downward rotation of the scapula during "pull-ups." The nerve innervation to the latissimus dorsi is the thoracodorsal (long scapular) nerve (C6 through C8).

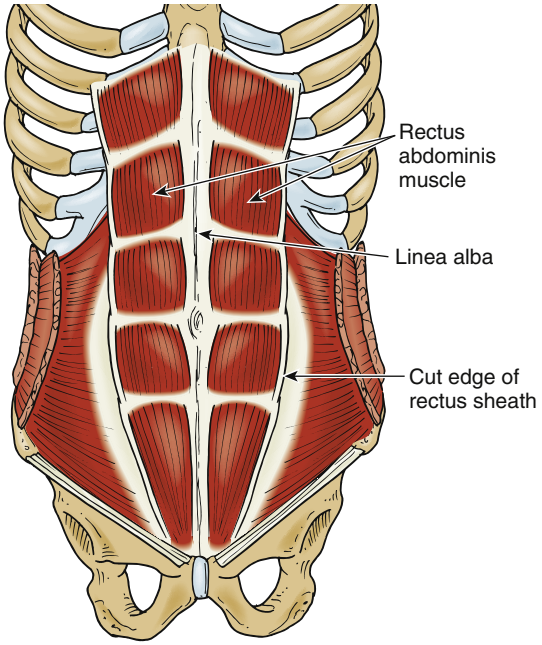
The *rectus abdominis* is a paired muscle running vertically on the side of the anterior abdominal wall, separated by a midline band of connective tissue (linea alba) and is contained within the strong rectus sheath (aponeuroses of lateral abdominal muscles) (Fig. 2.30). The origin of the rectus is the symphysis pubis, pubic crest, and pubic tubercle, and it inserts on the xiphoid process and costal cartilages of the fifth through seventh ribs superiorly. Its blood supply includes the inferior epigastric artery, the superior epigastric artery, and the lower segmental intercostal arteries, and innervation occurs via the thoracoabdominal nerves. The action of the rectus is to provide important postural support, truncal flexion, and stabilization of the pelvic tilt. It provides compression for the abdominal viscera and assists in expiration.



• **Fig. 2.29** Posterior dorsolateral trunk demonstrating the latissimus dorsi muscle.

Clinical Correlations

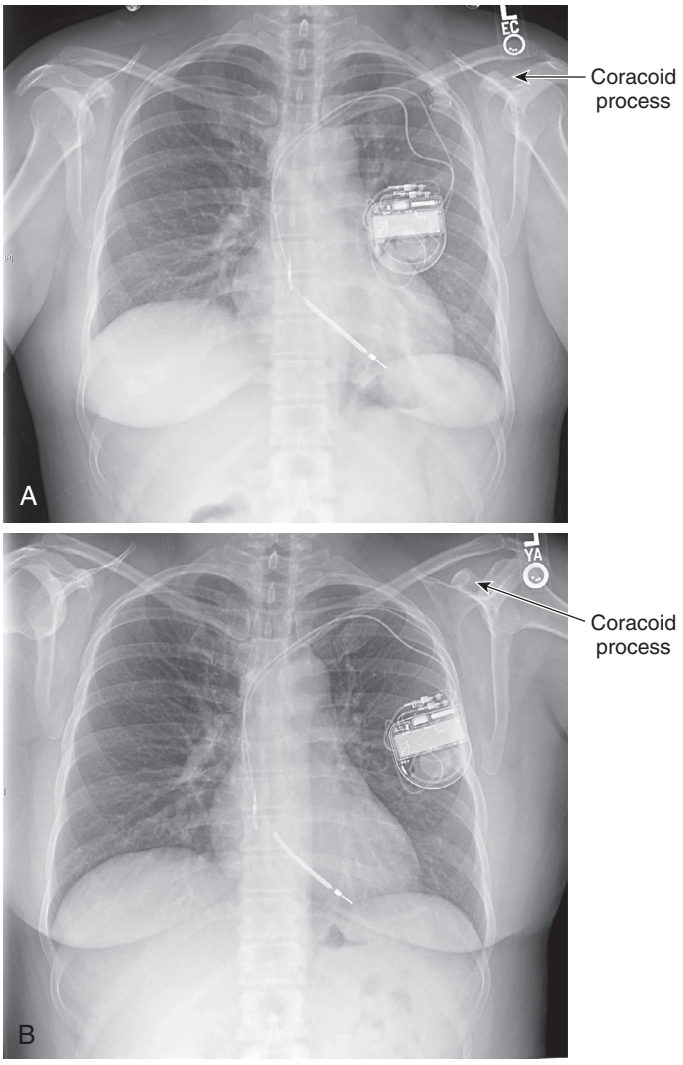
- The implanting physician should recognize that the CIED pulse generator, whether placed subcutaneously or subpectorally, will be subject to significant movement with normal chest motion. Patients who engage in more vigorous exertional activities could inadvertently facilitate displacement of the device inferiorly and/or laterally, even years after initial placement, if their activity level significantly changes (Fig. 2.31A–B).
- The usual approach to a pectoral subcutaneous transvenous pacemaker or defibrillator implant is to place the generator on top of the prepectoralis fascia (deep fascia). However, in patients with little subcutaneous tissue, an extra layer of protection may be achieved by dissecting the prepectoralis fascia off of the pectoralis major muscle and placing the generator under the prepectoralis fascia. (Fig. 2.32).
- When performing a subpectoral implant, it is helpful to note that the fibers of the pectoralis minor run perpendicular to those of the pectoralis major muscle, providing a visual distinction between these two muscles when operating in the subpectoral space (Figs. 2.1B and 2.33). The dissection should not extend into or through the pectoralis minor as the correct location for the device is to be placed on top of this muscle. Identification of the pectoralis minor muscle is required when performing a subpectoral CIED implant (Fig. 2.34).
- Identification of the deltoid muscle is of clinical importance when dissecting in the deltopectoral groove for cephalic vein access. Noting the direction of the muscle fibers and distinguishing them from the pectoralis major muscle helps define the correct plane of dissection (Fig. 2.35). In some individuals the deltoid and pectoral muscles overlap, essentially



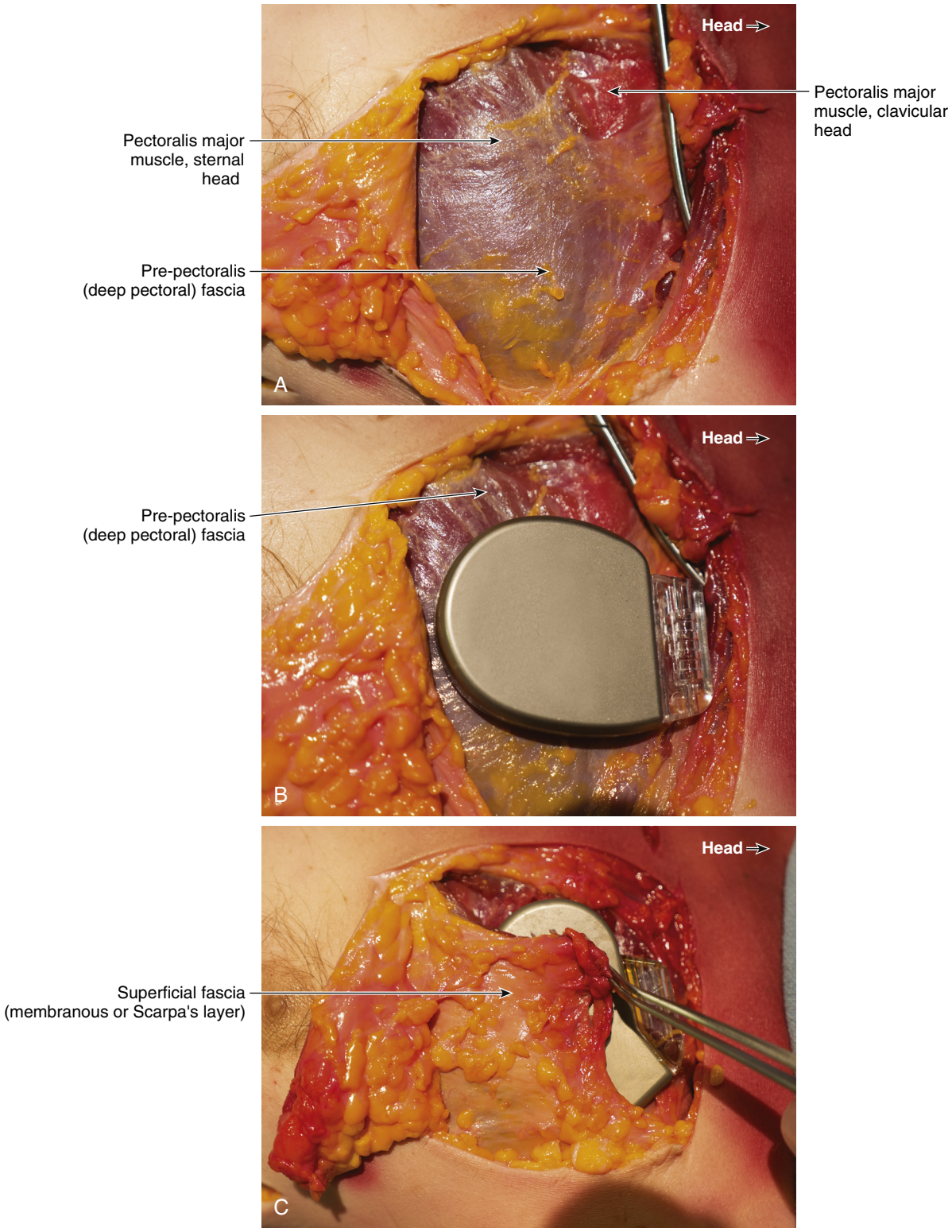
• **Fig. 2.30** Rectus abdominis muscle on the anterior abdominal wall.

obliterating access to the cephalic vein, and caution must be exercised to avoid trauma to the muscular tissue and excess bleeding.

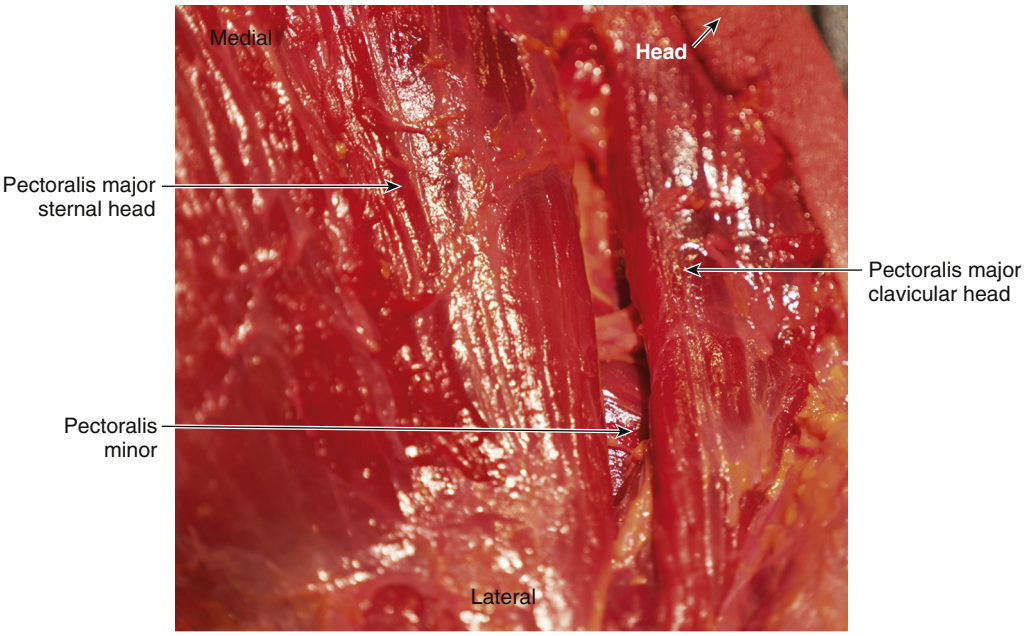
- When using the cephalic vein for CIED lead placement, the lead sleeve should never be sutured to the deltoid muscle as this can result in significant pain and limitation of upper extremity mobility and increase the risk of lead dislodgement. Similarly, pain can be experienced if the lead is allowed to loop laterally, impinging on the deltoid muscle (Fig. 2.36).
- The latissimus dorsi also serves as a posterior landmark when forming a pocket for the S-ICD. Attention should be paid to the ultimate position of the posterior border of the pulse generator in relationship to the latissimus dorsi. Flexion and extension could cause the anterior lip of the latissimus dorsi to abut the device, potentially causing discomfort or displacement. One solution to this problem is to tuck the posterior edge of the generator beneath the anterior lip of the latissimus dorsi, if the overall position of the pocket is considered acceptable as a defibrillation vector (Fig. 2.37).
- Occasionally pulse generators for pacemakers or ICDs (congenital heart disease or in children; see Chapter 11) are placed in the upper abdominal quadrant when the leads are placed on the epicardial surface of the heart. The pulse generator may be placed anterior to the rectus or posterior to the muscle but within the posterior sheath. Generators placed near the midline and below the muscle present a significant challenge when performing a generator replacement as the generator and/or lead may have unknowingly eroded into the abdominal cavity.



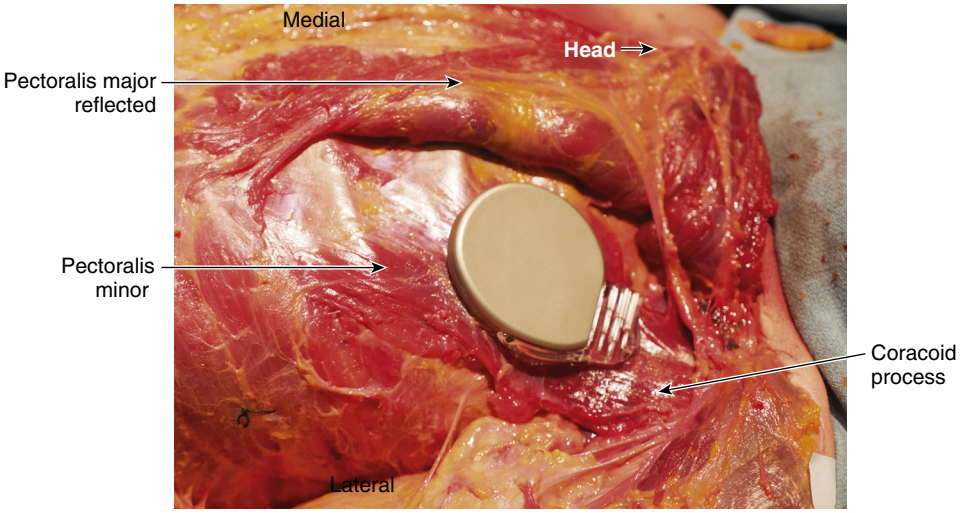
• **Fig. 2.31** Movement of an implantable defibrillator generator due to migration within the subpectoral space. (A) Original position of the generator. (B) Lateral migration of the generator, which occurred after the patient engaged in vigorous upper body exercises.



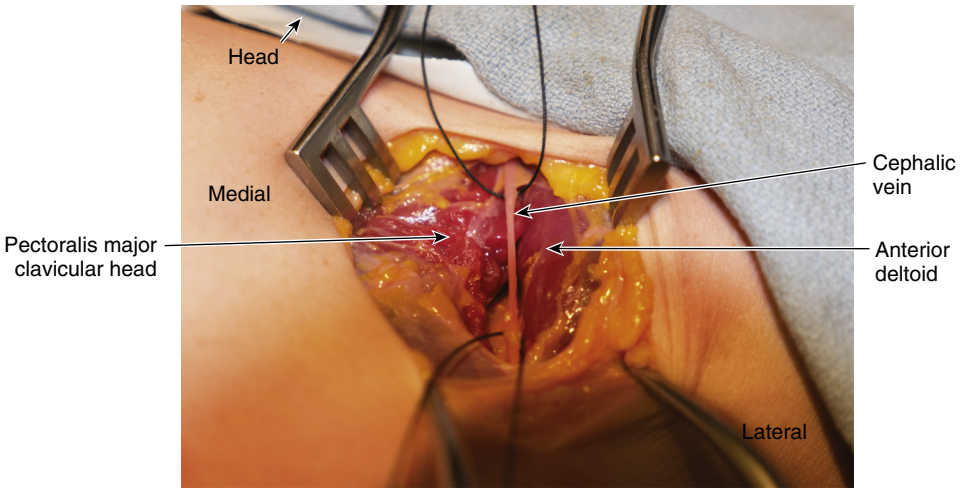
• **Fig. 2.32** Cadaveric dissection showing a dissection in the left upper chest. (A) Pectoralis major muscle with overlying fascia. (B) Defibrillator generator lying anterior to the pectoralis fascia, simulating the most common plane for formation of a subcutaneous pocket. (C) Generator placed on top of the prepectoralis fascia with the superficial fascia reflected back over the device.



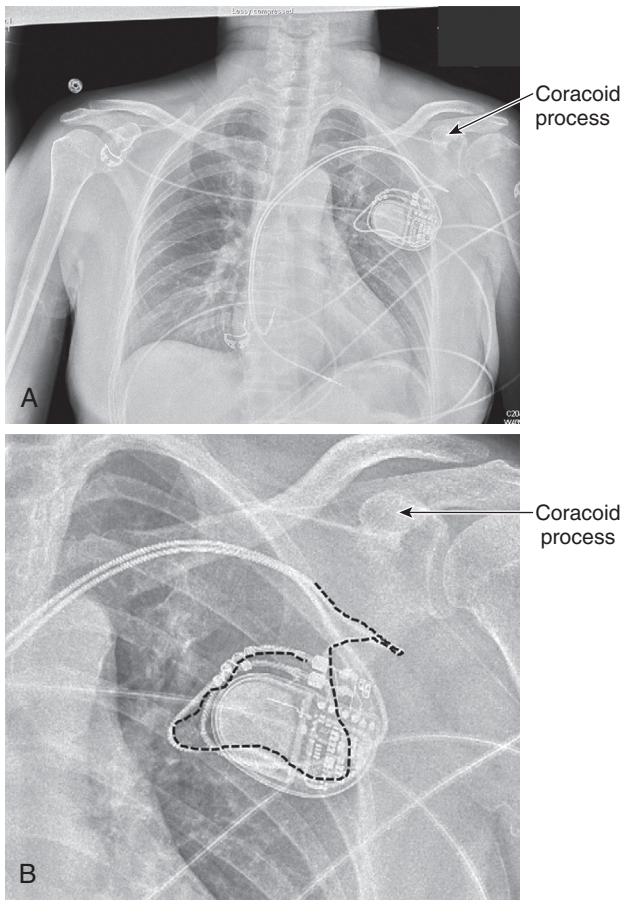
• **Fig. 2.33** Cadaveric dissection of the left subpectoral space demonstrating the separation of the tissue plane between the clavicular and sternal heads of the pectoralis major muscle. This separation provides an atraumatic approach to the subpectoral space. The orientation of the fibers of the pectoralis major and pectoralis minor muscles can be noted to run perpendicular to each other.



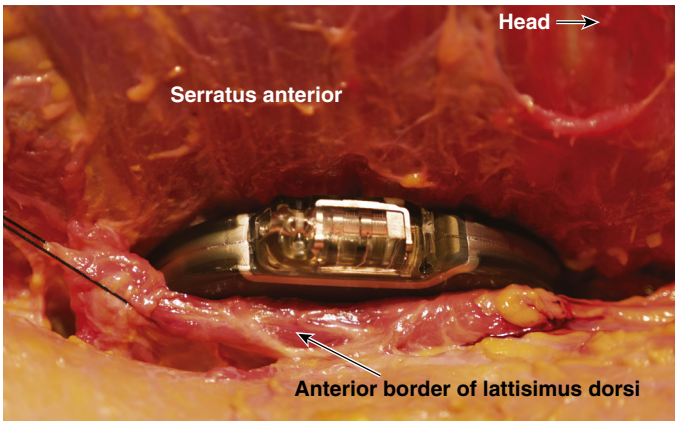
• **Fig. 2.34** Cadaveric dissection of the subpectoral space with a defibrillator generator lying directly on the pectoralis minor muscle, with the lateral edge of the generator placed medial to the coracoid process. Note that the pectoralis major muscle has been rolled back medially.



• **Fig. 2.35** Cadaveric dissection illustrating isolation of the left cephalic vein in preparation for vascular access via cutdown. Note the location of the vessel in the left deltopectoral groove with the pectoralis major muscle located medially and the anterior deltoid muscle located laterally.



• **Fig. 2.36** (A) Posterior-anterior chest radiograph illustrating a pacemaker lead looping laterally over the anterior deltoid muscle in the deltopectoral groove, resulting in pain and limitation of upper extremity mobility. (B) Close-up view with the loop of lead outlined (*black dotted line*). Note the relationship to the coracoid process, demonstrating the lateral position of the lead loop.

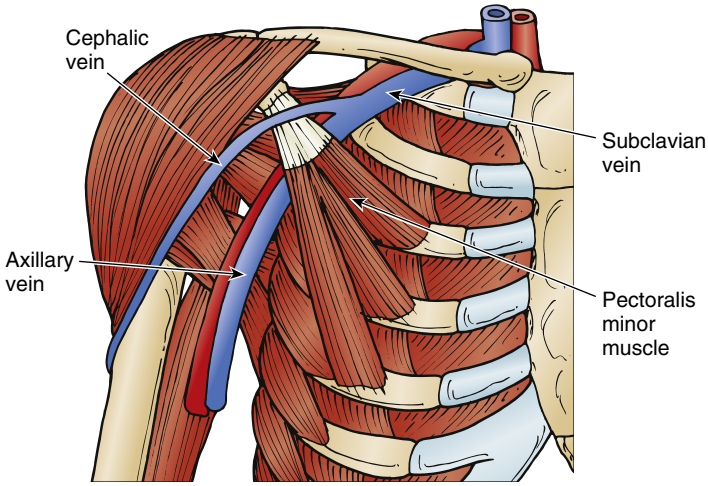


• **Fig. 2.37** Cadaveric dissection of the left upper chest showing placement of a subcutaneous implantable cardioverter-defibrillator on top of the serratus major muscle and beneath the anterior border of the latissimus dorsi muscle. The position relative to the lateral wall would be centered along the midaxillary line. This position provides an acceptable defibrillation vector for most patients without hindering latissimus dorsi mobility.

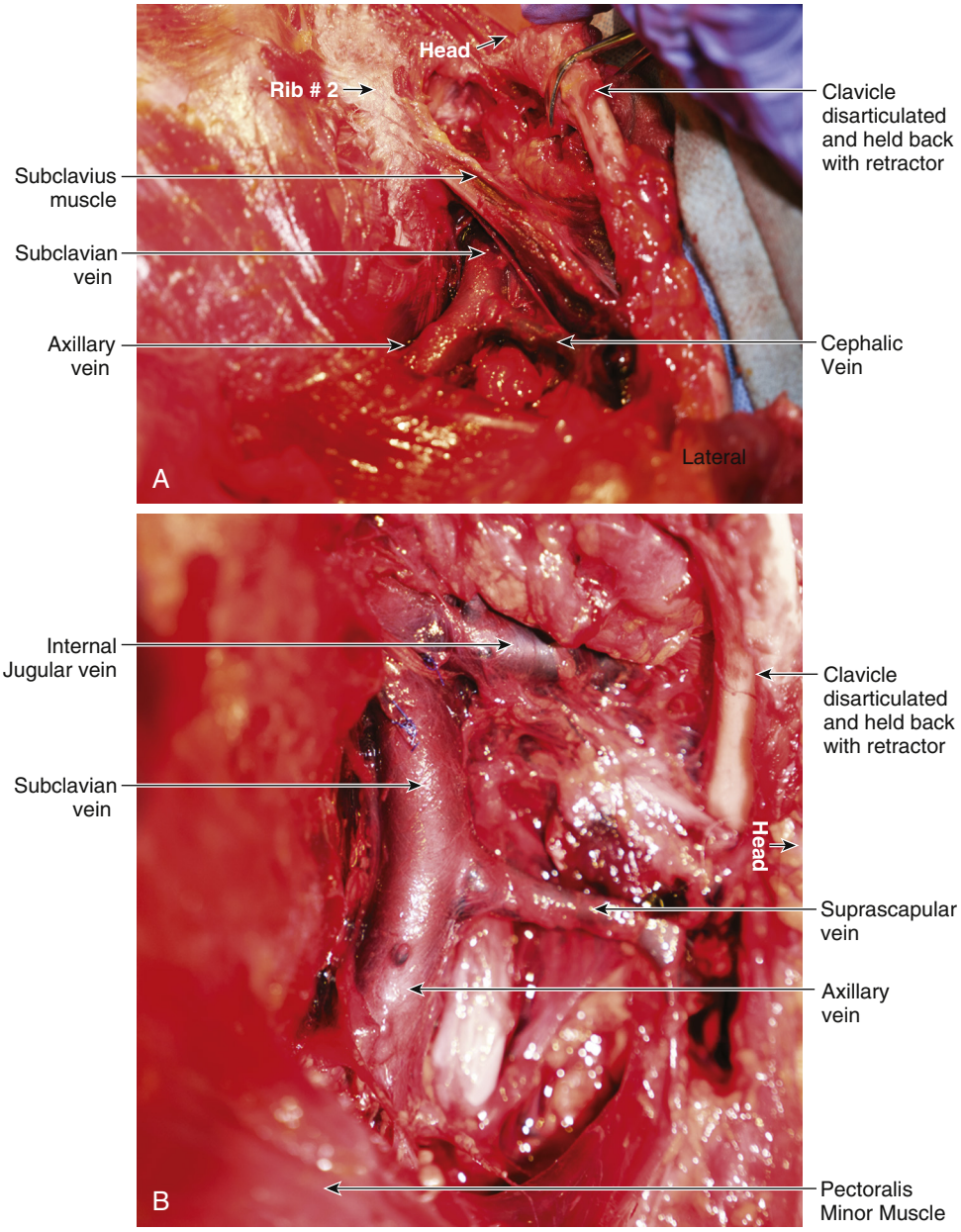
Venous Vasculature

Specific points of access occur in anatomic spaces allowing for safe venipuncture. The axillary, cephalic, and subclavian veins can all be accessed for transvenous lead implantation. Understanding the course and relationship to neighboring anatomic structures is important to perform a safe venipuncture.

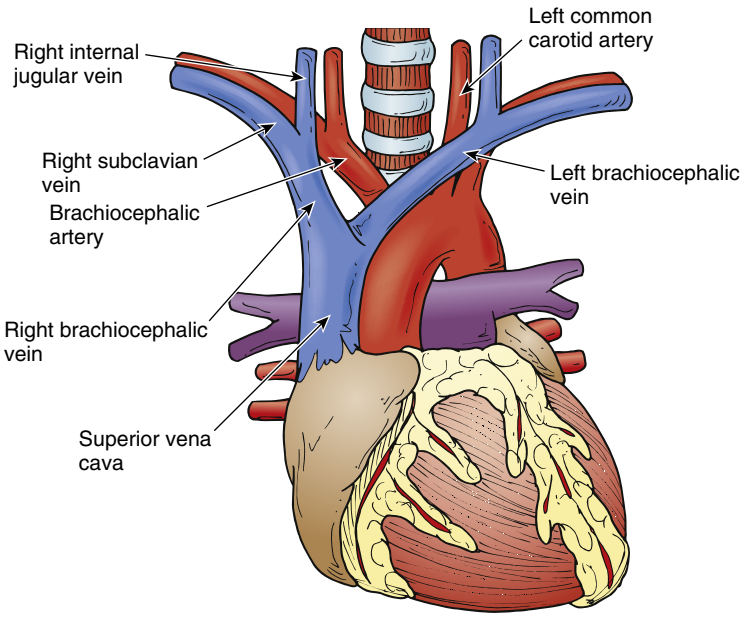
The *axillary vein* is a continuation of the brachial and basilic veins running from the lower margin of the teres major (one of the posterior scapulohumeral muscles) that forms the posterior wall of the axilla. The axillary vein courses beneath the tendon of the pectoralis minor muscle and the clavipectoral fascia and is accompanied by the axillary artery, which lies superior and posterior to the vein (Figs. 2.22 and 2.38). The axillary vein then ascends over the first rib, where it becomes the subclavian vein, to join the jugular vein and descend into the thoracic cavity as the brachiocephalic (innominate) vein (Figs. 2.39 and 2.40). Vessels



• **Fig. 2.38** Anatomic course of the axillary vein with the pectoralis major muscle removed. Note that the axillary vein passes posterior to the pectoralis minor muscle on its course to join the subclavian vein.



• **Fig. 2.39** Cadaveric dissection demonstrating the axillary vein and corresponding vessels in the left axilla. (A) Clavicle retracted with the axillary vein coursing beneath the subclavius muscle. (B) Further dissection of these vessels. Note the insertion of the left internal jugular vein and suprascapular vein into the subclavian vein.



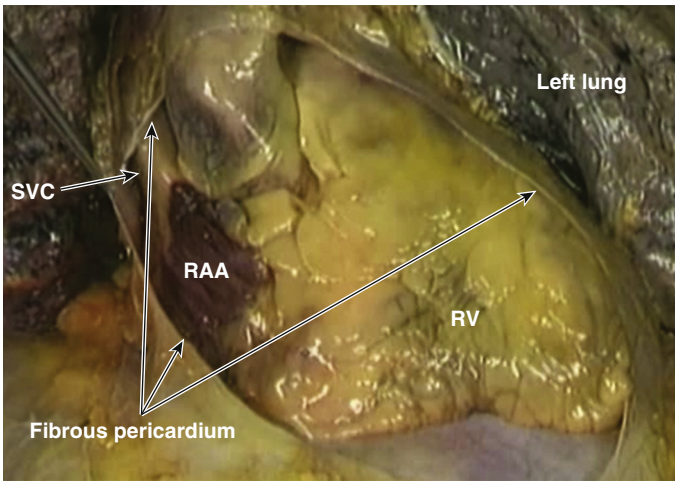
• **Fig. 2.40** Brachiocephalic (innominate) vein and its associated structures. Note that they form at the juncture of the subclavian and internal jugular veins bilaterally and merge to become the superior vena cava.

before this point are accessed outside of the chest and are at lower risk of pneumothorax than when accessing the subclavian vein in the intrathoracic space beneath the clavicle.

The *cephalic vein* arises from the lateral side of the upper extremity (see Figs. 2.22, 2.38, and 2.39). It ascends around the lateral border of the forearm and continues along the lateral border of the biceps. At the level of the deltopectoral groove, it takes a sharp turn posteriorly to pierce the clavipectoral fascia and then enters the axilla to terminate in the axillary vein.

The *subclavian vein* originates as a continuation of the axillary vein at the outer border of the first rib. It then courses beneath the clavicle to become the brachiocephalic (innominate) vein at the juncture with the internal jugular vein. The thoracic duct inserts into the left subclavian vein just lateral to the left internal jugular vein. The subclavian vein is a large vessel, generally measuring 1 to 2 cm in diameter. The subclavian vein has been the preferred choice for pacemaker and defibrillator lead placement. It has a large lumen to contain multiple leads and is available when other approaches prove inaccessible. The approach to this vessel must be as lateral as possible to avoid the thoracic duct and tendinous portion of the subclavius muscle.

The *brachiocephalic veins* (innominate) are formed by the union of the subclavian and internal jugular veins at the level of the sternoclavicular joint (Fig. 2.40; see also Fig. 2.14). Both brachiocephalic veins merge to form the superior vena cava. Unlike the axillary and cephalic vessels, there are no venous valves in either the right or left brachiocephalic vein. The left brachiocephalic vein is longer than the right side since the superior vena cava is located to the right side of the spine. As it crosses to the superior vena cava, the left brachiocephalic vein courses anterior to the left common carotid, brachiocephalic artery, and trachea as it passes superior to the aortic arch.



• **Fig. 2.41** View of anterior surface of the heart with anterior section of fibrous pericardium removed. SVC, superior vena cava; RAA, right atrial appendage; PA, pulmonary artery; RV, right ventricle.

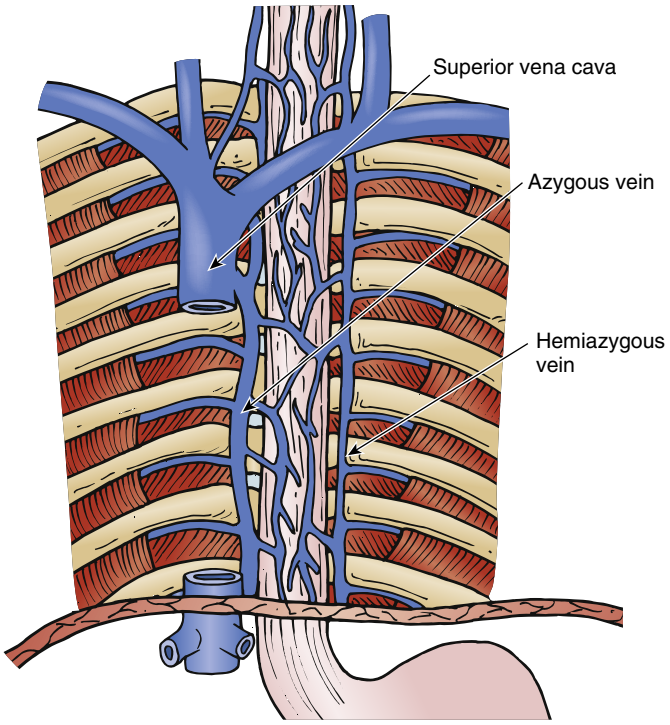
The superior vena cava is a large, valveless vessel formed by the union of the right and left brachiocephalic veins and inserts into the right atrium at the cavoatrial junction below the pericardial reflection. The superior vena cava begins behind the lower border of the first right costal cartilage and drains into the right atrium at the level of the third costal cartilage. The lower half of the superior vena cava is covered by the fibrous pericardium at the level of the second costal cartilage (Fig. 2.41).

The *azygos vein* is a unilateral vessel ascending into the thorax on the right side of the spine (Fig. 2.42). This vessel is part of the azygos venous system, which includes the

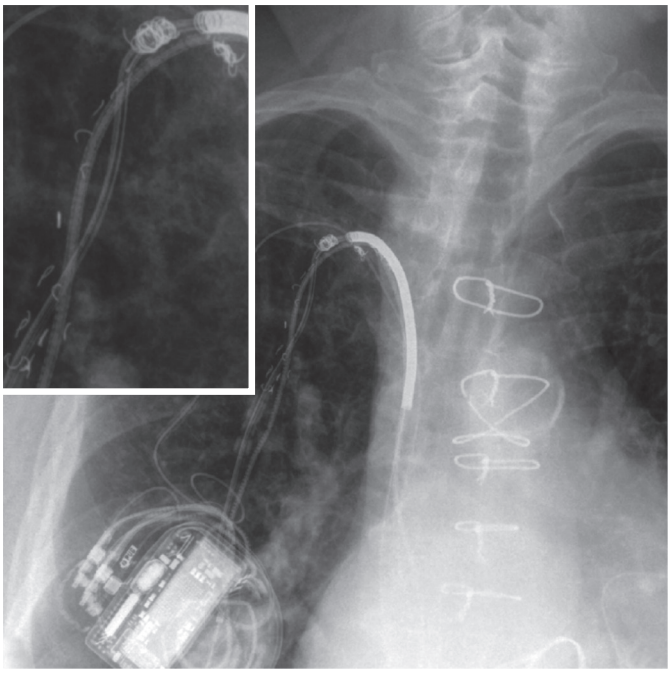
hemiazygos vein. It is formed by the union of the ascending lumbar and right subcostal veins at the T12 level, and ascends the posterior mediastinum to T5 to T6. It then arches over the right mainstem bronchus at the root of the right lung and drains into the superior vena cava through the posterior wall.

Clinical Correlations

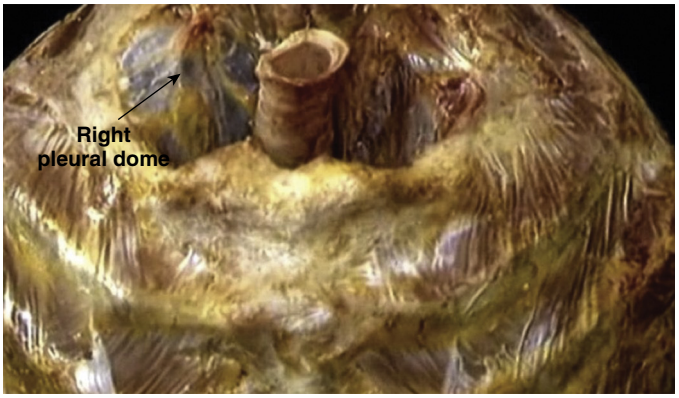
- The right brachiocephalic vein is shorter than the left. It runs posterior to the right costal cartilage of the first rib, then forms a right angle to join the superior vena cava. The right brachiocephalic vein courses medial and inferior to the dome of the right lung pleura. These anatomic differences have implications for venous access and lead placement. There is simply more "space" to advance the venipuncture needle before engaging the superior vena cava when performing a left subclavian venipuncture. This situation creates a tendency (when performing a right-sided procedure) toward a more medial entry into the venous system with a higher likelihood of entrapment within the subclavius musculotendinous structure with risk of lead fracture (Fig. 2.43). The pneumothorax risk is also higher as the dome of the lung has a higher projection on the right chest compared with the left (Fig. 2.44).
- The vascular elasticity of the upper extremity and mediastinal vessels is robust in young healthy individuals (Video 2.2). But as patients age, elasticity diminishes. Caution should always be exercised when advancing guidewires and sheaths through these structures, and the increased risk of perforation in older individuals should be anticipated (Fig. 2.45 and Video 2.3). Inadvertent puncture of the superior vena cava may occur from a guidewire, sheath, or lead tip, resulting in a vascular extravasation, pneumothorax, or pericardial tamponade, depending on the anatomic level of injury (puncture above or below the pericardial reflection) (Fig. 2.46 and Video 2.4). When a left-sided implant is performed, this situation could result in a contralateral pneumothorax. The sharp angle, as the right subclavian



• **Fig. 2.42** Azygous vein and a portion of the azygos venous system. Note that the azygous vein is located in the posterior mediastinum to the right of the vertebral bodies. Its posterior location provides an option for placement of a defibrillator lead in patients in whom standard lead placement has not resulted in an adequate defibrillation vector.



• **Fig. 2.43** Dual-coil transvenous defibrillator lead that was placed via the right subclavian vein. A fracture of the proximal coil is noted on the chest radiograph images. (From Crossley GH, Aznaurov MD, Danter MR, Ellis C. An extreme example of subclavian crush. *J Am Coll Cardiol Electrophysiol.* 2017;3[1]:83.)



• **Fig. 2.44** Cadaveric dissection illustrating the pleural dome of the right lung, which rises higher than that on the left. The chest muscles and clavicles have all been removed. Medial access of the right subclavian vein or inferior access of the right internal jugular vein presents a higher risk for pneumothorax to occur compared with left-sided venous access. (From Acland RD. *Acland's Atlas of Human Anatomy, 6 DVD Set*. Philadelphia: Lippincott Williams & Wilkins; 2003.)

joins the superior vena cava, should prompt the physician to curve the sheath and advance it with caution over the guidewire.

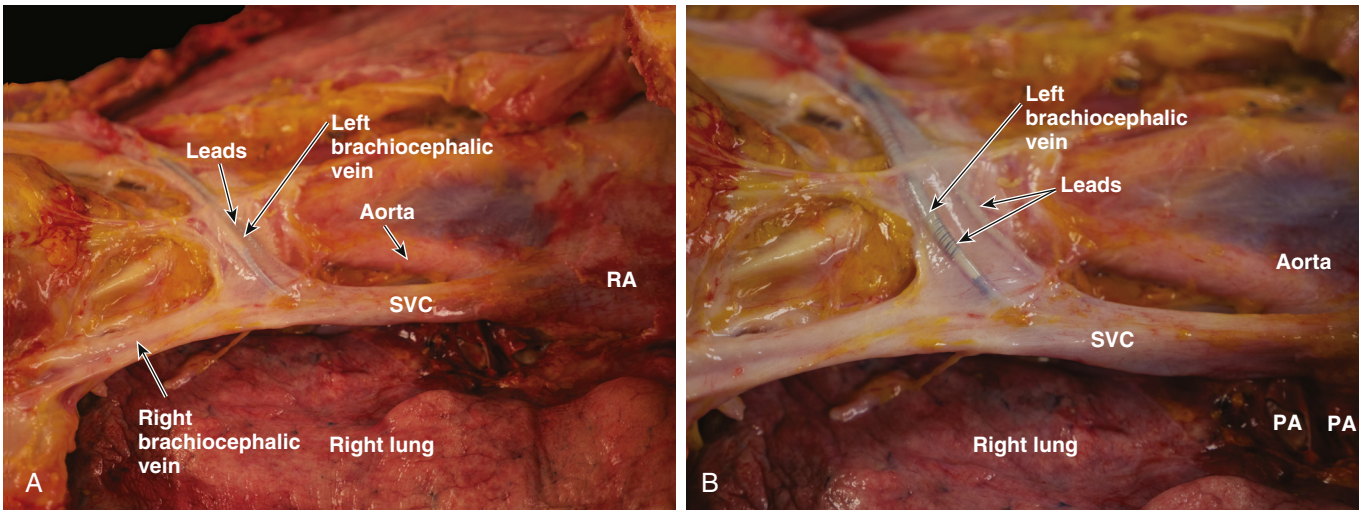
Venous Anomalies

A persistent left superior vena cava is the most common congenital venous anomaly of the mediastinum, occurring in 0.5% of the population (Fig. 2.47). Most often, when a left superior vena cava is present, a normal right superior cava is also present. The left superior vena cava drains into the coronary sinus, and as a consequence, there is no left-sided connection from the left axillary/subclavian vein to the right atrium other than retrograde through the coronary sinus os. The presence of a left superior vena cava does not preclude placement of a pacemaker or ICD lead, and techniques have been developed to improve success (see Chapters 8 and 11).

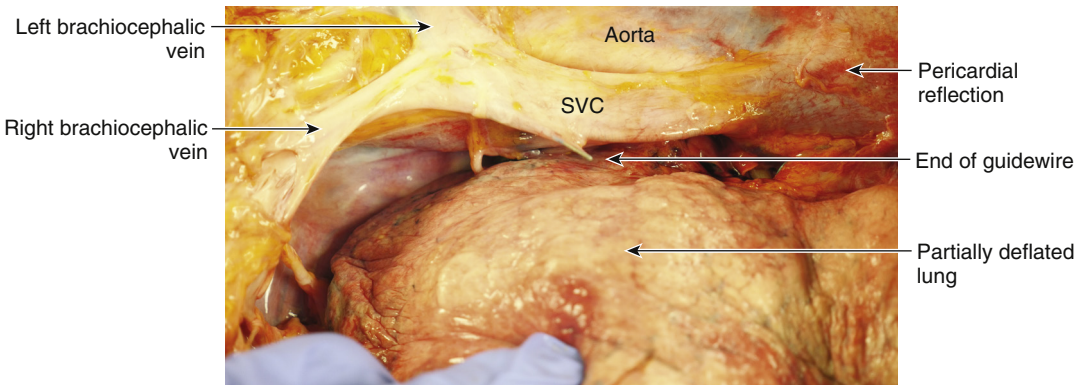
Heart

The heart is located in the middle mediastinum within the confines of the pericardial sac.[8–10] The three-dimensional orientation of the heart in the thorax is complex, and the anatomic designations are sometimes confusing when applied to the in vivo and clinical correlates. The focus of this section will be on anatomy particularly relevant to the implantation of transvenous pacemakers and implantable defibrillators.

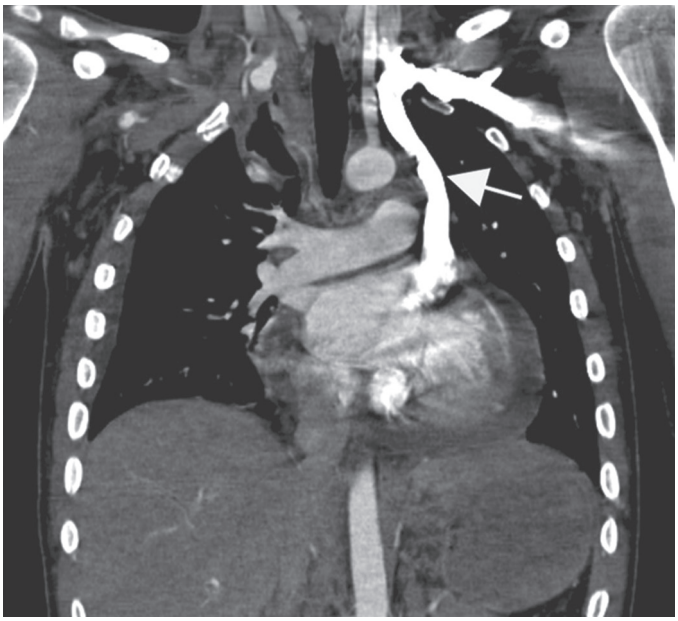
The heart is positioned in the thorax obliquely with the base facing posteriorly and rightward and the apex anteriorly and leftward. The right atrium is anterior and inferior to the left atrium. The right ventricle makes up the major portion of the anterior ventricular surface, except for a small strip of left ventricle that includes the apex. Both atria are located to the right of and posterior to their respective ventricles (Figs. 2.48 and 2.49).



• **Fig. 2.45** (A) Cadaveric dissection illustrating pacemaker leads and a defibrillator lead coursing through the left brachiocephalic (innominate) vein into the superior vena cava. (B) Closer view showing the leads visible through the left brachiocephalic vein and the relationship of the superior vena cava, aorta, and right lung. *PA,* pulmonary artery; *RA*, right atrium; *SVC*, superior vena cava.



• **Fig. 2.46** Cadaveric dissection simulating a perforation of the lateral wall of the superior vena cava during wire, sheath, or lead placement. *PA*, pulmonary artery; *SVC*, superior vena cava.



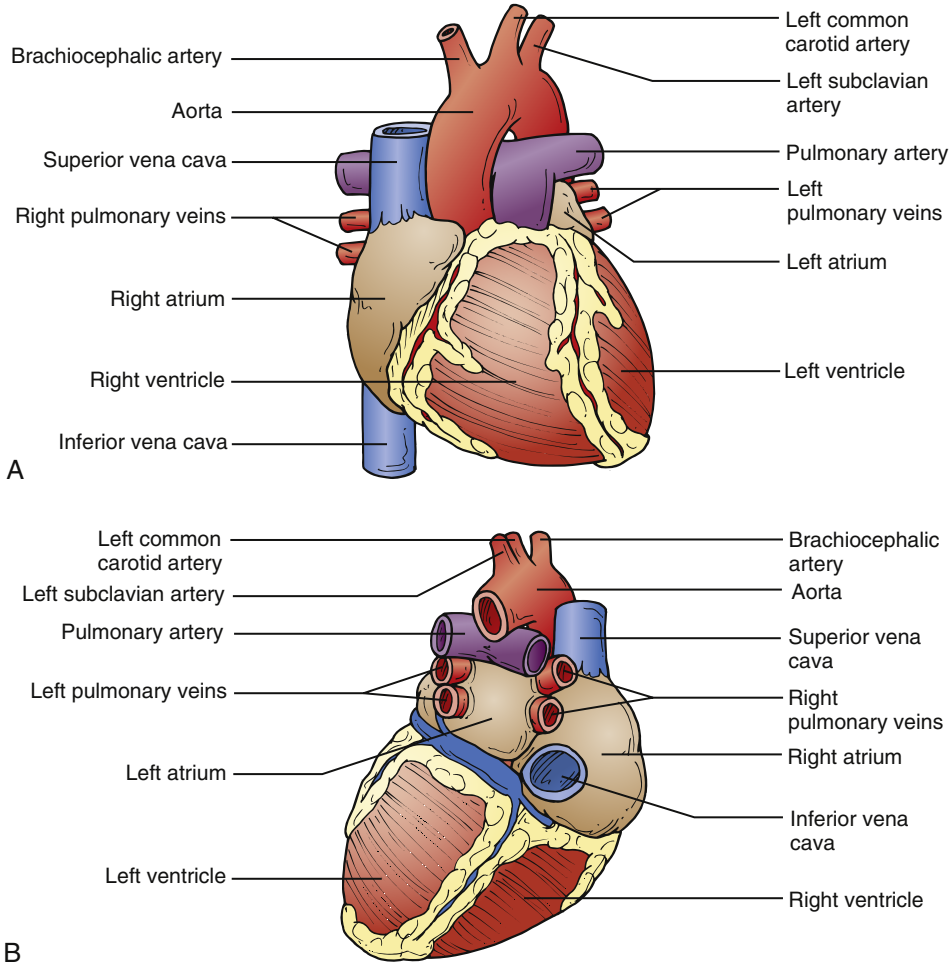
• **Fig. 2.47** Computed tomographic contrast image of a persistent left superior vena cava. (From Goyal SK, Punnam SR, Verma G, Ruberg FL. Persistent left superior vena cava: a case report and review of literature. *Cardiovasc Ultrasound.* 2008;6:50.)

Clinical Correlations

- Physicians who implant pacemakers and defibrillators currently rely on fluoroscopic imaging to guide lead placement and postoperative chest x-rays to confirm lead location. This imaging modality allows identification of the gross cardiac anatomic structures (Fig. 2.50). The desire to reduce radiation risk both to the physician and to the patient has led to the greater use of nonthorascopic imaging using three-dimensional electromagnetic imaging technology.

Right Atrium

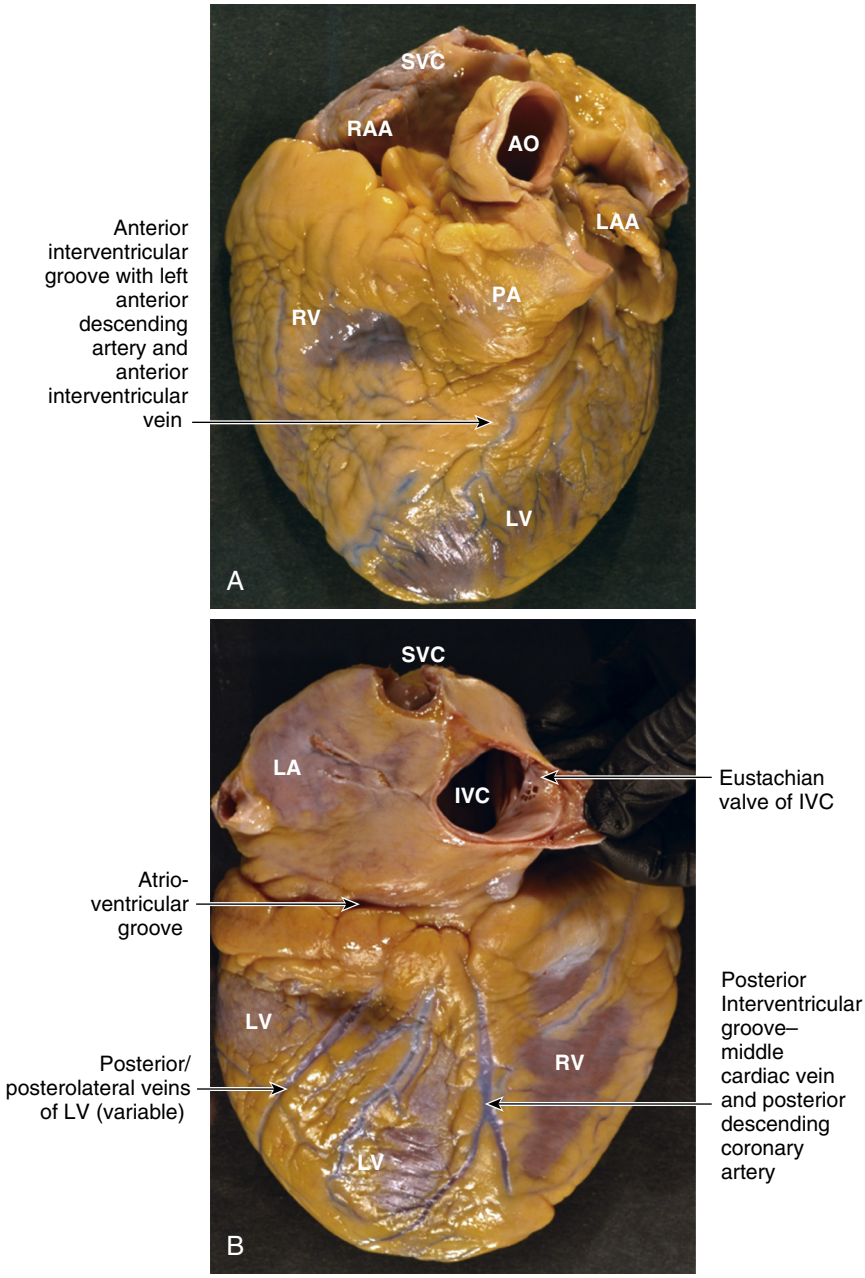
Each atrium consists of an appendage (or auricle), a venous portion, and an atrioventricular vestibule.[8,11–13] The right atrial appendage makes up the superior right border of the heart. It is larger in volume than the left atrial appendage, has a broad-based triangular appearance, and is separated externally from the right ventricle by the atrioventricular groove, which usually contains the right coronary artery. The external sulcus terminalis or terminal groove is located at the junction of the right atrial appendage with the venous portion of the atrium and marks the location of the subepicardial sinus node. Internally, the smooth-walled atrium, embryologically



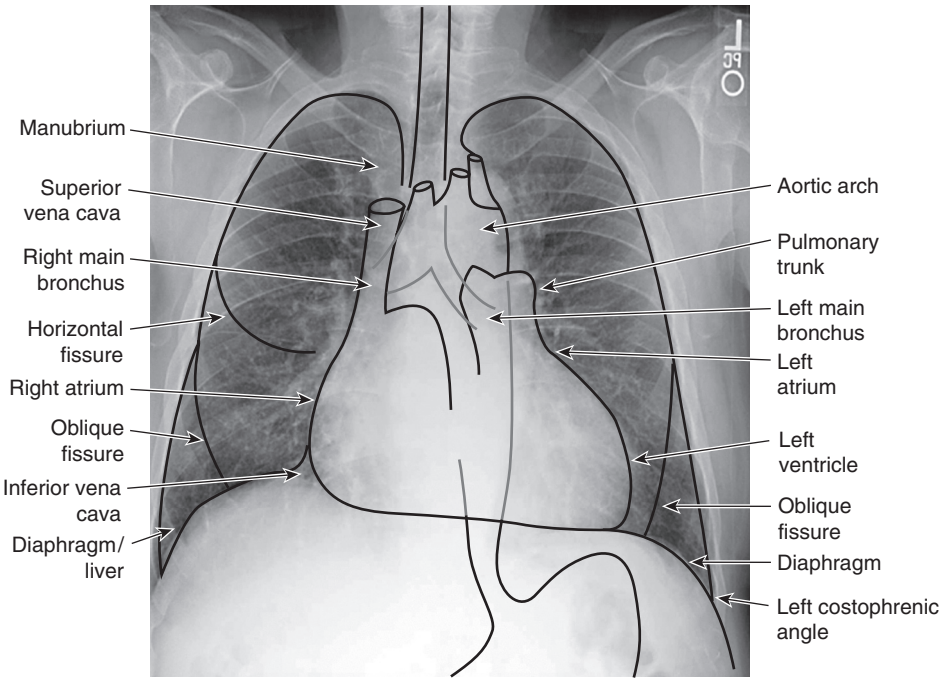
• **Fig. 2.48** External anatomy of the (A) anterior and (B) posterior views of the heart.

derived from the sinus venosus, receives the superior and inferior vena cavae and coronary sinus. The trabeculated appendage, derived from the embryonic atrium, is covered by the pectinate muscles, which extend circumferentially around the atrioventricular vestibule after originating at the crista terminalis (Video 2.5). Also called the terminal crest, this muscular ridge corresponds to the external sulcus terminalis and separates the smooth and trabeculated atrium. Extending between the superior and inferior vena cavae, it is more prominent at the superior vena cava orifice and nearly indistinct at the right side of the inferior vena cava opening (Figs. 2.51 and 2.52). The atrial wall normally measures 1 to 2 mm in thickness, and the appendage wall is so thin between the pectinate muscles that it appears translucent (Fig. 2.53).

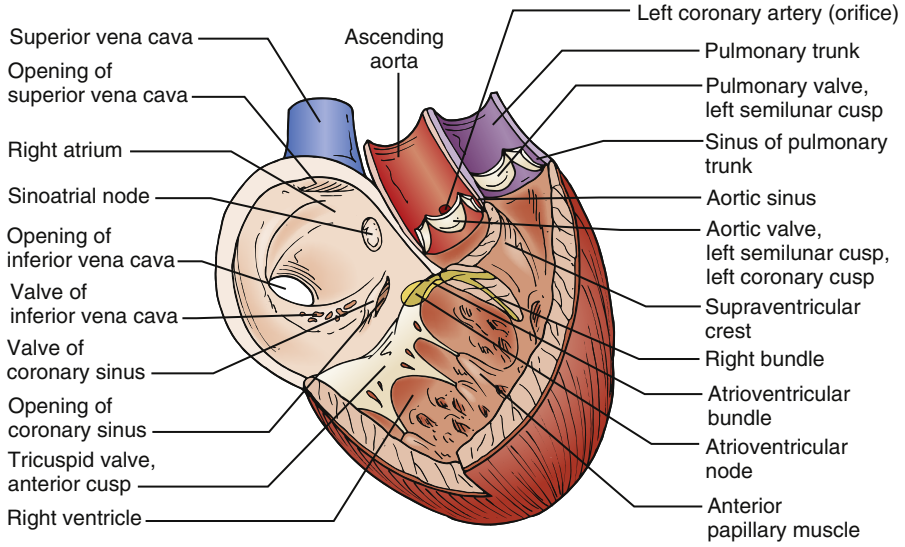
The opening of the superior vena cava into the upper portion of the right atrium is not internally well delineated and has no valve. In contrast, the atrial orifice of the inferior vena cava is usually demarcated by the Eustachian valve (or valve of the inferior



• **Fig. 2.49** (A) Anterior view of a formalin-fixed whole heart. The anterior interventricular groove is the location of the left anterior descending artery and anterior interventricular vein. (B) Corresponding posterior view. The posterior interventricular groove is the location of the posterior descending coronary artery and the middle cardiac vein. Note the focally fenestrated Eustachian valve of the inferior vena cava. *AO*, aorta; *IVC*, inferior vena cava; *LA*, left atrium; *LAA*, left atrial appendage; *LV*, left ventricle; *PA*, pulmonary artery; *RA,* right atrium; *RAA*, right atrial appendage; *RV*, right ventricle; *SVC*, superior vena cava.



• **Fig. 2.50** Posterior-anterior chest radiograph showing the shadow of the cardiac silhouette with key cardiac anatomic structures labeled.

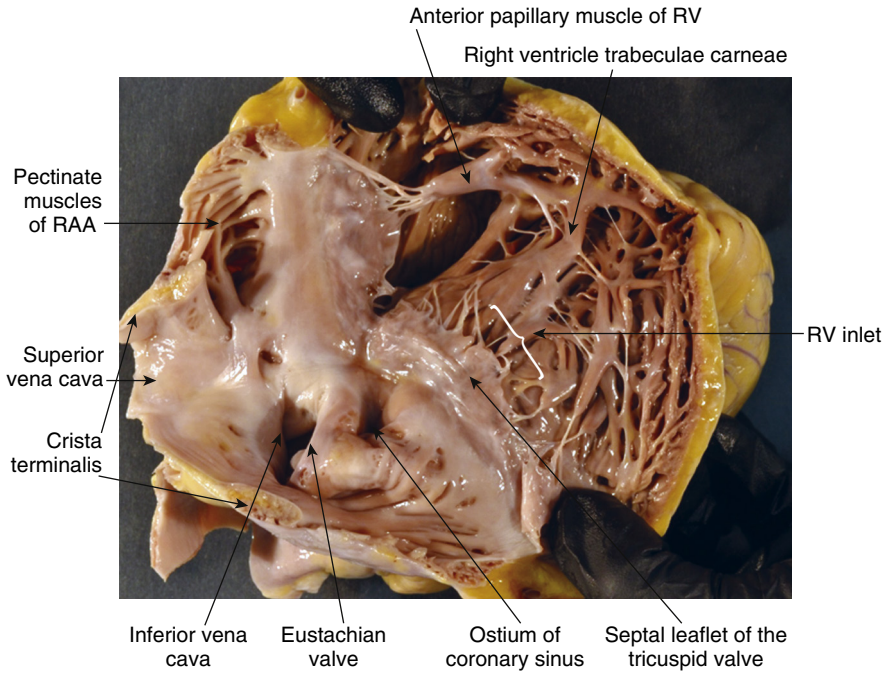


• **Fig. 2.51** Interior of the heart illustrating relationship of structures within the right atrium. (From Waschke J, Paulsen F. *Sobotta Atlas of Human Anatomy*, ed 15. Philadelphia: Elsevier; 2013.)

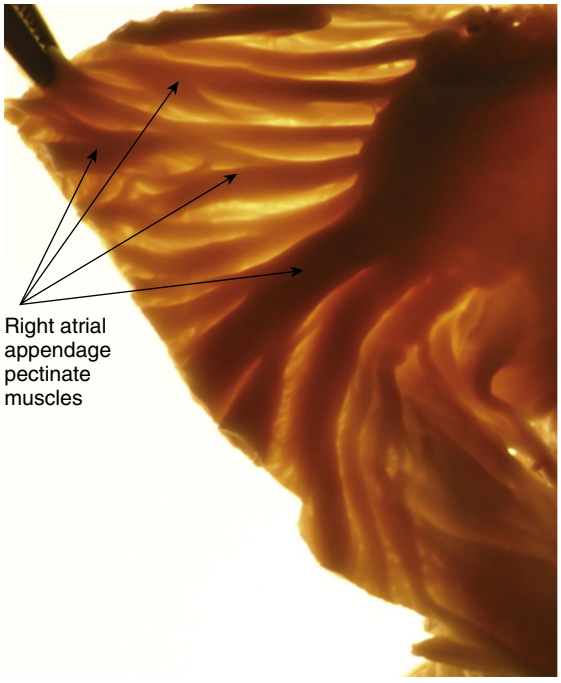
vena cava), located at the anterior border of the inferior vena cava ostium, an embryologic remnant that functioned in intrauterine life to direct blood from the inferior vena cava through the foramen ovale into the left atrium (Figs. 2.51, 2.52, and 2.54 through 2.56). The Eustachian valve may be absent or variable in size, and when fenestrated and lacelike, it is termed a Chiari network, which can also occur in the thebesian valve. A large Chiari network can prolapse through the tricuspid valve and become a risk for pacemaker and defibrillator leads to become entangled inadvertently.

The coronary sinus orifice is located just anterior to the medial extent of the Eustachian valve and is variably guarded by a valvelike crescentic fold termed the Thebesian valve. Like the Eustachian valve, it can have a variable configuration and may be fenestrated with risk of entanglement (Fig. 2.57). The Eustachian ligament is a fold between the Eustachian valve and the Thebesian valve, and is continuous with the tendon of Todaro, a fibrous structure that extends to the central fibrous body of the cardiac annular structure. These structures are part of the triangle of Koch, which marks the location of the atrioventricular (AV) node and is described further in the Conduction System section (see Figs. 2.51, 2.52, 2.54, 2.55, and 2.56).

The interatrial septum is located on the medial aspect of the right atrium and contains a central ovoid depression termed the fossa ovalis, surrounded by a muscular ridge called the

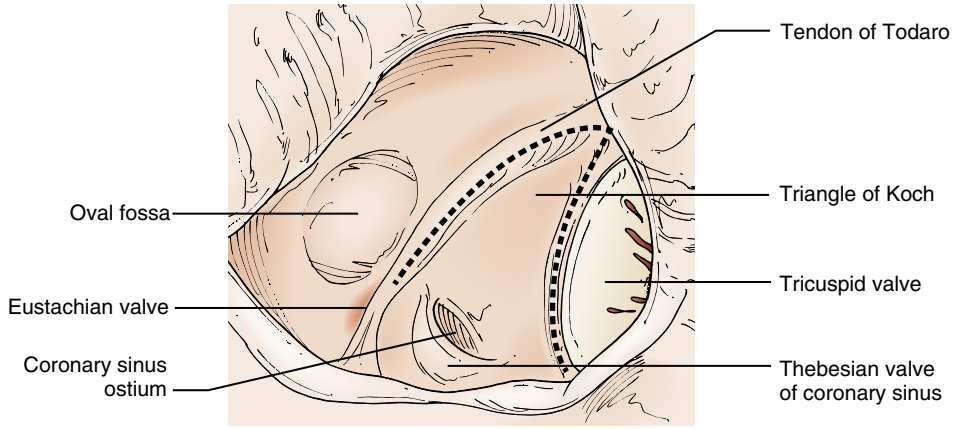


• **Fig. 2.52** Formalin-fixed heart demonstrating key internal anatomy of the right atrium and right ventricle. The right ventricular inlet is trabeculated, and the trabeculation becomes coarser toward the apex, in the apical trabecular portion. The crista terminalis identifies the location of the sinoatrial node. *RAA*, right atrial appendage; *RV*, right ventricle; *SVC*, superior vena cava.

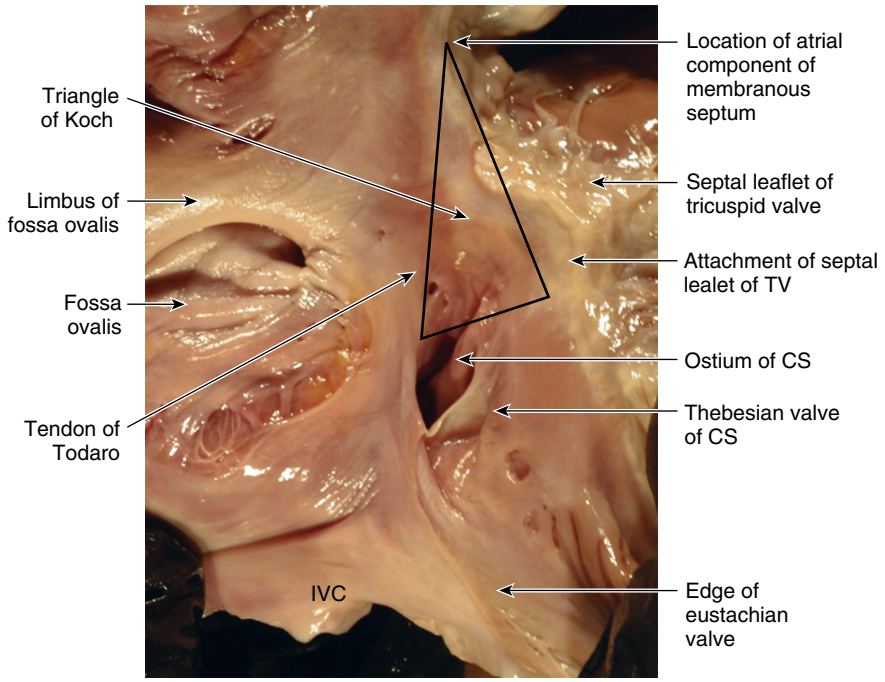


• **Fig. 2.53** Backlit formalin-fixed section from the right atrial appendage. The musculature of the right atrial appendage is arranged in bundles termed the pectinate muscles. The atrial muscle tissue between the pectinate muscles is very thin, as demonstrated here by transillumination of the right atrial appendage.

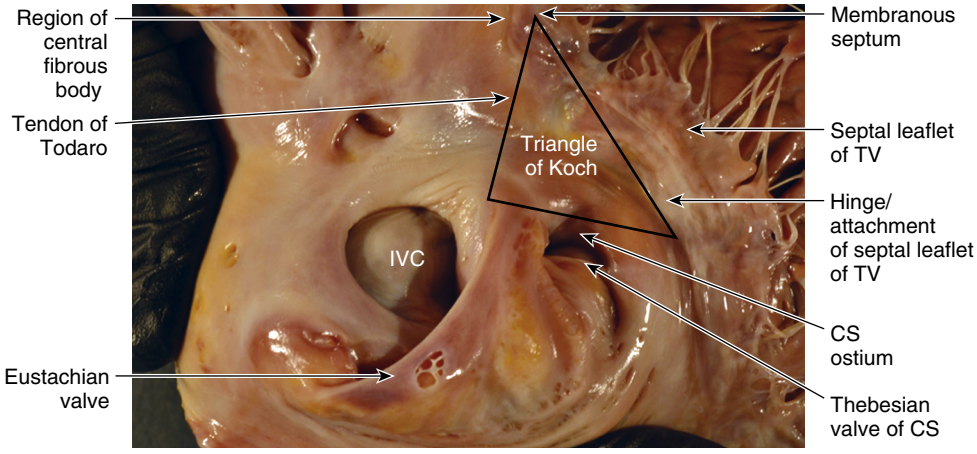
limbus fossa ovalis (see Figs. 2.55 and 2.57). In approximately 25% of adults, there is incomplete fusion of the limbus with the fossa, resulting in a patent foramen ovale. In most cases, there is functional closure of the foramen because of the pressure differences between the left and right atria, but in some persons, incomplete closure results in a secundum atrial septal defect (Fig. 2.58). Depending on the size of the patent foramen ovale or atrial septal defect and clinical indication, closure devices can be implanted percutaneously to address this problem (Video 2.6). The smooth-walled atrioventricular vestibule extends to the tricuspid annulus, the fibrous structure to which the tricuspid valve leaflets are attached.



• **Fig. 2.54** Structures that define the triangle of Koch, which is bounded by the tricuspid valve septal leaflet attachment, the ostium of the coronary sinus, and the tendon of Todaro. Noted is the Thebesian valve of the coronary sinus, guarding the coronary sinus ostium. (From Anderson RH, Cook AC. The structure and components of the atrial chambers. Europace. 2007;9:vi3–vi9.)



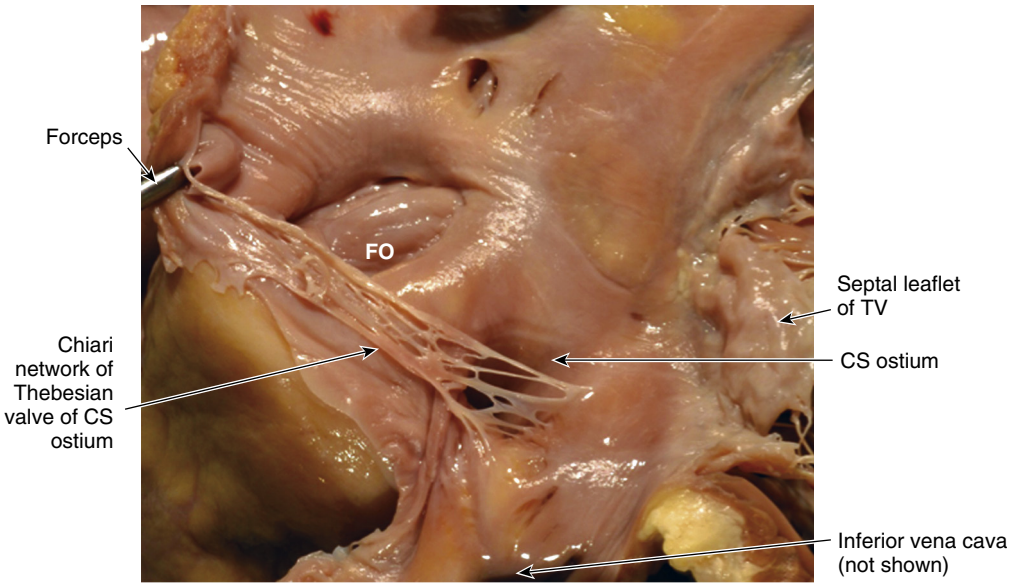
• **Fig. 2.55** Formalin-fixed section of the right atrium demonstrating the septal structures and the triangle of Koch (*black lines*). *CS*, coronary sinus; *IVC*, inferior vena cava; *TV*, tricuspid valve of coronary sinus.



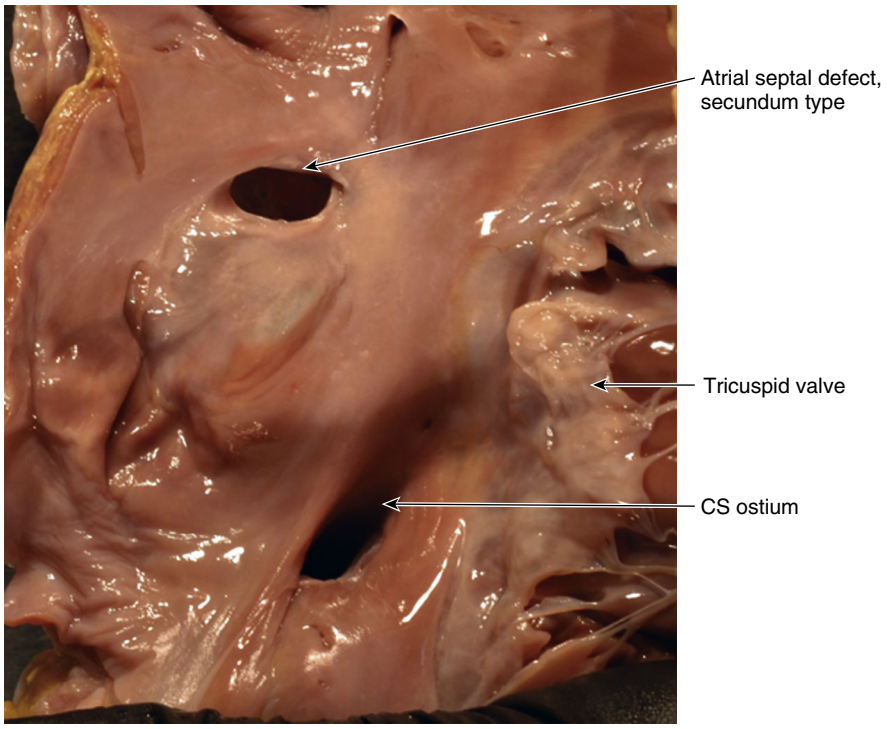
• **Fig. 2.56** Formalin-fixed section of the right atrium demonstrating the continuity between the free border of the eustachian valve and the tendon of Todaro. The tendon of Todaro courses through the Eustachian ridge to attach to the central fibrous body, which is the location of the penetrating bundle of His. The triangle of Koch is outlined (*black lines*). The anterior border is the attachment of the septal leaflet of the tricuspid valve, the inferior border is the ostium of the coronary sinus and the vestibule just anterior to it, and the superior border is the tendon of Todaro. The triangle of Koch contains the atrioventricular node and its fast and slow pathway inputs. *CS*, coronary sinus; *TV*, tricuspid valve.

Clinical Correlations

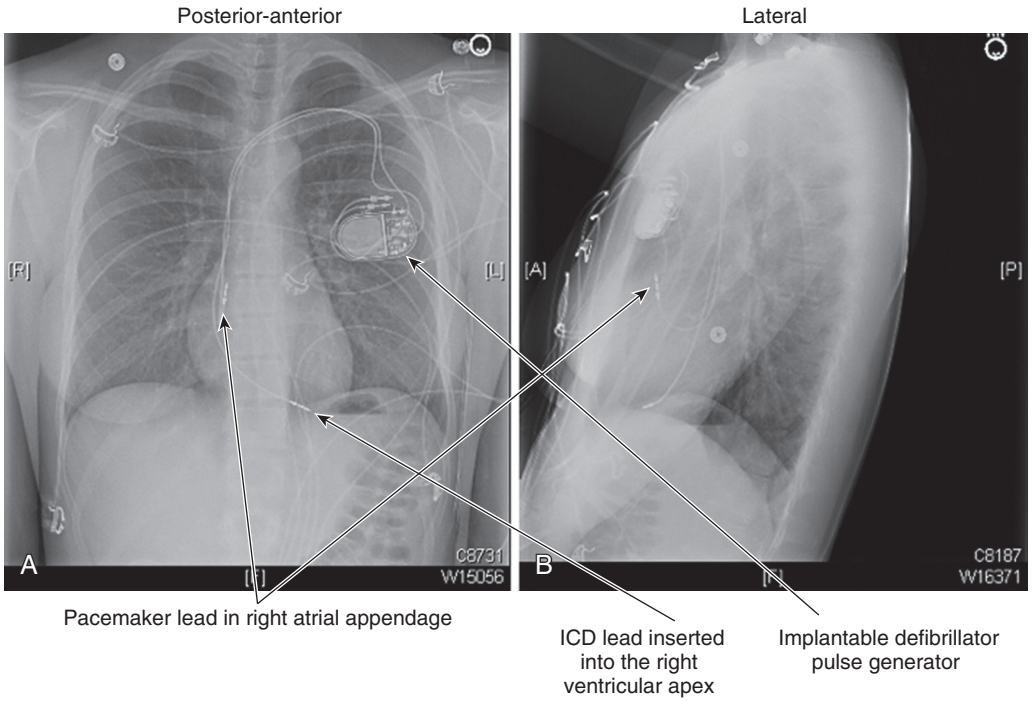
- The right atrial appendage has been the preferred location for placement of the right atrial pacing lead (Fig. 2.59). The reason for this is that the pouchlike structure provides a stable position for the lead and reduces the risk of dislodgement or stimulation of the phrenic nerve (Video 2.7). The P-wave size and pacing thresholds are generally good in this position. Placement in this location also likely reduces the risk of perforation because the appendage wall is thicker than the right atrial free wall. However, perforations can occur regardless of placement (Fig. 2.60).
- The right atrial appendage may not be present after cardiothoracic surgery, because after removal of the venous cannula used during cardiac bypass, the pursestring suture around the cannula is tied following decannulation. This results in necrosis of the right atrial appendage, leaving a small indentation at the orifice of the appendage. A lead can be placed successfully in this remnant with stability, but scar tissue in this location may result in poor electrical parameters, necessitating a search for an alternate site for lead placement.
- Placing the lead in atrial sites other than the right atrial appendage can provide lead stability with good electrical



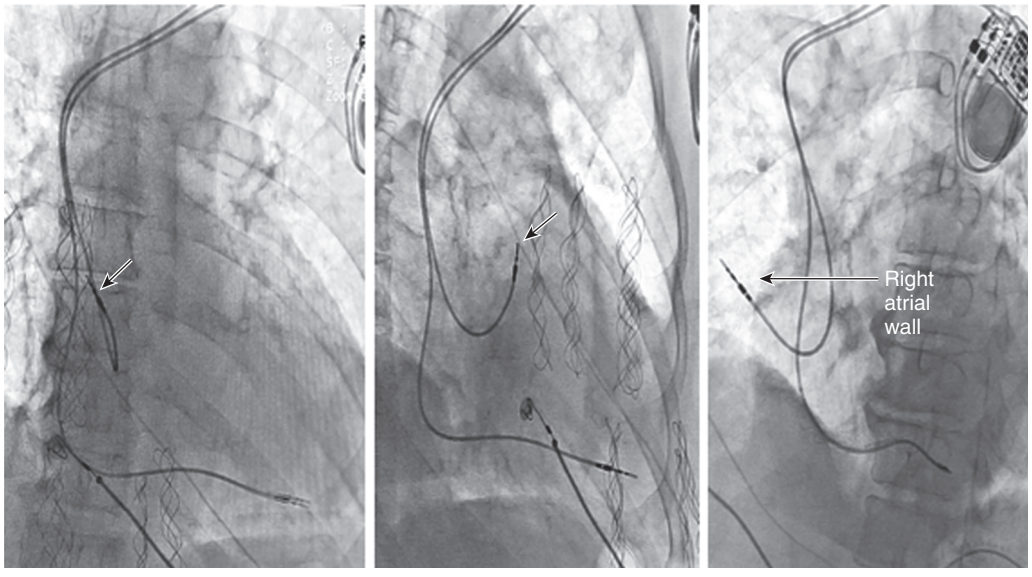
• **Fig. 2.57** Formalin-fixed section of the right atrium demonstrating a Chiari network extending from the Thebesian valve of the coronary sinus. The network is held across the ostium of the coronary sinus with forceps for demonstration. *CS*, coronary sinus; *FO*, fossa ovalis; *TV*, tricuspid valve.



• **Fig. 2.58** Formalin-fixed section of the right atrial septum demonstrating an atrial septal defect, secundum type. *CS*, coronary sinus.

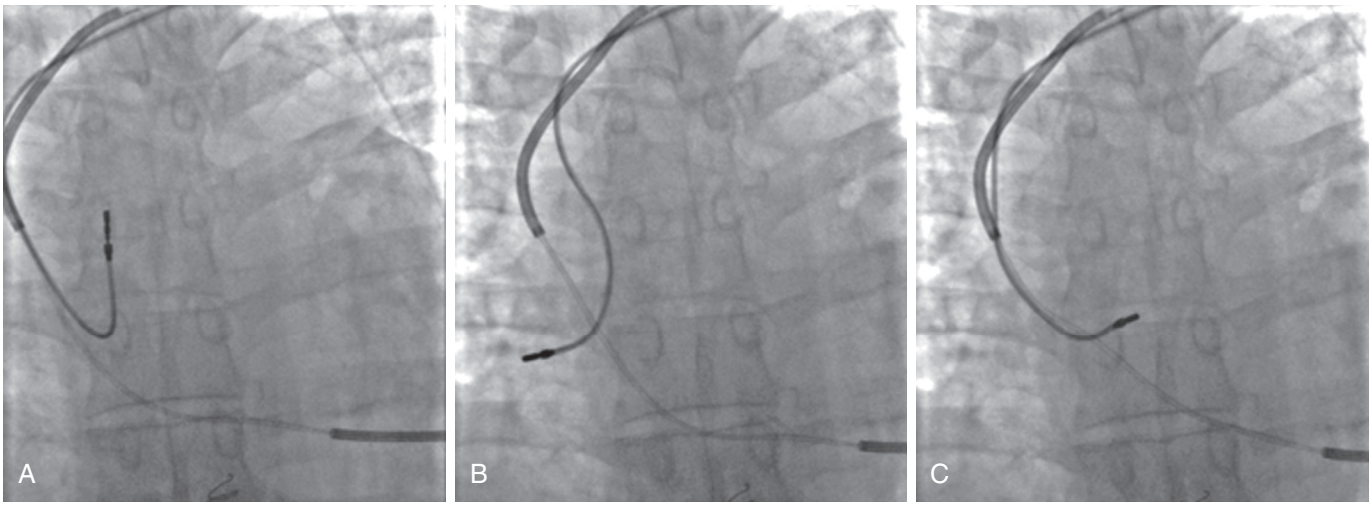


• **Fig. 2.59** (A) Posterior-anterior and (B) lateral chest radiographs with implanted dual-chamber implantable cardioverter defibrillator. The atrial pacing lead is positioned in the right atrial appendage.

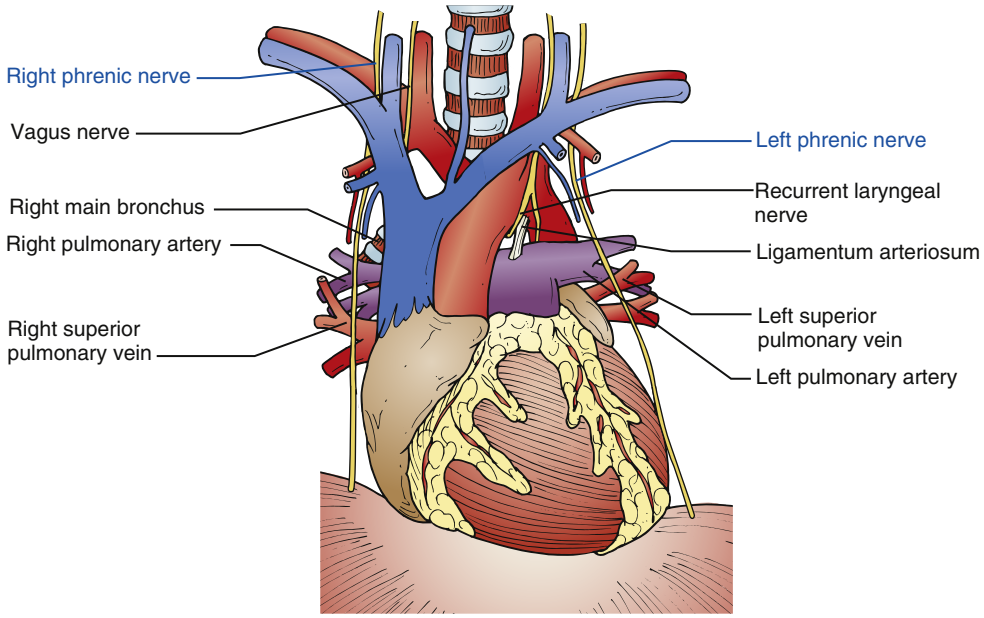


• **Fig. 2.60** Fluoroscopic images obtained during an atrial pacing lead reposition procedure, indicated for malfunction identified during interrogation of a dual-chamber pacemaker 1 week after implantation. The right atrial lead was implanted into the right atrial appendage. At follow-up, the atrial lead was found to have absence of pacing and sensing. A posterior-anterior chest radiograph was not diagnostic for a perforation or dislodgement of the lead. However, by using multiple fluoroscopic images, the lead can clearly be observed beyond the atrial silhouette on the left anterior oblique image. The short arrows show the position of the atrial lead.

parameters (Fig. 2.61 and Video 2.8). However, there are several important caveats. When placing the lead in a lateral position, the possibility of phrenic nerve stimulation must be considered. The phrenic nerves originate in the neck from C3 to C5 and then course on the outer surface of the fibrous pericardial sac between the lung and the heart to innervate the diaphragm (Fig. 2.62). The right phrenic nerve passes along the right atrium and right upper pulmonary vein, while the left phrenic nerve is adjacent to the left atrium and ventricle. Thus, pacing should be tested at high outputs to be certain



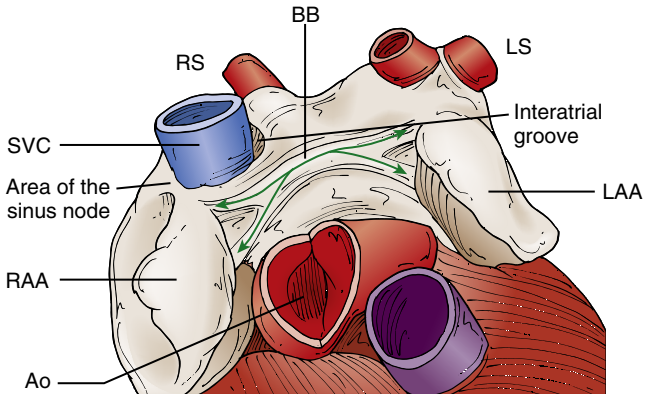
• **Fig. 2.61** Anterior-posterior fluoroscopic images demonstrating the placement of an atrial pacing lead into alternative atrial sites. (A) Standard position in the right atrial appendage. (B) Placement in the posterolateral wall. (C) Placement of the lead in the right atrial septum.



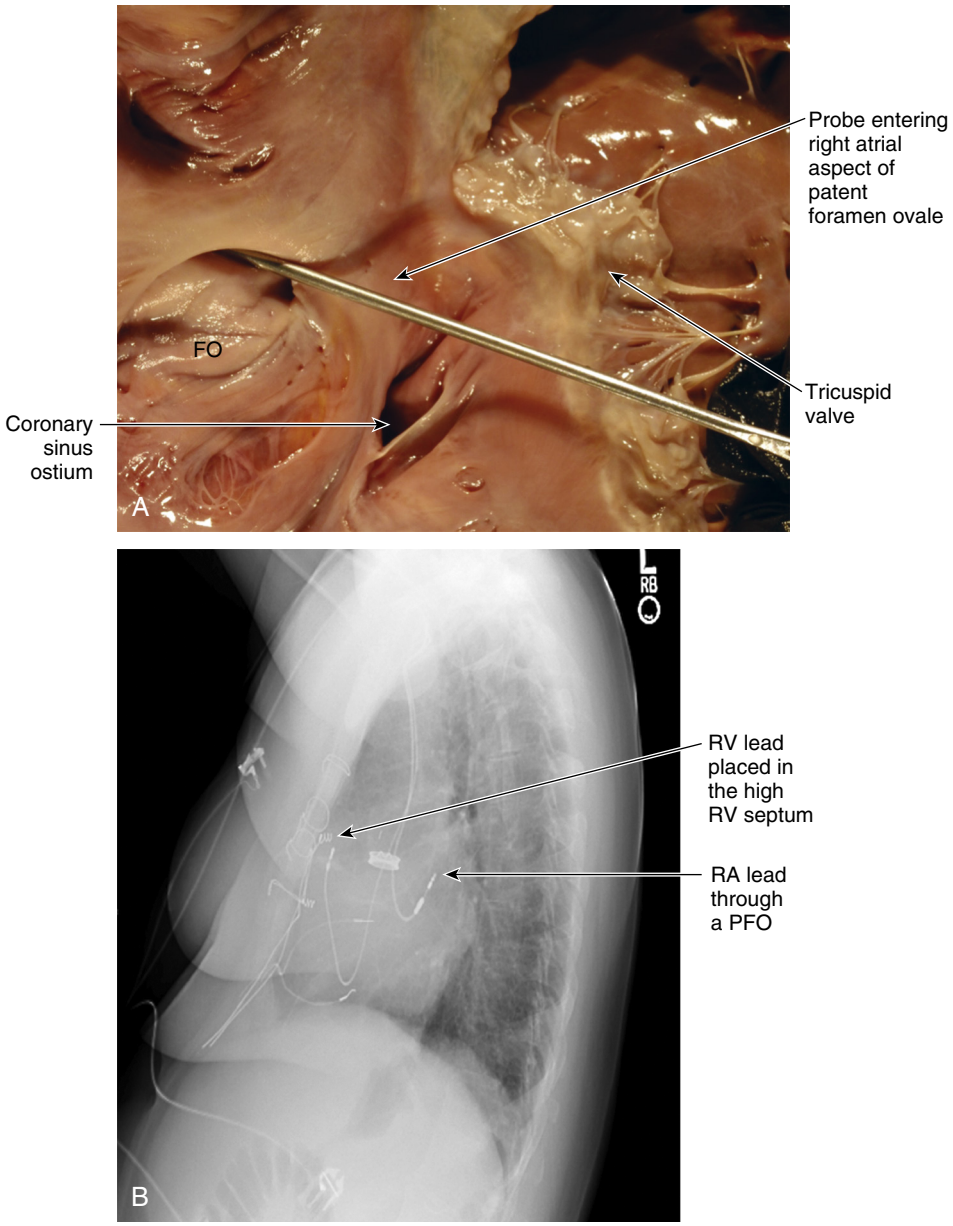
• **Fig. 2.62** Course of the right and left phrenic nerve. The nerve courses in the fibrous pericardium to each hemidiaphragm.

that phrenic nerve stimulation does not occur when placing the atrial lead in the lateral atrial wall. If it does, an alternate lead location should be sought, such as a posterolateral or septal position, if the appendage is not acceptable.

- More recently, there has been interest in septal positioning of the right atrial lead.[14–16] This approach takes advantage of the right-to-left atrial fast conducting fibers (Bachmann's bundle). Bachmann's bundle represents the anterior–superior conduction above the fossa ovalis, while muscular margins below the fossa provide posterior pathways[8,11–13] (Fig. 2.63). The area of the septum primum also has conducting fibers to the left atrium. Using a septal position has been shown in some studies to provide shorter PR intervals and P-wave duration. This may have the advantage of increasing the opportunity for native conduction to the ventricle, thus avoiding unnecessary right ventricular (RV) pacing. With more rapid conduction to the left atrium, there is less of a chance for simultaneous AV activation during cardiac resynchronization therapy (CRT) pacing, thus allowing more advantage of CRT. Furthermore, it is postulated that septal pacing may reduce atrial fibrillation. *Targeting specific septal sites can be challenging,* and care must be taken to avoid inadvertent placement of the lead through a patent foramen ovale (Fig. 2.64). Additionally, the smooth wall of the septum and lead angle required to reach this area may contribute to a higher risk of dislodgement.



• **Fig. 2.63** Location of Bachmann's bundle (BB) as it crosses the interatrial septum. The view is of the anterior atrium with the aorta pulled forward. The area of the sinus node is noted, as is the interatrial groove. *Ao*, aorta; *LAA*, left atrial appendage; *LS and RS*, left superior and right superior pulmonary veins; *RAA*, right atrial appendage; *SCV*, superior caval vein (superior vena cava). (From Ho SY, Ernst S. *Anatomy for Cardiac Electrophysiologists: A Practical Handbook.* Minneapolis, MN: Cardiotext Publishing; 2012:18.)



• **Fig. 2.64** (A) Right atrium with a patent foramen ovale. A probe is shown entering the patent foramen ovale. (B) Lateral chest radiograph showing a posteriorly directed right atrial lead inadvertently placed through a patent foramen ovale into the left atrium. The right ventricular lead is located in the high right ventricular septum. *PFO*, patent foramen ovale; *RA*, right atrial; *RV*, right ventricular; *TV*, tricuspid valve.

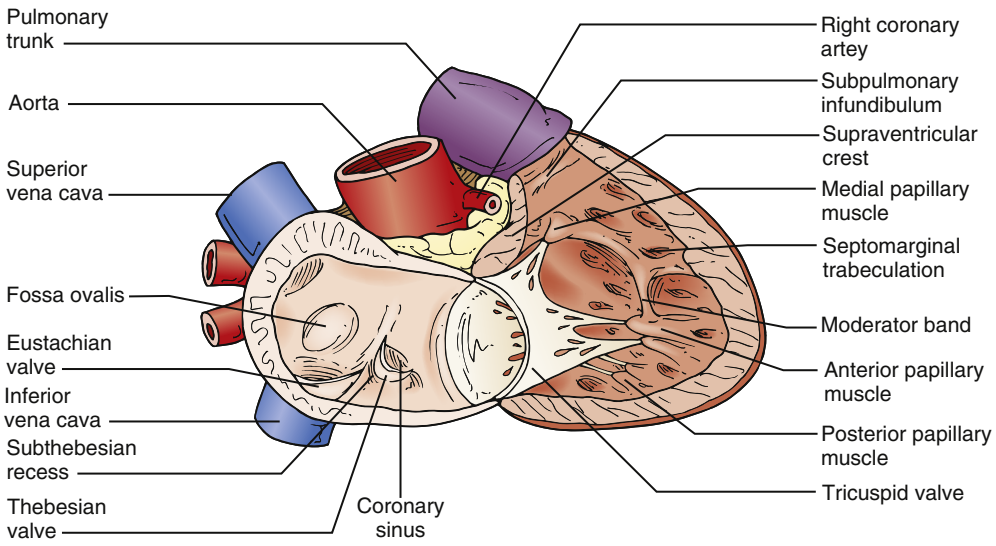
Right Ventricle and Tricuspid Valve

The right ventricle makes up the major portion of the anterior surface and inferior border of the heart, and like the left ventricle, the RV cavity has three major anatomic components: the inlet, the apical-trabecular portion, and the outlet[8–10] (Fig. 2.65). The inlet supports the tricuspid valve, extending from the annulus of the tricuspid valve orifice to the attachments of the chordae tendineae at the tips of the papillary muscles. The inlet is somewhat trabeculated, but the trabeculation becomes progressively coarser toward the apex. This coarse trabeculation is a characteristic anatomic feature differentiating the anatomic right and left ventricles (see Fig. 2.52). The right ventricular outflow tract, termed the conus arteriosus or infundibulum, is relatively smooth walled and supports the pulmonic valve, which has three semilunar leaflets, attached partly to the right ventricular wall and partly to the origin of the pulmonary artery trunk. The trabeculated inlet and smooth outlet are separated by a thick muscular ridge called the supraventricular crest (crista supraventricularis). The supraventricular crest is encompassed by the two arms of the Y-shaped septomarginal trabeculation, a broad, muscular trabecula that extends inferiorly along the septum to become the moderator band, which connects to the base of the anterior papillary muscle. The moderator band is of variable length and thickness, and is occasionally absent. The septoparietal trabeculations extend from the anterior moderator band onto the parietal right ventricular wall (Figs. 2.66 and 2.67).

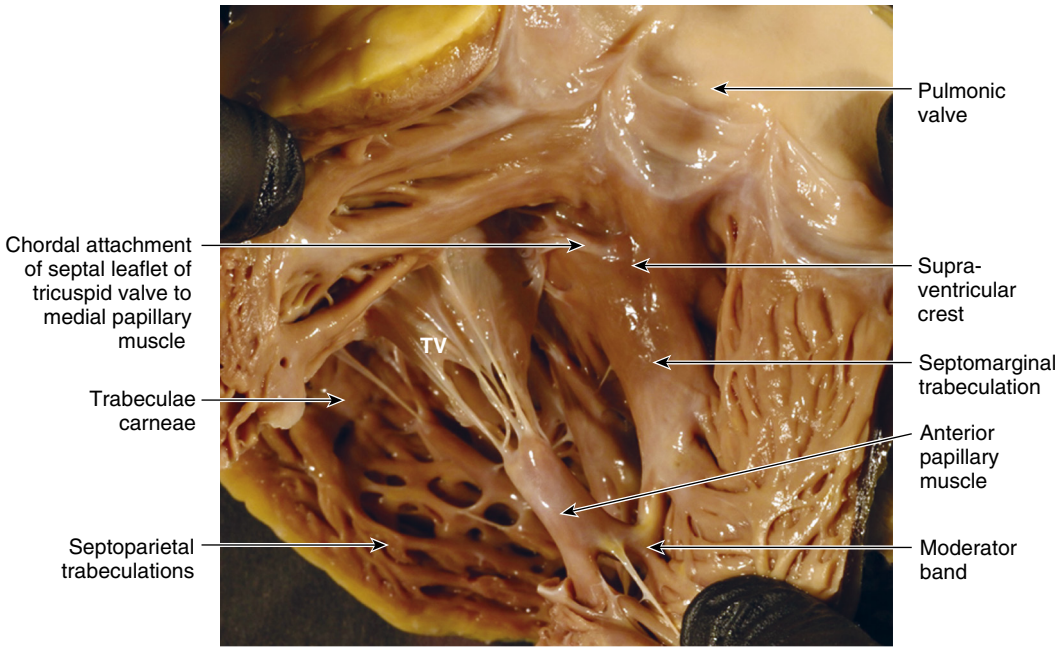
The muscular ventricular septum is continuous superiorly with the ventricular portion of the membranous septum, which is separated from the atrial portion of the membranous septum by the attachment of the septal leaflet of the tricuspid valve. The tricuspid valve has three leaflets, which are anchored by chordae tendineae to the papillary muscles (see Fig. 2.67). Unique to the tricuspid valve is the direct attachment of chordae tendineae from the septal leaflet to the upper muscular ventricular septum just below the ventricular portion of the membranous septum (in addition to its attachment to the posterior papillary muscle). This attachment is located just above the septomarginal trabeculation and considered to be the rudimentary medial papillary muscle, which may be no more than a tendinous patch (see Figs. 2.66 and 2.67). This unique configuration marks the site where the right bundle branch emerges from the septum to traverse the septomarginal trabeculation. The larger and more prominent anterior papillary muscle receives chordae from the anterior and posterior tricuspid leaflets and also has a prominent and consistent connection to the septum by the moderator band, which extends from the inferior aspect of the septomarginal trabeculation into the anterior papillary muscle and from there into the parietal wall of the right ventricle, carrying a major fascicle of the right bundle branch (see Fig. 2.66). The trabeculated appearance of the ventricular wall results from the trabeculae carneae, which consist of irregular muscular ridges and protrusions (see Figs. 2.52 and 2.66).

Clinical Correlations

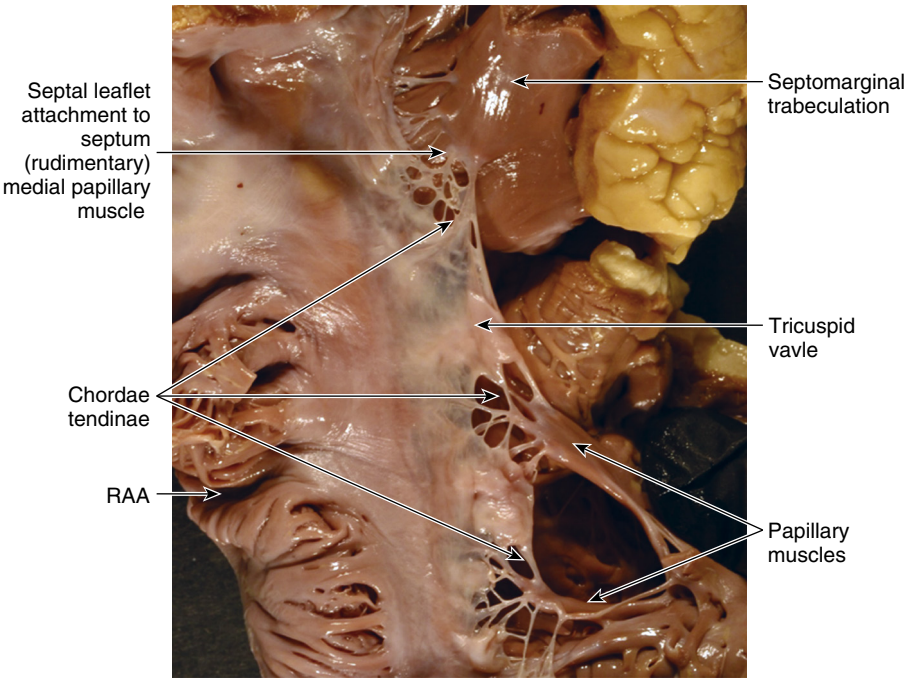
- Right ventricular lead placement is often dictated by the course of the lead as it passes through the chordae tendineae and around the papillary muscles (Video 2.9 and Fig. 2.68). The muscular trabeculae are used to anchor distal lead tips for passive fixation (tined leads) but may also affect the position of active (screw-in) leads. In heavily trabeculated RV chambers, the lead may inadvertently engage the myocardium of the moderator band or be placed into thick trabeculae. Often this results in lead instability and electrical parameters that are less than ideal (Video 2.10). The fluoroscopic or x-ray appearance may be a clue as the lead will not be sufficiently advanced to the true apical position (Fig. 2.69). In all



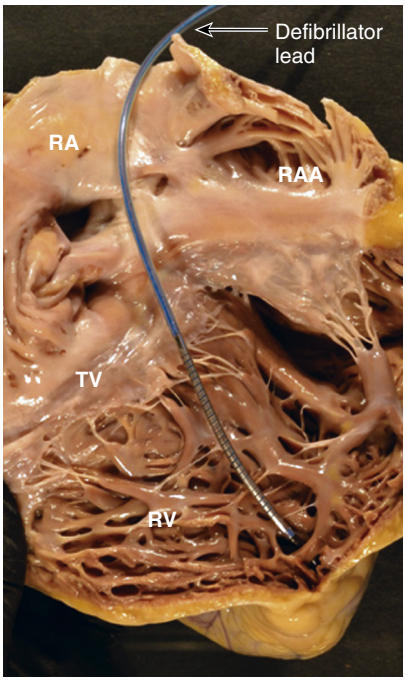
• **Fig. 2.65** Internal structures of the right atrium and the right ventricle.



• **Fig. 2.66** Key internal structures of the right ventricular outflow tract are shown in this formalin-fixed specimen. The chordal attachment of the septal leaflet of the tricuspid valve attaches to a rudimentary medial papillary muscle, seen here as an endocardial fibrous patch just above the septomarginal trabeculation. This marks the location of the His bundle on the right septum. The septomarginal trabeculation is a broad muscle bundle with the superior aspect marked by the supraventricular crest, a broad band separating the trabeculated right ventricular inlet (see Fig. 2.52) from the relatively smooth right ventricular outflow tract. Branches of the conducting system extend from the septomarginal trabeculation into the moderator band, the anterior papillary muscle, and the septoparietal trabeculations on the free wall of the right ventricle.



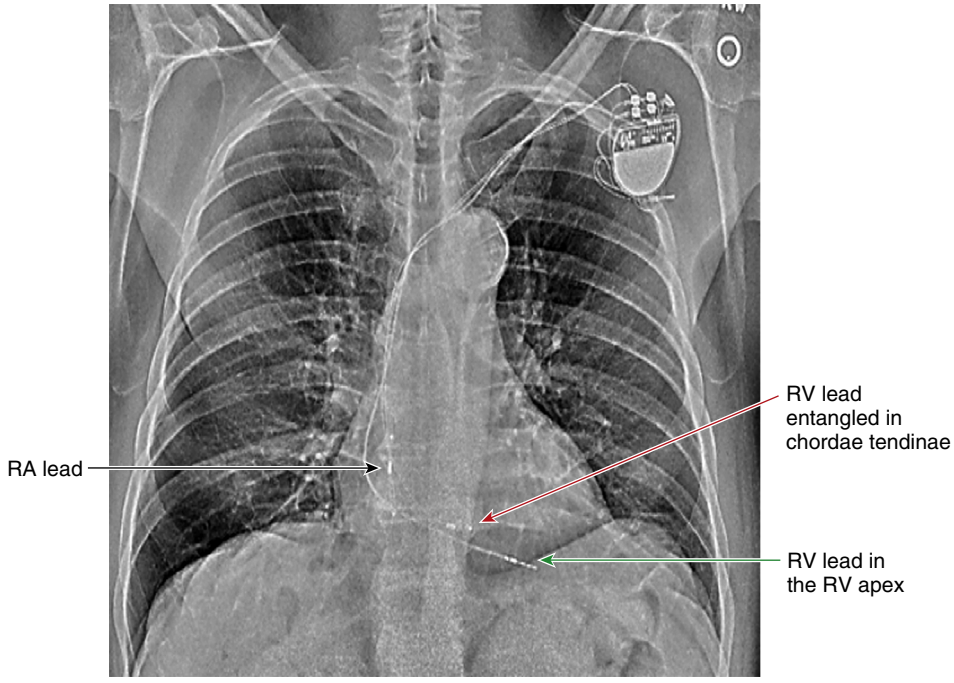
• **Fig. 2.67** Formalin-fixed segment of right atrium and ventricle showing the septal leaflet of the tricuspid valve and adjacent structures. The chordae tendineae of the septal leaflet of the tricuspid valve attach directly to the septum via a rudimentary medial papillary muscle, seen here as an endocardial fibrous patch just above the septomarginal trabeculation (incompletely seen). *RAA*, right atrial appendage.



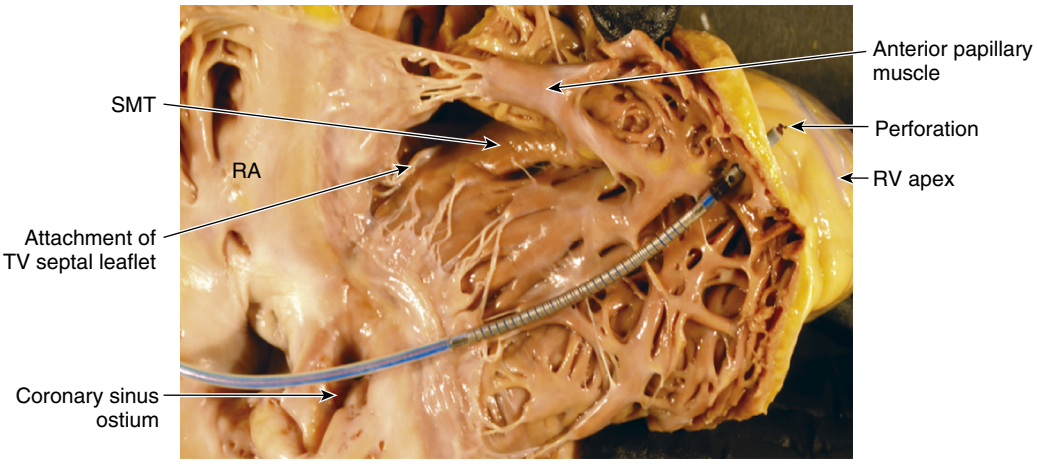
• **Fig. 2.68** Formalin-fixed heart with simulated example of optimal course of a transvenous implantable defibrillator lead into the right ventricle, passing across the tricuspid valve and positioned in the right ventricular apex. *RA*, right atrium; *RAA*, right atrial appendage; *RV*, right ventricle; *TV*, tricuspid valve.

implants, caution must be exercised when advancing the lead into the right ventricle to avoid major changes in direction while the lead is within the trabeculae, as this may result in entanglement of the lead in the trabeculae. The lead should be withdrawn to free the tip before it is readvanced. The right ventricular free wall should be avoided as the wall thinness may make this position more susceptible to perforation (Fig. 2.70).

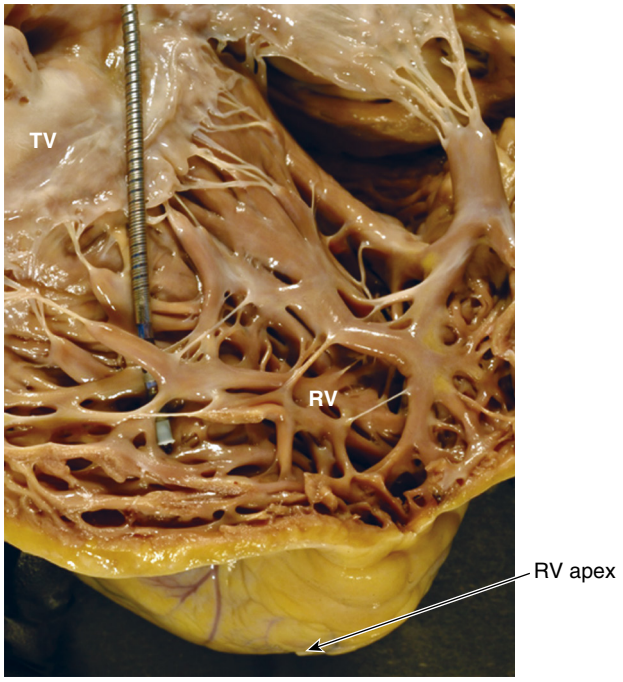
- The finding of mild tricuspid insufficiency after placement of RV pacemaker or defibrillator leads is often observed on echocardiography.[17–20] Moderate or severe tricuspid insufficiency can occur and be clinically significant. The causes of tricuspid insufficiency include leaflet perforation, leaflet entrapment (often of the septal leaflet against the septum with resultant fixed fibrosis), or interference with proper coaptation of the leaflets—worsened with multiple leads. Tricuspid insufficiency can also result from right atrial or coronary sinus leads if loops of the lead cross the tricuspid annulus, causing entrapment of the septal leaflet or inadequate leaflet coaptation (Figs. 2.71 to 2.73).
- Perforation of cardiac leads is a major and potentially life-threatening complication of lead placement and is further discussed in Chapter 13. Although the overall risk is low, any transvenous lead can potentially perforate owing to factors that include distal tip tension, motion of the lead



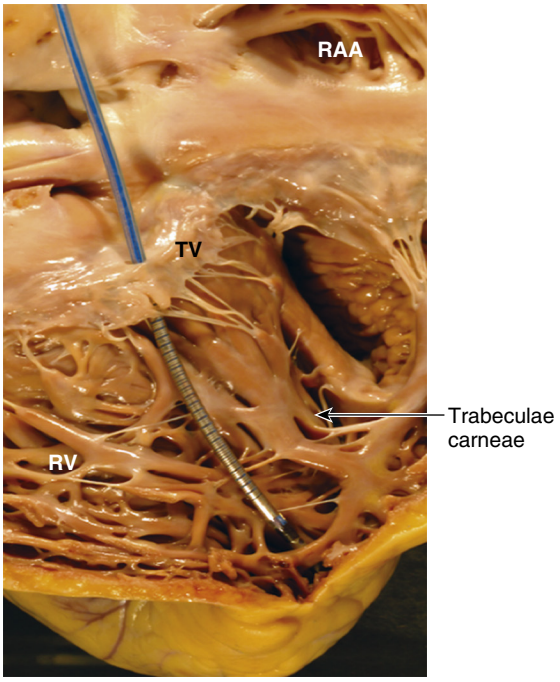
• **Fig. 2.69** Posterior-anterior chest radiograph in a patient with a dual-chamber pacemaker. The original pacemaker lead became entangled in the tricuspid chordae tendineae and could not be manually removed (*red arrow*). The lead tip is seen just at the lateral thoracic vertebral margin, which is consistent with a position just beyond the tricuspid valve annulus. The pacemaker lead (*green arrow*) is appropriately positioned in the right ventricular apex. A pacemaker lead inserted into the right atrium (*black arrow*) is positioned in the right atrial appendage. *RA*, right atrium; *RV*, right ventricle.



• **Fig. 2.70** Formalin-fixed heart with simulated perforation of the right ventricular apex by a transvenous implantable defibrillator lead. *RA*, right atrium; *RV*, right ventricle; *SMT*, septomarginal trabeculation; *TV*, tricuspid valve.



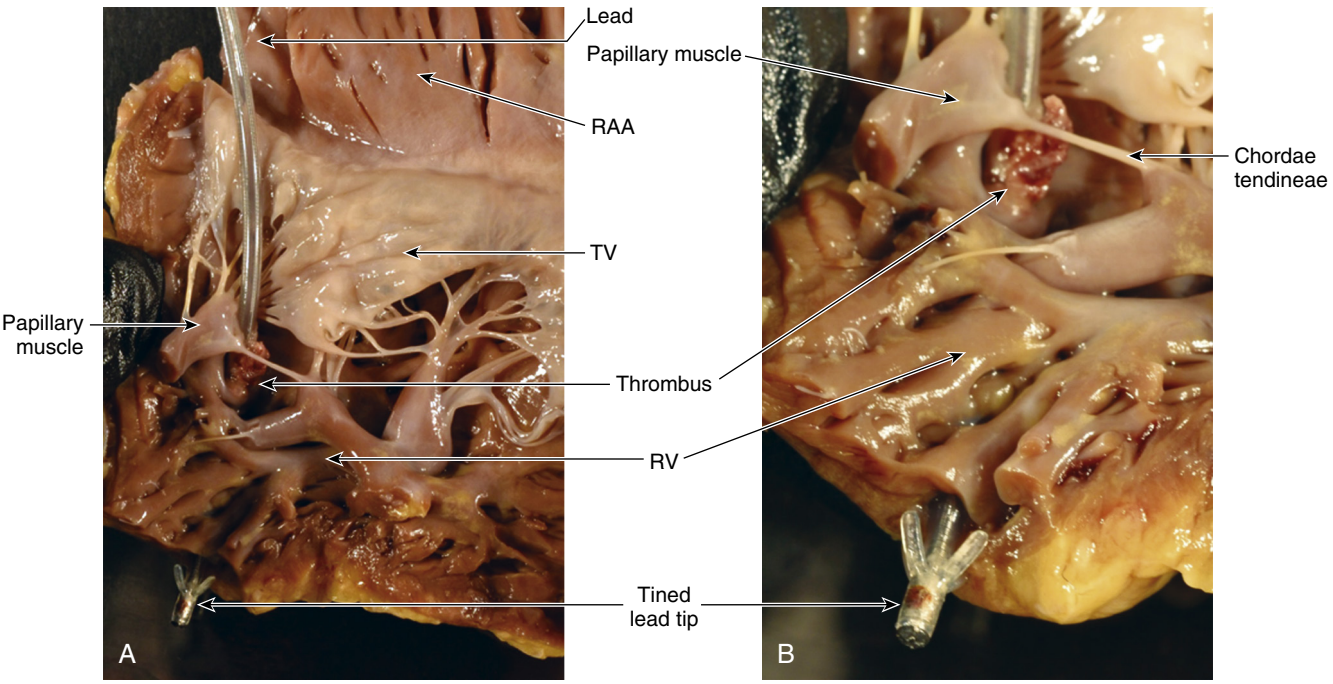
• **Fig. 2.71** Formalin-fixed heart with simulated entrapment of a transvenous implantable defibrillator lead in the right ventricular trabeculae carneae. *RV*, right ventricle; *TV*, tricuspid valve.



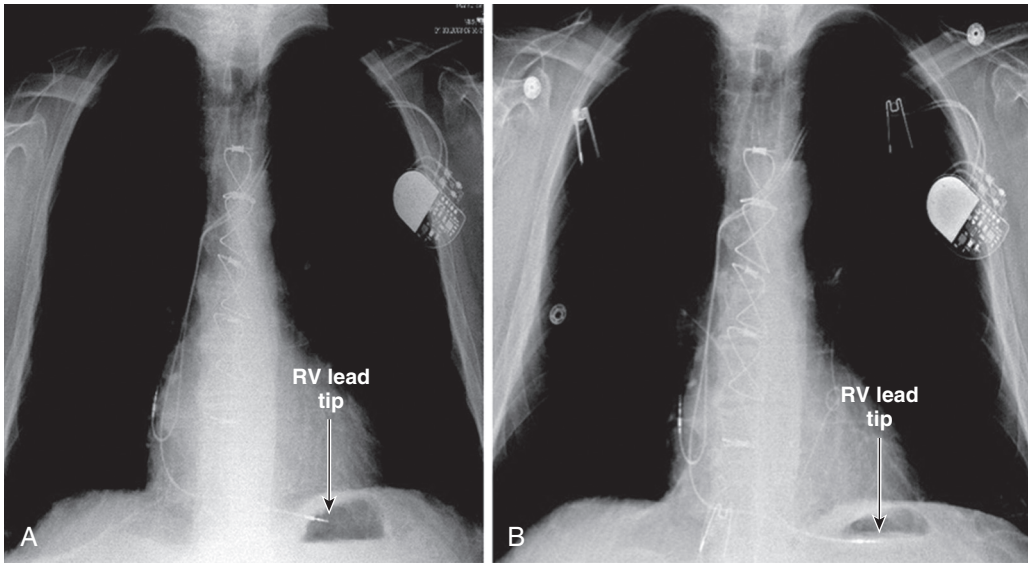
• **Fig. 2.72** Formalin-fixed heart with simulated perforation of tricuspid valve by a transvenous implantable defibrillator lead. *RAA*, right atrial appendage; *RV*, right ventricle; *TV*, tricuspid valve.

within the heart, thinness of the myocardium, and myocardial tissue integrity. Acute perforations are often associated with pericardial hemorrhage and cardiac tamponade. Perforations can occur more distant from the initial implant procedure and may not present with clinical signs of pericardial hemorrhage or tamponade. The screw of the lead may engage the visceral and parietal portions of the pericardium, "entrapping" the layers in proximity such that bleeding into the pericardial sac does not occur. Signs and symptoms may be chest pain, abnormal chest radiograph, or abnormalities of electrical pacing parameters. The findings can be subtle and, if present, should raise high clinical concern for perforation (Fig. 2.74).

- An alternative and popular position for placing right ventricular pacing or defibrillation leads is the smooth-walled right ventricular outflow track or subpulmonic septum (Fig. 2.75 and Video 2.11). This is often preferred to avoid the thin-walled RV apex and can be placed in the low, mid-, or high septum

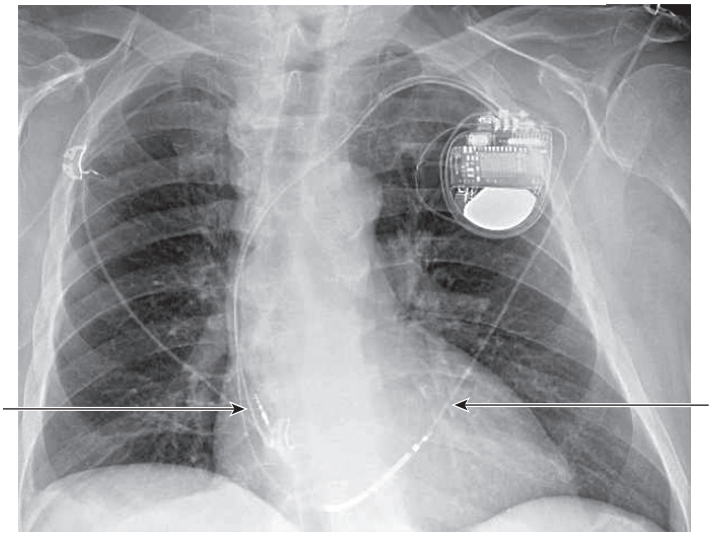


• **Fig. 2.73** Formalin-fixed heart from an autopsy specimen showing a portion of the tricuspid valve and right ventricle. (A) Tined right ventricular lead that is entrapped by the chordae tendineae–papillary muscle complex of the tricuspid valve, with visible thrombus adherent to the lead at this site. The tip of the lead is visible as a result of the autopsy-related incision. (B) Closer view of the entrapped lead and thrombus. *RAA,* right atrial appendage; *RV,* right ventricle; *TV,* tricuspid valve.

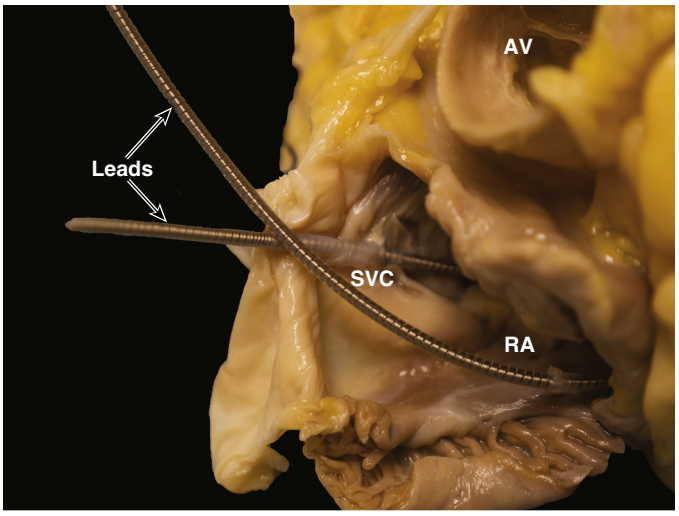


• **Fig. 2.74** Overpenetrated posterior-anterior chest radiograph allows visualization of the course of a right ventricular pacing lead. (A) Lead ending in the fluoroscopic position of the right ventricular apex the day after initial implantation of a dual-chamber pacemaker. (B) One week after implantation, showing the lead position located farther to the patient's left when compared with the initial chest radiograph. Interrogation of the device and echocardiography confirmed a lead perforation. *CXR,* chest radiograph.

depending on the electrical parameters obtained. Use of the RV septal position has also been proposed to obtain improved paced hemodynamics with less interventricular dyssynchrony.[21–27] The suggested target area is the septomarginal trabecular zone of the right ventricular septum, although others have proposed that retaining true synchrony can be achieved only through the technique of His-bundle pacing. The His-bundle pacing technique uses a specific guide sheath to position the lead such that only the His bundle is captured, thereby preserving normal conduction through the bundle branches.[28–32]



• **Fig. 2.75** Posterior-anterior chest radiograph demonstrating a dual-chamber implantable cardioverter defibrillator with the right ventricular lead positioned in the right ventricle septum. *RA,* right atrium; *RAA,* right atrial appendage; *RV,* right ventricular.



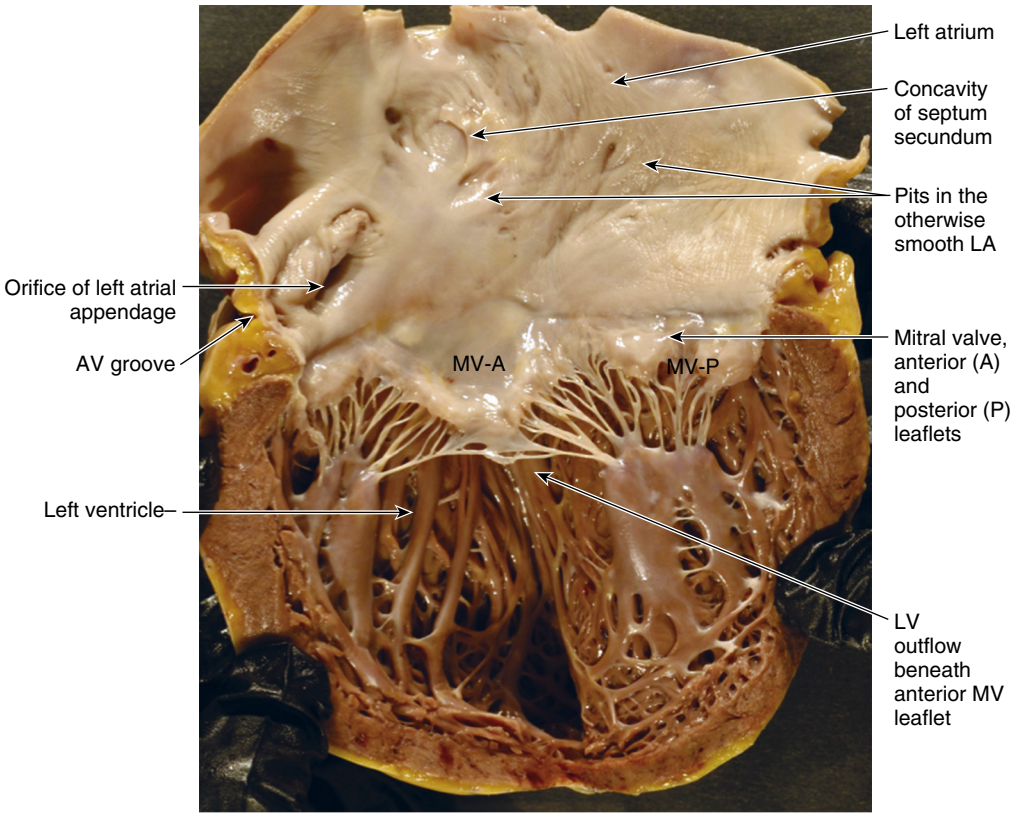
• **Fig. 2.76** Formalin-fixed heart showing two transvenous defibrillator leads passing through the superior vena cava into the right atrium. Fibrosis is apparent at points of contact with the superior vena cava and right atrial wall. *AV*, aortic valve; *RA*, right atrium; *SVC*, superior vena cava.

- Chronic leads will develop adhesions at any point where the lead makes contact with myocardial tissue or vascular endothelium. This becomes important when attempting to remove or extract a lead as these adhesions affix the leads to the adjacent structures, posing obstacles to lead removal and potential vascular avulsion with a catastrophic outcome. Lead extraction techniques are beyond the scope of this book (Fig. 2.76).

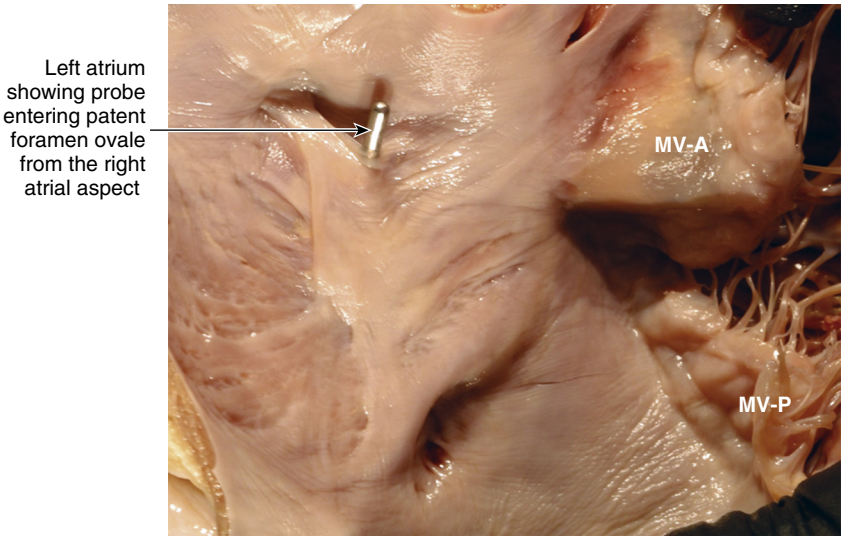
Left Atrium, Left Ventricle, and Mitral and Aortic Valves

Like the right atrium, the left atrium has an auricular appendage, a venous portion, and an atrioventricular vestibule.[8,33] The left atrial appendage is more tubular and elongated than the right, with a distinct orifice connecting to the atrial body. It is lined by pectinate muscles, but in contrast to the right atrium, these are confined to the appendage and do not extend to the atrioventricular vestibule (Fig. 2.77). The left atrial wall is somewhat thicker than the right atrium (approximately 3 mm thick), but the translucent appearance between pectinate muscles is also present. There is no crista terminalis in the left atrium. The smooth-walled body of the left atrium with the ostia of the pulmonary veins makes up the major portion of the atrium, formed by the incorporation of the proximal pulmonary veins into the embryonic atrium. The left atrial body is larger than the right and has a generally smooth transition to the atrioventricular vestibule, although it can contain a number of pits and troughs (Fig. 2.78). It receives the four pulmonary veins in a variable configuration; muscular fibers of the left atrium usually extend in a tube- or sleevelike circumferential fashion onto the attachments of the pulmonary veins (Fig. 2.79). The atrial septum includes a concavity corresponding to the embryologic ostium secundum. As in the right atrium, the atrial and ventricular myocardium are electrically isolated by a plane of fibrofatty tissue (the atrioventricular groove) located at the hinge lines of the atrioventricular valves (tricuspid valve on the right and mitral valve on the left) (see Fig. 2.77).

There are a number of interatrial myocardial strands that compose the subepicardial myoarchitecture of the atria. One prominent component is the Bachmann's bundle, usually the largest of the interatrial bundles, extending as a broad muscle bundle on the anterior atrial wall between the right and left atrial appendages in a plane parallel to the atrioventricular junction (see Fig. 2.63). There are a number of other muscle bridges that connect various structures of the atria and adjacent vessels, including interatrial connections and connections between the left atrium and the coronary sinus and the right atrium and the right pulmonary veins.[8,11]



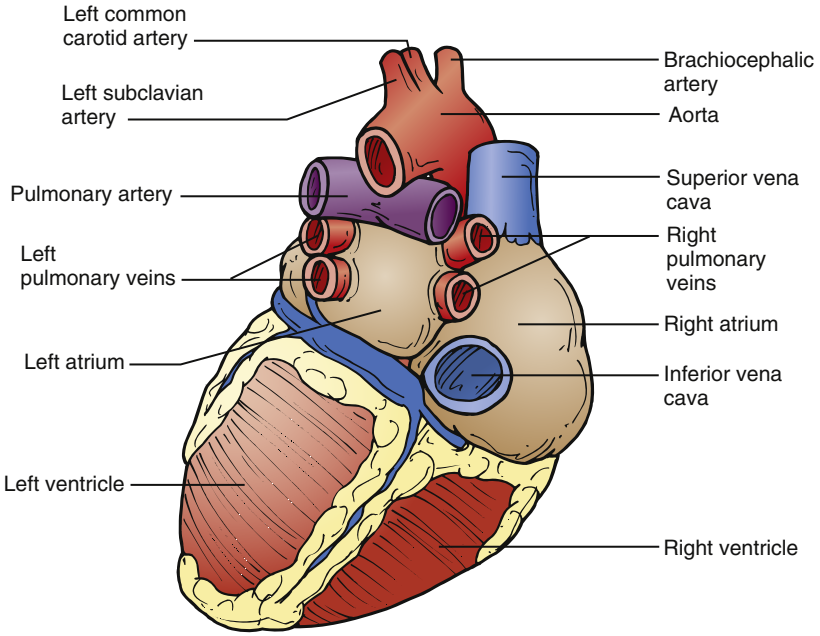
• **Fig. 2.77** Formalin-fixed heart showing the left atrium, mitral valve, and left ventricle. Note that the left ventricle trabeculae carneae become more prominent toward the apex. The left atrial appendage, not visualized in this photograph, is the only trabeculated part of the otherwise relatively smooth left atrium. There are some shallow pits and irregularities in the otherwise smooth left atrium. The left atrial appendage has a well-delineated orifice. There is no counterpart to the right atrial crista terminalis. The atrioventricular groove contains fatty tissue, which surrounds the arteries and veins that course through this region. The left ventricular outflow tract is located behind the anterior leaflet of the mitral valve, which is structurally in fibrous continuity with the aortic valve. *AV*, atrioventricular; *LA,* left atrium; *LV*, left ventricle; MV, mitral valve.



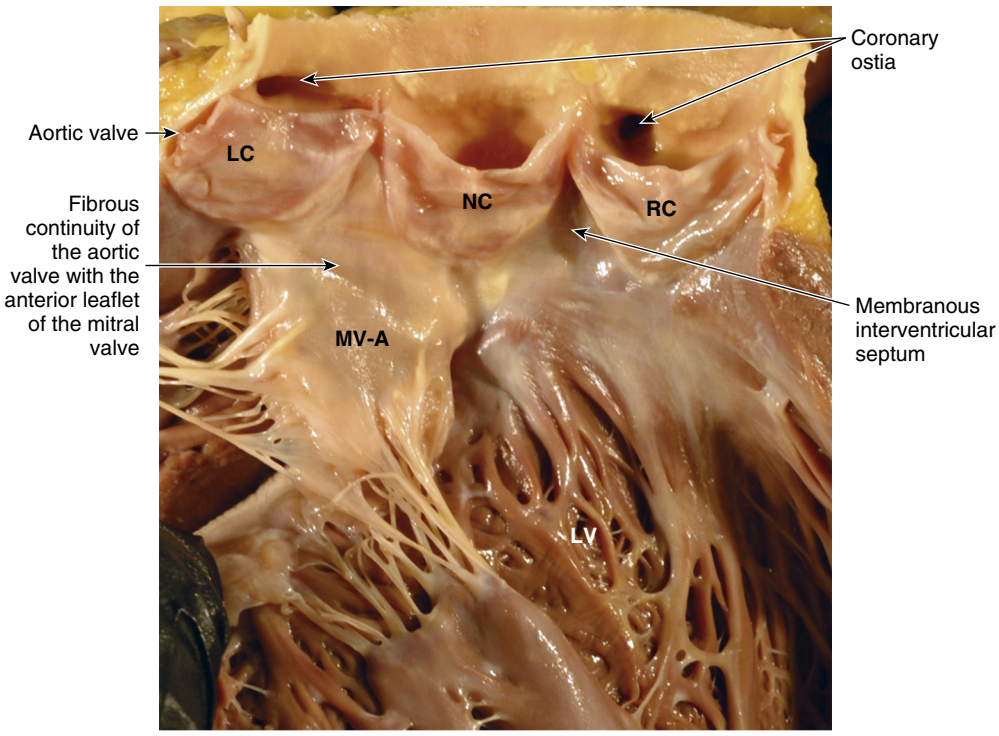
• **Fig. 2.78** Formalin-fixed heart section of left atrium, corresponding to the image in Fig. 2.64A. A probe is shown entering the left atrium from the right atrial side through a patent foramen ovale. *MV-A*, mitral valve anterior leaflet; *MV-P*, mitral valve posterior leaflet.

Left Ventricle and Mitral and Aortic Valves

The left ventricle has an inlet, associated with the mitral valve, and an outlet, associated with the aortic valve. In contrast to the right heart, the inlet and outlet are in close proximity. The anterior leaflet of the mitral valve is in fibrous continuity with the aortic valve, producing the "subaortic curtain" that separates the inflow and outflow in the left ventricle (see Figs. 2.51, 2.77, and 2.80). The mitral leaflets are distinct in configuration and attachment to the well-defined papillary muscles. The left



• **Fig. 2.79** Illustration showing the position of the pulmonary veins. Anatomic abnormalities can include a common ostium to the superior and inferior veins or early bifurcation of the veins.



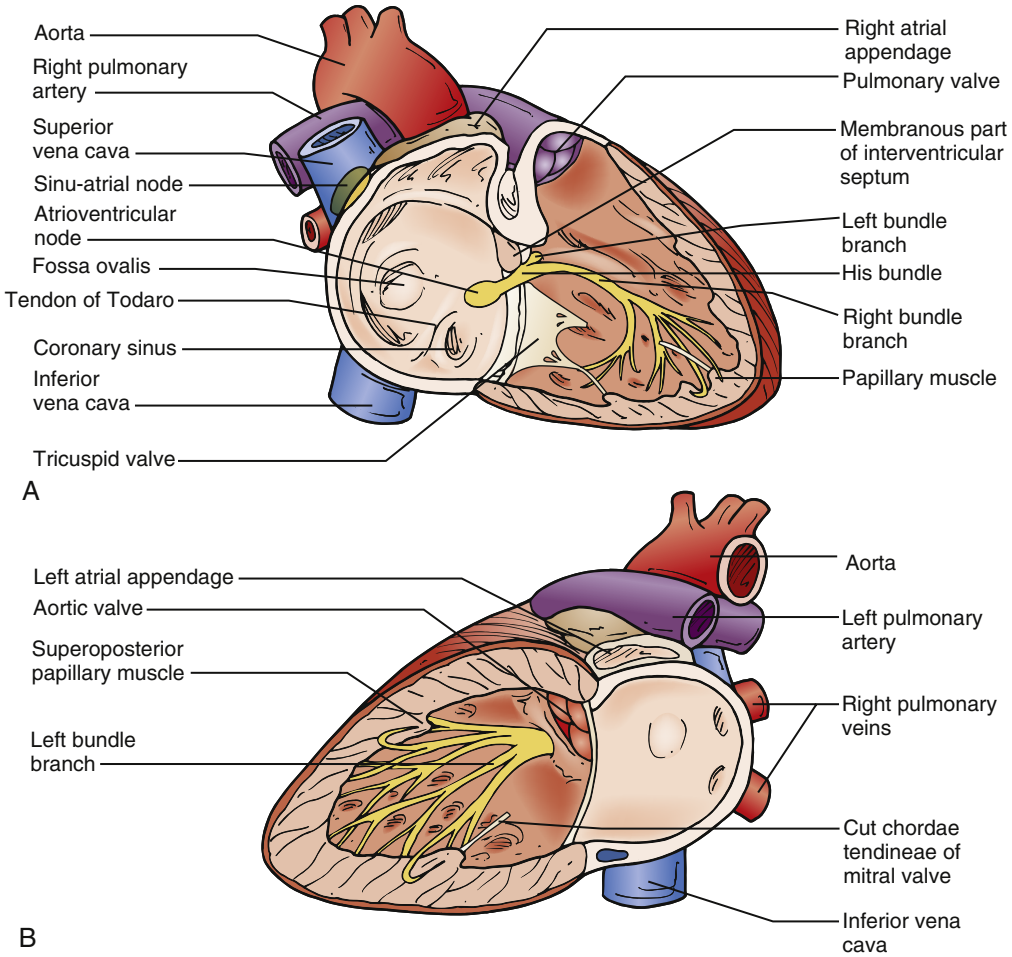
• **Fig. 2.80** Formalin-fixed heart section showing the left ventricular outflow tract with the aorta opened, allowing visualization of the aortic valve, a portion of the mitral valve, and the basal left ventricle. The membranous interventricular septum marks the site where the His bundle exits the septum and forms the base of the infravalvular trigone between the right and noncoronary cusps of the aortic valve. *LC,* left coronary cusp of aortic valve; *MV-A*, mitral valve anterior leaflet; *NC,* noncoronary cusp of aortic valve; *RC,* right coronary cusp of aortic valve.

ventricle is more finely trabeculated than the right, and the trabeculae are more prominent in the apical region than on the septum. The aortic valve has three leaflets with semilunar attachments to the aortic wall, including adjacent segments of left ventricle in the bases of two of the sinuses of Valsalva. The aortic leaflets (cusps) and sinuses are termed the right and left coronary and noncoronary leaflets. The membranous component of the interventricular septum forms the base of the infravalvular trigone between the right and noncoronary leaflets, which is also the location of the left His bundle as it exits the ventricular septum to arborize in the left ventricle.

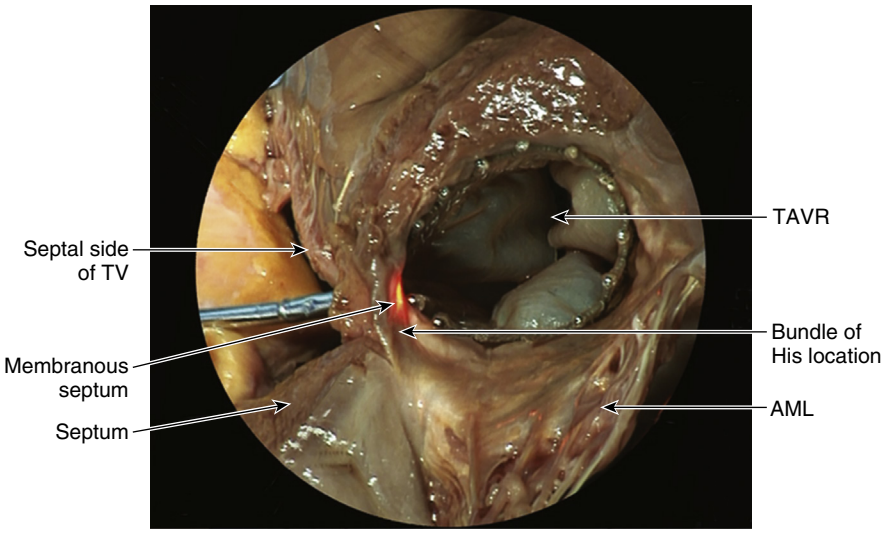
Conduction System

The conduction system consists of the pacemaker tissue in the sinus and atrioventricular nodes and the system of conduction fibers that convey the electrical excitation to the cardiomyocytes of the atria and ventricles (Fig. 2.81).

The sinus node functions as the cardiac pacemaker and is located at the junction of the superior vena cava and the right atrium, usually in the lateral aspect of the sulcus terminalis. The superior aspect is located subepicardially, while the inferior aspect extends into the myocardium and is located closer to the subendocardium of the crista terminalis. It is described as crescent or tadpole shaped, and has a mean length of 13.5 mm (range of 8 to 25 mm) in the adult heart. Less commonly, it straddles the summit of the terminal crest with a horseshoe-shaped crest of the right atrial appendage[8] (see Figs. 2.52 and 2.81). Specialized internodal tracts between the sinus and the AV nodes have not been identified, and the impulse is conducted through the atrial cardiomyocytes. The primary pathways through the atrial musculature include the crista terminalis and the margin of the fossa ovale. Conduction to the left atrium is primarily through the musculature of the Bachmann's bundle, although there are additional and variable pathways related to the myoarchitecture of the atria, previously described[8,11] (see Fig. 2.63). The AV node is located in the atrial muscular portion of the atrioventricular septum. The AV node and the bilateral atrial musculature are separated from the ventricular musculature by the fibrofatty tissue of the atrioventricular groove where the atrioventricular valves attach. The location of the AV node is defined by the triangle of Koch, as follows: the tendon of Todaro, which extends from the free border of the Eustachian valve of the inferior vena cava orifice to the central fibrous body at the atrial portion of the membranous septum; the hinge/annular attachment of the septal leaflet of the tricuspid valve; and the ostium of the coronary sinus (see Figs. 2.54 through 2.56). The AV node contains compact and transitional zones and abuts the central fibrous body, which it enters to become the penetrating atrioventricular bundle of His.



• **Fig. 2.81** (A) Anterior and (B) posterior views of the conduction system.

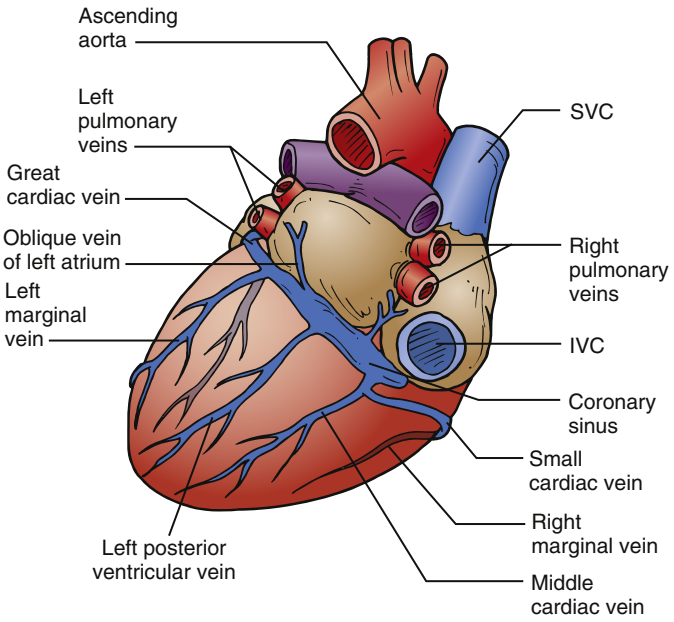


• **Fig. 2.82** Medtronic Core Valve transaortic valve replacement is shown in its position placed within the aortic annulus. An illuminated probe shows the proximity of the membranous septum to the bundle of His. *AML*, anterior mitral valve leaflet; *AV*, aortic valve; *TAVR*, transaortic valve replacement; *TV*, tricuspid valve.

The atrioventricular bundle of His continues from the AV node along the lower border of the ventricular membranous septum on the crest of the muscular ventricular septum to split into right and left branches. The right bundle branch is a rounded, relatively discrete group of fibers that courses inferiorly toward the RV apex on the right side of the interventricular septum in the septomarginal trabeculation, with few other septal branches. It passes through the consistently located but variably prominent moderator band into the base of the anterior papillary muscle, where it divides extensively into the subendocardial fascicles, which extend into the parietal wall of the right ventricle. The left bundle branch passes through the ventricular septum between the membranous and muscular components, in the intercuspal space between the right and noncoronary cusps (see Fig. 2.81). In contrast to the right bundle, it consists of multiple fine interdigitating fascicles that arborize in the subendocardial left ventricular septum, extending apically as three main divisions: anterior, septal, and posterior. After extending to the base of the papillary muscles, the fascicles are distributed to the left ventricle. Along their courses in the ventricles, the fascicles divide and become continuous with the subendocardial plexus of specialized ventricular conduction cells, called Purkinje cells.

Clinical Correlations

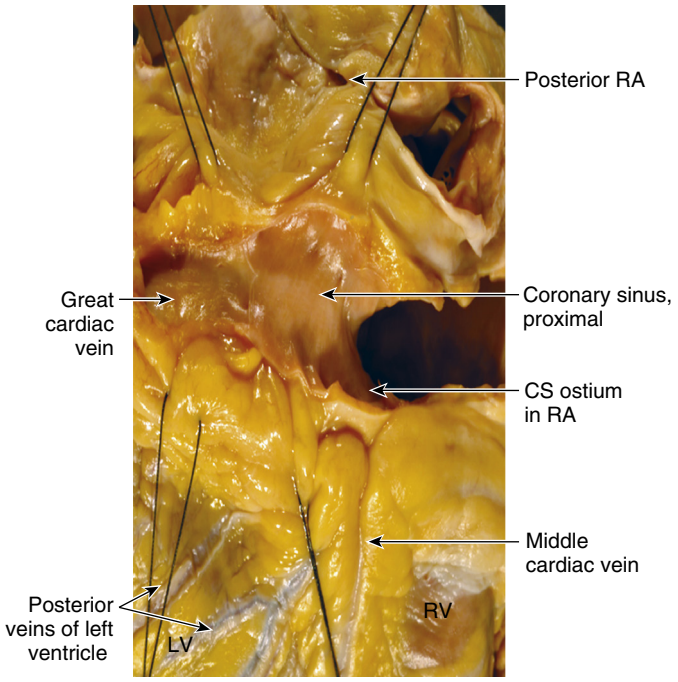
- Disease of the conduction system is multifactorial and beyond the scope of this chapter but forms the basis for therapeutic pacing. Specific anatomic relationships create situations in which disease of, or damage to, nearby structures results in conduction abnormalities. The sinus node is susceptible to injury during cannulation for cardiopulmonary bypass, which could lead to the need for a pacemaker. The positions of the AV node and His bundle in the septum make them susceptible to injury during surgical procedures affecting septal areas, such as mitral/aortic or tricuspid valve disease, valve replacements, or valve repairs. Recent percutaneous approaches to the aortic valve (transaortic valve replacement) are associated with a risk of AV node block or new left bundle branch block, possibly necessitating pacemaker therapy, although this risk is lower with the newer low-profile valves (Fig. 2.82).



• **Fig. 2.83** Illustration of the cardiac veins.

Venous Anatomy Including the Coronary Sinus

The major cardiac veins are located on the external surface of the heart and drain into the coronary sinus and then into the right atrium[34,35] (see Figs. 2.49B and 2.83). The 2- to 3-cm-long coronary sinus is the major conduit for the drainage of cardiac venous blood and is located in the



• **Fig. 2.84** Formalin-fixed heart section showing the posterior atrioventricular groove with longitudinally opened coronary sinus and great cardiac vein held open by sutures. The coronary sinus ostium opens into the right atrium. The first few centimeters (proximal) of the coronary sinus generally have a muscular sleeve of variable length. *CS*, coronary sinus; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

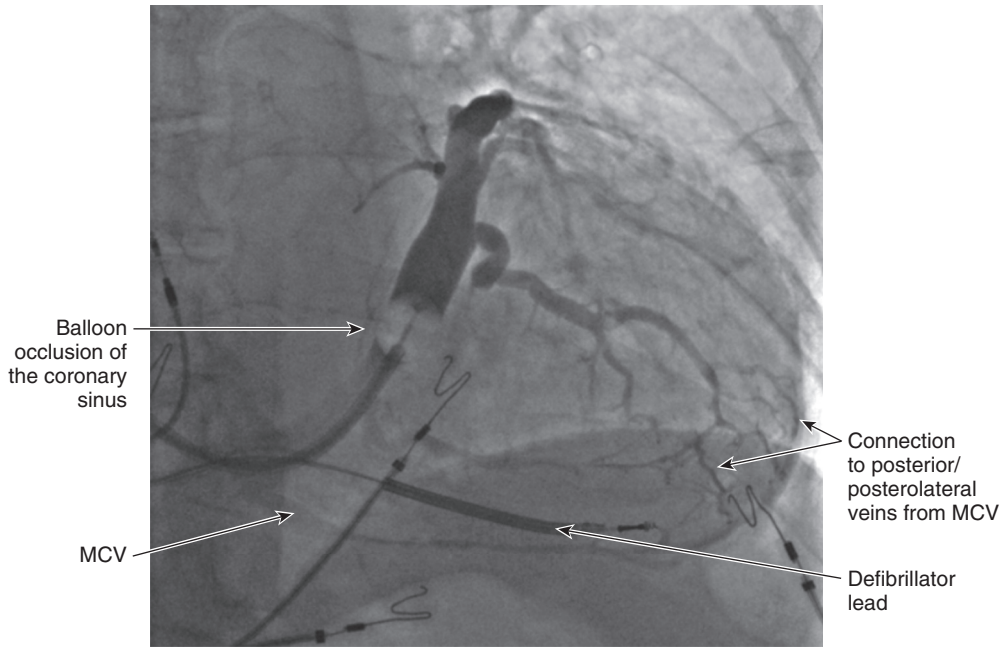
posterior atrioventricular groove with its orifice in the right atrium between the ostium of the inferior vena cava and the tricuspid valve (Figs. 2.84 and 2.85). It is continuous with the great cardiac vein; its other major tributaries are the posterior (inferior) vein of the left ventricle, the middle cardiac vein, the small cardiac vein, and the oblique vein of the left atrium.

Anteriorly from the apex, the great cardiac vein originates as the anterior interventricular vein and drains upward in the anterior interventricular sulcus adjacent to the left anterior descending coronary artery. The great cardiac vein then turns leftward in the anterior atrioventricular groove, along the course of the left circumflex coronary artery. The great cardiac vein becomes the coronary sinus when it joins the oblique vein of the left atrium, also termed the vein of Marshall. This remnant of the embryologic left common cardinal vein is located on the posterior left atrium between the left atrial appendage and the left inferior pulmonary vein. It is continuous with the ligament of the left superior vena cava or ligament of Marshall, is quite small, and may be atretic. Its orifice in the coronary sinus is generally adjacent to the filmy unicuspid or bicuspid valve of Vieussens, present in 80% to 90% of hearts. This valve can be present even when the vein of Marshall is atretic and marks the junction of the great cardiac vein with the coronary sinus. The inferior (or posterior) vein of the left ventricle usually enters the midportion of the coronary sinus but sometimes enters the great cardiac vein near the origin of the coronary sinus. The middle cardiac vein starts at the posterior apex and drains upward in the posterior interventricular groove adjacent to the posterior descending coronary artery, usually entering the coronary sinus near its atrial ostium. In some cases, the middle cardiac vein has a separate opening into the right atrium. The middle cardiac vein can also connect with the great cardiac vein at the anterior apex, resulting in a complete venous circle including the coronary sinus (see Figs. 2.82 and 2.85).

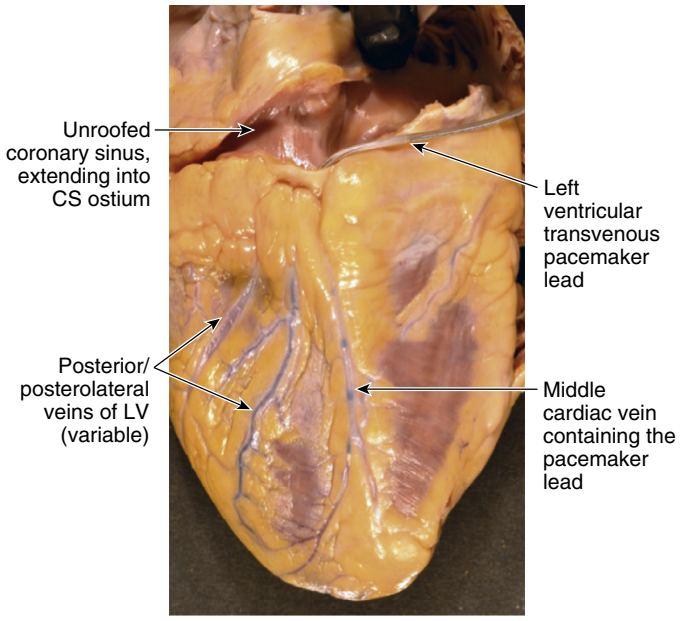
There are a number of variably present and smaller venous branches. The left marginal (or obtuse marginal) vein of the left ventricle, which drains much of the left ventricular myocardium, enters the great cardiac vein. The small cardiac vein is located in the posterior right atrioventricular groove and opens into the coronary sinus near the atrial end. The right marginal vein, located along the acute border of the right ventricle, drains much of the anterior right ventricle and may either connect with the small cardiac vein or open directly into the right atrium. The smallest of the veins are the Thebesian veins, which open into all cardiac cavities, particularly in the right atrium and ventricle. With valveless orifices and tiny lumina, they are difficult to demonstrate, although they are known to connect arterioles and venules with both subendocardial sinusoids and cardiac chambers.

Clinical Correlations

- The coronary sinus orifice has a muscular sleeve continuous with the left atrium that extends 2 to 5 cm into the vessel (see Fig. 2.84).
- Placement of transvenous pacemaker leads into coronary sinus branches has provided a safe and effective method of pacing the left ventricle for cardiac resynchronization therapy. The coronary sinus orifice can be difficult to access owing to anatomic variants of the Thebesian valve (Video 2.12) and acute angled take-offs, as well as when the orifice of the middle cardiac vein is separate from the main coronary sinus. Generally, posterior or lateral branches are targeted for left ventricular lead placement, although the middle cardiac vein can be used when the former are not available if the vein extends to and supplies branches to the lateral left ventricular wall (see Chapter 8 and Fig. 2.86). The venous branches of the coronary sinus are surprisingly variable, with some patients having multiple branches and others having only a few.
- Most of the major cardiac veins contain valves at their orifices, but there are few valves within the coronary sinus; the most common is the thin valve of Vieussens at the entrance of the great cardiac vein, which generally does not pose a significant problem for cannulation of this vessel. Occasionally the valve is more robust and a guidewire may be deflected into the vein of Marshall, which generally takes off at this juncture between the great cardiac vein and the valve of Vieussens.
- An independent middle cardiac vein orifice can be inadvertently cannulated when attempting to place a right ventricular pacing or defibrillation lead, which in the anterior-posterior fluoroscopic view may appear similar to placement in the right ventricle. Therefore, it is always important to assess lead position in multiple fluoroscopic views. There



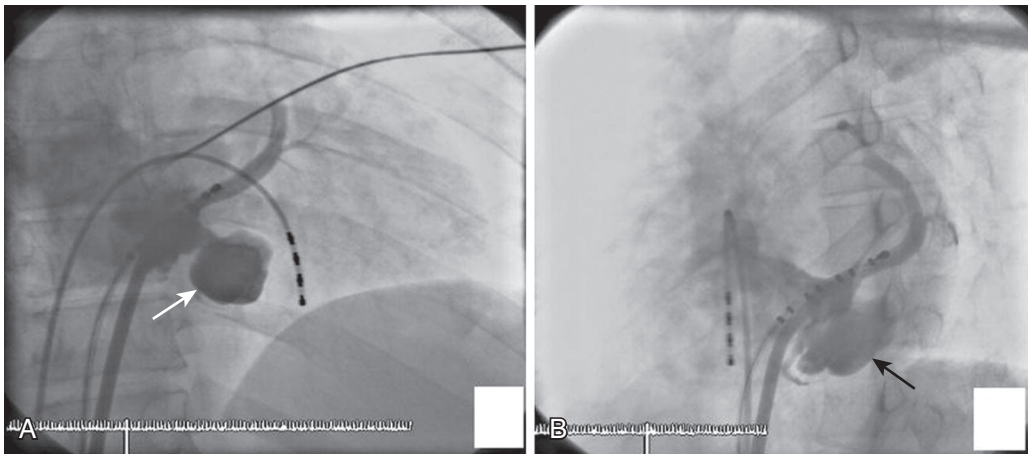
• **Fig. 2.85** Fluoroscopic images showing the continuity between the middle cardiac vein and the posterolateral vein branches of the coronary sinus. *MCV*, middle cardiac vein.



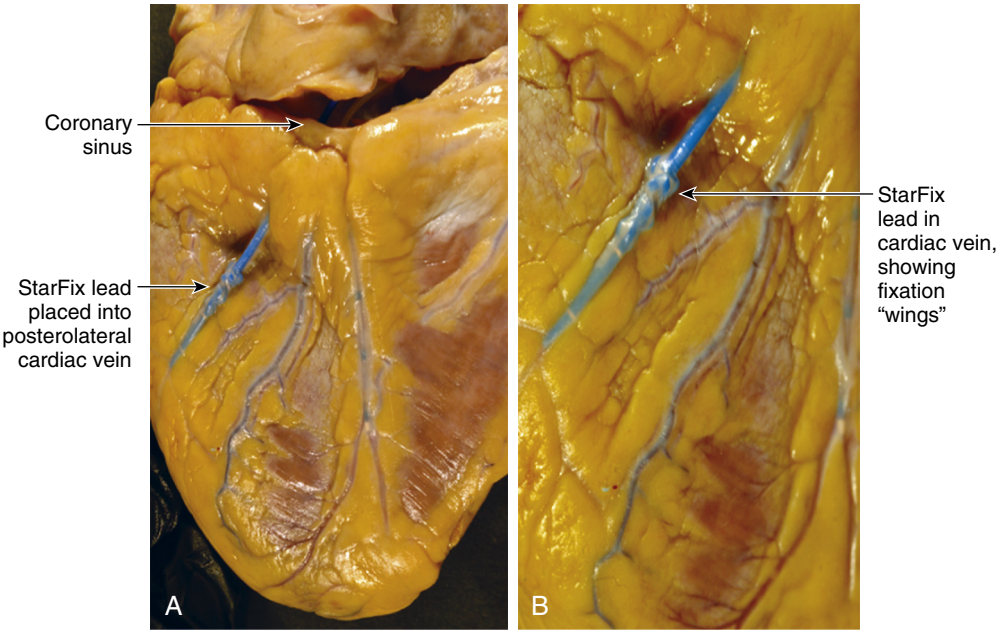
• **Fig. 2.86** Formalin-fixed heart showing the posterior external heart with unroofed and longitudinally opened coronary sinus *(CS)* extending into the right atrial CS ostial junction. In this simulation, a left ventricular quadripolar pacemaker lead has been advanced into the middle cardiac vein. *LV,* left ventricle.

are occasions in which the coronary sinus is purposely used to place a lead for ventricular pacing. This may be the choice in a situation when the patient has a mechanical tricuspid valve.

- As discussed in Chapter 8, when placing LV leads into posterior-lateral and lateral branches of the coronary sinus, the threshold at which phrenic nerve stimulation occurs should be tested (see Fig. 2.62).
- Anatomic variants of the coronary sinus can be found in up to 10% of patients without cardiac arrhythmias.[36] These can be in the form of fusiform dilation or aneurysmal dilatation, creating challenges in coronary sinus cannulation for CRT implantation. Coronary sinus diverticula have also been rarely described in association with arrhythmias, in particular as the location of posteroseptal accessory atrioventricular pathways[37–41] (Fig. 2.87).
- Prior cardiac surgery can result in stenosis of the coronary sinus as a result of retrograde cardioplegia administered during cardiopulmonary bypass. Encountering obstructions in the coronary sinus or at the take-off to branches can often be managed with specialized guide sheaths and, if necessary, venoplasty of the obstruction.[42,43]
- Lead placement for left ventricular pacing via selected branches of the coronary sinus produces unique challenges including inadvertent movement and dislodgement. Various passive fixation mechanisms including tines, canted tips, and "corkscrew" configurations have been developed to provide pressure against the smooth-walled venous branch to stabilize the lead once it is positioned. One active fixation left ventricular lead, the Medtronic Attain StarFix, uses deployable circumferential plastic wings to secure the lead in the desired location (Fig. 2.88). While this lead may be more stable, removal presents significantly more challenges than a passive fixation lead placed in a similar position and may preclude reimplantation of a new lead within the same vessel.[44,45]



• **Fig. 2.87** Fluoroscopic (A) anteroposterior and (B) left anterior oblique images showing contrast venography of a coronary sinus diverticulum *(arrow)*. In this patient a posteroseptal accessory pathway was mapped to the coronary sinus diverticulum. (From Al Fagih A, Zahrani GA, Hebaishi A, et al. Coronary sinus diverticulum as a cause of resistant posteroseptal pathway ablation. *J Saudi Heart Assoc.* 2011;23:41–44.)



• **Fig. 2.88** Formalin-fixed heart showing posterior external anatomy with longitudinally opened coronary sinus. (A) Simulated view of a StarFix lead (Medtronic) that has been placed into a posterolateral cardiac vein from the coronary sinus. (B) Close-up view that shows the fixation "wings" of this specific lead.

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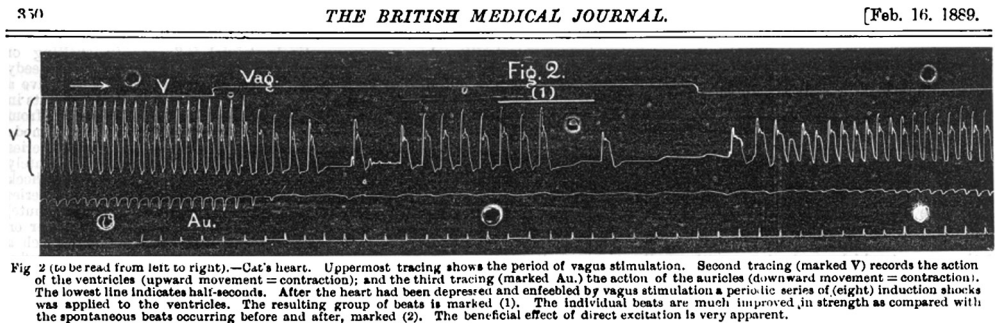
TABLE 1.1 History of Electrotherapy and Cardiac Pacing

Year	Observation/Development	Scientist
1580	Syncope and association with slow pulse	Geronimo Mercuriale
1600	Restarted an arrested pigeon's heart by a simple flick of the finger	William Harvey
1791	Electricity can stimulate living tissue	Luigi Galvani
1800	Devised the first electric battery	Alessandro Volta
1850	Induced ventricular fibrillation by electrical current in a dog heart	Carl Ludwig
1872	First successful resuscitation using external cardiac stimulation	Duchenne de Boulogne
1882	Heart rate change and arrhythmias through direct electrical current to the heart	Hugo von Ziemssen
1880–1890	Electrical currents could initiate ventricular fibrillation but strong electrical shocks can also potentially defibrillate the heart	Jon McWilliam, Jean-Louis Prevost, and Frederic Batelli
1892	The electrocardiogram	William Einthoven
1927	Used transvenous and transthoracic electrodes to pace various chambers of the dog heart	M. Marmrostein
1928	With alternative current and a needle inserted into a ventricle, intermittent electrical stimulation was able to resuscitate a child born with a cardiac arrest	Mark Lidwell
1932	The first "artificial pacemaker" (Hymanotor)	Albert Hyman, Henry C. Hyman
1949	First catheter electrode for pacing	Wilfred Bigelow, John A. Hopps
1951	External (transcutaneous) cardiac pacemaker	Paul Zoll
1957	First battery-operated wearable pacemaker	Earl Bakken, C. Walton Lillehei
1958	First implantable pacemaker	Rune Elmqvist and Ake Senning
1958	Endocardial pacing electrode	Seymour Furman, John Schwedel
1960	The lithium-iodide battery	Wilson Greatbatch
1972	Radioisotope pacemaker	Victor Parsonnet
1978	First dual-chamber pacemaker	H.D. Funke
Early 1980s	Steroid-eluting leads	
Mid-1980s	Rate-responsive pacemaker	
1990s	Microprocessor-driven pacemakers	
2000s	Biventricular pacemakers	

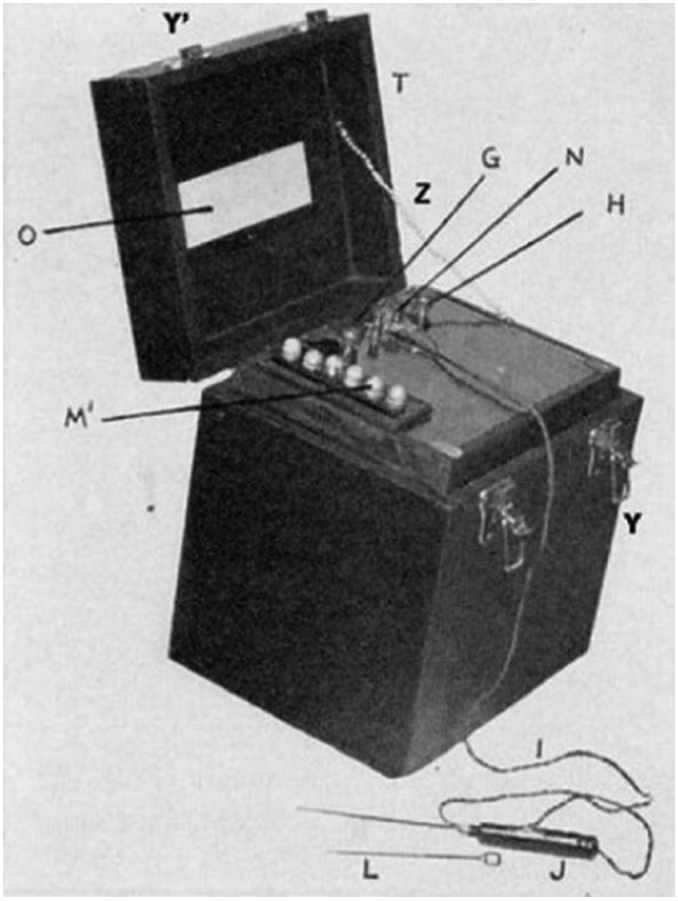
with his system was approximately 11 days, but his initial work became a starting point for development of the implantable pacemaker. Aubrey Leatham and Geoffrey Davies improved Zoll's transcutaneous stimulator by developing circuitry that could sense the electrocardiogram and pace only when needed. It had a fixed rate and two output ranges, thus creating the first "demand" pacemaker.[9]

C. Walton Lillehei found that a pacemaker, attached with a wire electrode directly placed in a dog's heart, could stimulate at lower voltages and with shorter pulse widths. In January 1957, a 3-year-old girl, who had developed complete heart block after open-heart surgery to correct a congenital defect, was paced using the Lillehei system and ultimately regained conducted sinus rhythm. The initial pulse generator, however, was totally dependent on external power from a wall socket (Fig. 1.4). A 3-hour power outage in October 1957 led to the death of a baby who was pacer dependent. Clearly, better technology was needed.

Recognizing this need, Earl Bakken, an electrical engineer in Minneapolis working out of his garage, developed a battery-operated pulse generator for pacemakers modeled after a transistorized metronome.[1,10] Reported in *Popular Electronics* in April 1956, the oscillator circuit pulse generator had been invented by the Massachusetts Institute of Technology radiation laboratory during World War II.[1] The device was miniaturized, self-contained in a Kiwi shoe polish can, and implanted as a functioning single-chamber pacemaker. This was the first time a battery power source was used for pacing—but the pulse generator remained external (Figs. 1.5 and 1.6).



• **Fig. 1.1** Tracing from John Alexander McWilliam's experiments in 1889 using induction shocks applied to the ventricle. He showed that vagal stimulation can inhibit the heart and that ventricular stimulation using induction shocks results in contraction and ejection. (From McWilliam JA. Electrical stimulation of the heart in man. *Br Med J*. 1889;1:348–350.)



• **Fig. 1.2** Albert Hyman's "artificial pacemaker" (1932). (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)

• **Fig. 1.3** The first external transcutaneous cardiac pacemaker (Paul Zoll, MD, 1951). (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)

The first fully implanted artificial pacemaker was placed on October 8, 1958, in a 43-year-old man with complete heart block. Ake Senning implanted the pacemaker, which was attached to epicardial electrodes placed subcutaneously in the epigastrium (Fig. 1.7). However, the pulse generator suffered from longevity issues, lasting only hours to a few days. In 1958, Schwedel and Furman, using a lumenless Cournand electrode catheter with a copper wire and an exposed metal tip, paced the endocardial surface of the heart of a 69-year-old man with "complete AV dissociation" (Fig. 1.8). In 1959, Wilson Greatbatch developed a fully implantable pacemaker pulse generator that was smaller (2 cm^3), used less current, and lasted longer. However, others claimed "firsts" regarding permanent transvenous pacemaker implants at about that time.[11–14]

By the 1960s, various manufacturers, including Medtronic (under Earl Bakken), Electrodyne (founded by Zoll), Wilson Greatbatch, and Elema Schonander, started producing different versions of pacemakers (Fig. 1.9). In the 1970s, widespread manufacturing of pacemakers began. The first pacemaker was a single-chamber device with fixed-rate pacing and no ability to sense. Subsequent devices were demand pacemakers.

Progress was rapid, and further key developments in pacemaker technology included (1) improved battery longevity; (2) bipolar pacing; (3) programmability of rate, atrioventricular (AV) interval, and refractory periods; (4) rate-responsive pacing; (5) blanking and refractory period settings to avoid oversensing, crosstalk, and pacemaker-mediated tachycardia; (6) transtelephonic and, eventually, sophisticated remote monitoring; (7) data storage; and (8) cardiac resynchronization therapy.

Battery Technology

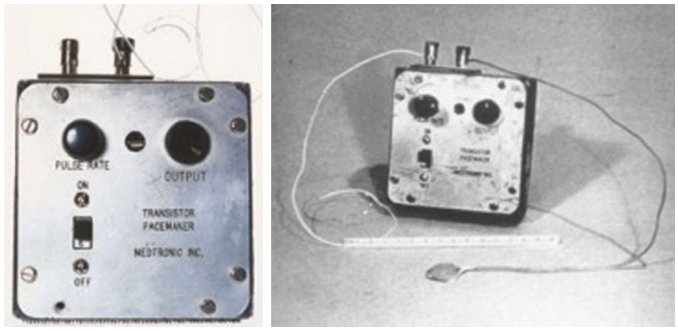
The first implantable pacemaker used nickel-cadmium rechargeable cells lasting hours at best. This was followed by pacemakers using Ruben-Mallory (zinc-mercuric oxide) cells that drove a two-transistor oscillator circuit (transformer-coupled blocking) encapsulated with an epoxy resin. This battery, used in most pacemakers implanted in the 1960s, had poor longevity and was subject to corrosion.[7] Nuclear-powered and rechargeable batteries were tried but abandoned.

In the early 1970s, Wilson Greatbatch pioneered the first lithium-iodide solid-state battery that had better longevity and was corrosion free. This battery remains the standard for pacemakers to this day.[1] Miniaturization of the pacemaker from large, abdominally implanted devices to something slightly larger than a quarter was possible owing to advances in battery and circuitry technology.

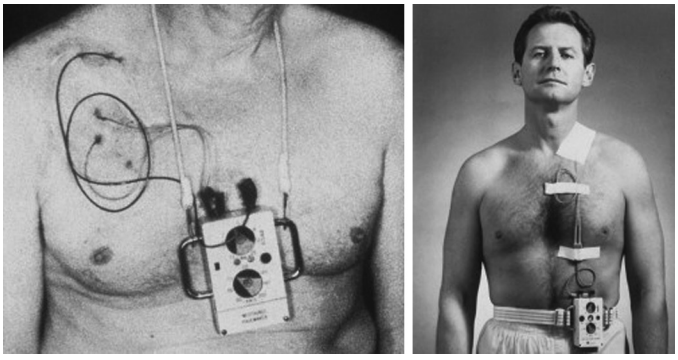
Programmable Pulse Generators

In 1961, the General Electric Company manufactured a device whose rate could be altered to pace at 70 beats/min or 100 beats/min via a magnetic switch, resulting in a crude form of "rate response." Medtronic subsequently introduced a programmable pacemaker in 1972 that had a gear train attached to a small bar magnet so that the rate could be programmed externally. The reed switch developed by the Cordis Corporation was an improvement over the earlier switches because it could be activated by an external programmer or magnet. By

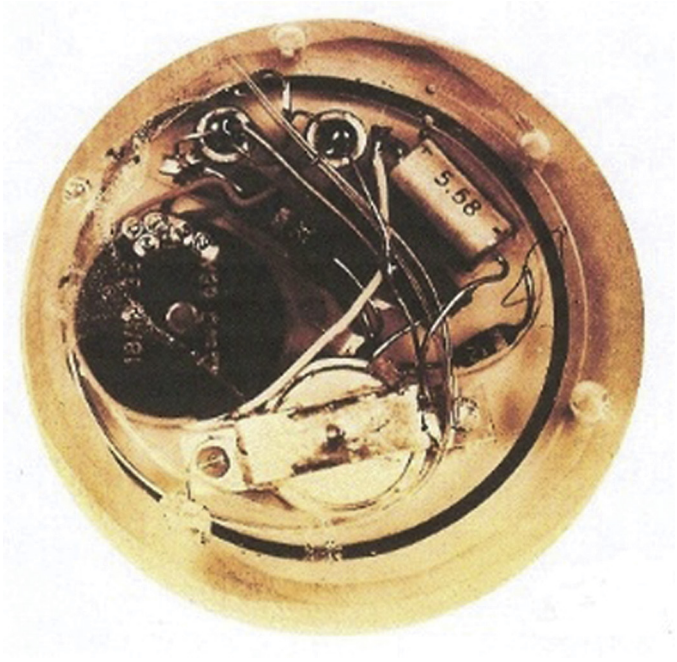
• **Fig. 1.4** Walton Lillehei with a young patient who is being paced. This particular wearable device needed alternating current main power for its operation. (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)



• **Fig. 1.5** The first battery-powered external pacemaker developed by Earl Bakken (1956). (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)



• **Fig. 1.6** Patients with wearable pacemakers (1958). (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)



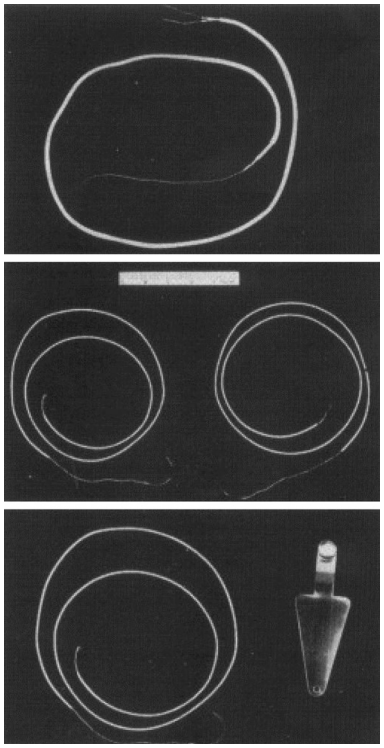
• **Fig. 1.7** The first fully implantable cardiac pacemaker (Ake Senning and Rune Elmqvist, 1958). This device was limited by poor battery life. (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)

1973, Medtronic introduced a pacemaker that could be programmed by external radiofrequency signals.

Critical programming features (rate, AV interval, and refractory periods, as mentioned earlier) improved the capability to adapt pacing to meet the physiologic needs of patients. Single- and dual-chamber pacemakers with multiple pacing modes (VVI, DDD, DDI, and VOO, among others) were developed (Table 1.2). Hysteresis was added to maximize native ventricular capture in patients with single-chamber ventricular pacemakers.

Although dual-chamber pacemakers had distinct advantages, there was the problem of pacemaker-mediated tachycardia.[15] To prevent pacemaker-mediated tachycardia, the timing cycle of postventricular atrial refractory period (PVARP) was developed. Crosstalk was another troublesome problem. The postatrial ventricular blanking period (PAVB) was developed so that the ventricular channel could not sense the atrial pacing output (or other electrical signals), inadvertently inhibiting ventricular pacing.[16]

Introduction of rate-responsive pacing for chronotropic incompetence has been attributed to Donaldson and Rickards,[17] but several types of rate-responsive physiologic pacing were developed at about the same time[18] that assessed movement, QT interval, temperature, respiration, and so forth. Other rate-responsive features included the addition of rate-responsive



• **Fig. 1.8** Early pacemaker electrodes. An indifferent electrode *(bottom)* is also shown. (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)

TABLE 1.2 Timing Cycles of a Modern-Day Pacemaker

A modern-day dual-chamber pacemaker has the following fundamental timing cycles:

- Lower rate interval
- Ventricular refractory period
- Atrioventricular delay (sensed and paced)
- Postventricular atrial refractory period (PVARP)
- Upper rate limit: maximum tracking of sensed events

The following are derived from the fundamental timing cycles:

- Ventriculoatrial interval/atrial escape interval
- Total atrial refractory period (TARP)

• **Fig. 1.9** Early pacemaker models from the 1960s. (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)

PVARP and AV intervals. Rate-responsive pacing algorithms vary among manufacturers and can include translation of measurements from an accelerometer, minute ventilation, vibration, and other methodologies to meet physiologic needs.

Auto-Programmability

Subsequently, automatic atrial and ventricular sensing, impedance measurements, and pacing thresholds were developed. The auto-capture threshold capability allowed the device to adjust its output constantly, thereby improving longevity.

Data Logging and Telemetry

Advancements in data storage allowed for collection of information concerning the percentage of atrial and ventricular pacing and sensing, tachycardia detection and atrial arrhythmia burden, stored lead- and device-related alerts, and sensing, pacing, and impedance trends. The density of information available advanced the pacemaker from a device that stimulated the heart to a device that stimulated but also provided a broad array of diagnostic parameters important in the surveillance of pacemaker function and arrhythmia management; it also allowed for optimization of individual clinical decision making.

Transtelephonic monitoring, introduced in the 1970s, remained the mainstay of remote monitoring for decades. Monitoring subsequently evolved into modern wireless telemetry encompassing a wide range of programmed settings and parameters and measurements of impedance, lead function, battery status, arrhythmia monitoring, and rate histogram data. Modern remote monitoring offers various programmed preset alerts to allow for a timely response to critical arrhythmic events. The ability to store vital lead-, device-, and arrhythmia-related data for clinical use and programming represents a major milestone. Remote monitoring, now a reality,[19] has been associated with improved survival for unclear reasons.

Necessity Is the Mother of Invention

Advancements in pacemaker technology were not all electrically driven. Early implantation was somewhat complex and performed by surgeons. As implantation evolved, cardiologists took the helm and implants were percutaneous. This required a completely new series of implant paraphernalia and adjunctive equipment. These advancements include leads, stylets, anchoring sleeves, sheaths, and implant and programming equipment that we now take for granted but that required an extensive amount of work to make implantations possible with minimal risk for adverse outcomes and good long-term success.

Defibrillation and the Development of Implantable Defibrillators

The development of the defibrillator followed a similarly remarkable, but serpiginous, course. In 1791, Italian physician and philosopher Luigi Galvani showed that a simple electrical spark could make the legs of a dead frog dance, marking the birth of the study of bioelectricity that would eventually lead to defibrillation. In 1850, German physiologist Carl Ludwig induced ventricular fibrillation via electrical current applied directly to a dog heart. John McWilliam in the 1880s did similar experiments and noted "fibrillar contractions" with electrical stimulation. He also realized the relationship between heart failure, syncope, and sudden death.

By the 1890s, Jean-Louis Prevost and Frederick Batelli at the University of Geneva could revive arrested animals with a capacitor discharge delivered directly to the heart. In Moscow, one of their graduate students, Lina Sistern, believed that she could bring back the dead; one of her young students, Naum Gurvitch, was one of the first to suggest use of biphasic waveform to deliver a shock.[1] However, it would be more than 100 years later before biphasic shocks were incorporated in implantable cardioverter-defibrillators (ICDs) for defibrillation.

It was not until many years later that these concepts were tested in people. A surgeon at the University Hospitals of Cleveland, Claude Beck, was investigating the effects of alternating current delivered directly to exposed hearts of animals to put them into ventricular fibrillation. His initial use of defibrillation, however, began in the operating room on open hearts to resuscitate those who developed ventricular fibrillation during surgical procedures. The first such attempt occurred in 1947 when a 14-year-old boy's heart fibrillated during surgery. Forty-five minutes of open-heart massage was not enough to resuscitate him. In desperation, Beck, using a crude device consisting of a transformer, a resistor, and two metal tablespoons with wooden handles, delivered a 110-V alternating current shock to the heart. The first shock failed but the second one restored normal rhythm and the patient survived.[20]

Subsequently, external alternating current and then direct current defibrillators became available. Pioneered by Bernard Lown in the 1960s, defibrillators were used for cardiac arrest resuscitation in hospitalized patients. The first portable defibrillator, built in 1965 by Hymen Wade and weighing approximately 150 lb, was designed to be plugged into an ambulance's starter battery, converting the 12-V battery to 4000 V. When the capacitors discharged, the current passed through the inductor, effectively delivering a hefty 80 Å of current.[21]

About that time, Michel Mirowski, working in Tel Aviv, Israel, was influenced by the fact that his mentor, colleague, and friend, Harry Heller, had died suddenly from recurrent ventricular tachycardia. He thought that an implantable defibrillator could detect ventricular fibrillation and provide immediate defibrillation. Had it been available, Heller may have survived. Mirowski, working with Morton Mower, Alois Langer, and Stephen "Doc" Heilman (a physician and engineer), in collaboration with Albert Mendeloff and Bernard Tabatznik, developed a prototype of the first ICD, which was tested in dogs. The initial company Medrad and then Intec was formed; ICDs were made in a Pittsburgh garage. Initial reactions were mixed; the early capability of these devices was

limited as they were quite large, weighing about 9 oz.[22] Nevertheless, in February 1980, the first device was implanted by Phil Reid at the Johns Hopkins Hospital in a 57-year-old female patient.

Although there was significant early controversy over the credibility of the ICD, the inventors remained undaunted, and in 1985, the first ICD was approved by the U.S. Food and Drug Administration (FDA) for implanting into humans. The initial indication for use in humans required at least two episodes of documented cardiac arrest from ventricular fibrillation requiring resuscitation. Owing to limited supplies, patients would have to wait in the hospital for days to weeks before a device became available because each was handmade.[22]

The original devices, implanted in the abdomen with two sensing electrodes screwed into the epicardium and two epicardial patch electrodes, could deliver 30 J of energy (Fig. 1.10). Ultimately, a percutaneous lead with sensing and defibrillation capabilities could be implanted into the heart. The ability to detect ventricular fibrillation accurately was an obstacle completely distinct from the sensing required for pacemakers. To detect ventricular fibrillation, a sensing circuit known as *probability density function* was developed. Devices were nonprogrammable but had selectable rate cutoffs. The shocks were initially monophasic. Early devices were not capable of pacing and had no electrogram storage. There was no capability of determining why a patient received a shock.

A major advance occurred with mass production of the ICD. In 1985, Intec was acquired by Cardiac Pacemakers. One of the important advances was the incorporation of integrated circuit technology in lieu of discrete components. In 1988, the Ventak P device became multiprogrammable with regard to rate and energy delivery. Nevertheless, many of these devices (>20%) did not achieve effective defibrillation, especially through implanted transvenous leads rather than epicardial patch electrodes.[23,24]

One early advance was the ability to program different zones of therapy—this required the development of completely new detection algorithms, since the detection for ventricular fibrillation would be distinctly different from ventricular tachycardia. Nearly simultaneously, in 1993, the CPI Ventak-P device and the Medtronic PCD were released. These devices were multiprogrammable and capable of antibradycardia and antitachycardia pacing.

The early to mid-1990s saw remarkable advances, which included the transvenous ICD lead, initially tunneled to an abdominal generator and then to the prepectoral generator

• **Fig. 1.10** Cardiac implantable electrical device models from the 1980s. (From Aquilina O. A brief history of cardiac pacing. *Images Paediatr Cardiol*. 2006;8:17–81.)

TABLE 1.3 Milestones in the Development of the Implantable Cardioverter-Defibrillator

1966	Conception
1969	First experimental model
1969	First transvenous defibrillation
1975	First animal implant (Mirowski)
1980	First human implant
1982	Addition of cardioverting capability
1985	Approval by the U.S. Food and Drug Administration
1988	First programmable device implanted (Ventak P)
1989	First multiprogrammable device implanted (PCD)
1993	Pectoral implantation (PCD Jewel) inactive
1994	Pectoral implantation ("active can")
1995	First dual-chamber implantable cardioverter defibrillator implanted (Defender)
1996	First implantation of a stand-alone atrial defibrillator
1997	First implant of a combined atrial-ventricular defibrillation system
1999	First implant of a heart failure defibrillator (Contak CD)

From Luderitz B. We have come a long way with device therapy: historical perspectives on antiarrhythmic electrotherapy. *J Cardiovasc Electrophysiol*. 2002;13(1 Suppl):S2–S8.

placement. The "active" can was a major achievement, eliminating the need for a separate "patch" electrode placed in the prepectoral space.

Despite the first commercial external defibrillator using a biphasic waveform, external and implantable defibrillators used monophasic waveforms during early development. Biphasic waveforms were ultimately found to be more effective and safer (i.e., less likely to reinitiate ventricular fibrillation).[25] Adoption of biphasic waveforms, shock vectors, and multicoil leads and refinements in capacitors and waveform delivery all followed rapidly. The ICD became an effective device capable of interrupting ventricular tachyarrhythmias with a high degree of success. The device was small enough to be implanted in most patients in the upper chest.

Modern ICDs have batteries that last a decade, can sense and pace in the atria and the ventricles, can detect and deliver therapy (antitachycardia pacing and/or shocks) for ventricular tachycardia and ventricular fibrillation (if necessary), and have circuitry to detect and discriminate supraventricular tachycardia from ventricular tachycardia. They are also equipped with rate-responsive pacing and AV delay extension algorithms to promote intrinsic AV nodal conduction. ICDs incorporate multiple sensors and provide detailed information regarding arrhythmias and therapy delivery. They allow for wireless communication with the programmer and the remote monitoring transmitter; the latter advance allows ICDs to be monitored remotely with a high degree of accuracy. Table 1.3 summarizes the milestones of ICD development.

Progress in Cardiac Implantable Devices in the Twenty-First Century

Cardiac Pacing for Heart Failure

The finding that conduction disturbances resulted in left ventricular (LV) dysfunction formed the basis for developing pacing therapies for heart failure. Cardiac resynchronization therapy (CRT) arose from seminal research showing that LV and biventricular pacing results in improvement of LV function in a population of patients with depressed systolic function and left bundle branch block.[26–28] CRT has become valuable therapy for selected patients with LV dysfunction, heart failure, and a wide QRS. This therapy has been revolutionary in patients who otherwise would have required a heart transplant or left ventricular assist device or would have no other viable treatment options.

CRT positively affects atrioventricular, interventricular, and intraventricular synchrony, resulting in favorable reverse remodeling.[29] Multiple randomized trials have shown that CRT improves mortality, reduces heart failure hospitalization, improves LV ejection fraction, promotes reverse remodeling (reducing LV volumes), and improves quality of life, functional capacity, and exercise tolerance.[26,27] CRT has also been shown to be beneficial in mild heart failure (New York Heart Association Classes I and II).[28]

Technological Advancements in Cardiac Resynchronization Therapy

From the original days of epicardial LV lead placement, almost all LV leads are now placed transvenously into lateral branches of the coronary sinus. Early leads were unipolar and stylet driven. Further developments led to multipolar leads, allowing for various pacing configurations to help reduce diaphragmatic stimulation and to allow selection of the best pacing vector with the lowest capture threshold, thereby also improving battery life. Most LV leads are now quadripolar, allowing an even wider flexibility of pacing vectors.[30] More recently, multipoint pacing and various adaptive algorithms have been introduced that may enhance therapy.[30,31] Additional programming characteristics may help determine the amount of left and right ventricular capture and pacing characteristics of all the leads.

Important to the development of CRT transvenous leads was the introduction of fixation methods. These were generally offered as a variety of angled or spiraled curves of the distal tips. A unique "active fixation" lead was the Medtronic Starfix lead. This lead incorporates a mechanism to deploy "splines" that hug the inside of the vessel wall, preventing dislodgement. The initial popularity of the Starfix lead was tempered by its unipolar configuration (allowing only two possible pacing vectors), as well as significant difficulty in extracting the lead if necessary. A newer version of an active fixation lead with a side helix (Medtronic LV Attain Stability, Model 20066) is in feasibility studies, and early data appear promising.[32,33] Other companies (e.g., Biotronik and Boston Scientific) have created leads that address the problem of lead dislodgment as well.

Advancements in lead delivery systems improved the success rate of transvenous LV lead placement. Variable curved outer guide catheters allowed access to difficult coronary sinus take-offs, whereas inner guide catheters helped subselect coronary sinus branches, thereby assisting lead delivery. Innovations in lead design, device programming, and delivery systems have resulted in tremendous improvement in CRT capabilities.[34]

Other advances in CRT include AdaptivCRT (Medtronic)[35] and LV endocardial pacing guided by real-time imaging of late-activating segments and dv/dt measurements. In patients with heart failure, these can be linked to, and guided by, real-time pulmonary artery pressures from devices such the CARDIOMEMS device,[36] thereby optimizing volume status and forward flow, improving functional class and LV ejection fraction, and potentially reducing heart failure hospitalizations.

Transthoracic Impedance Measurements for Heart Failure Monitoring

Some ICDs utilize thoracic impedance measurements to assess the patient's volume status. This particular feature tracks intrathoracic impedance and displays the trends in a graphical format. Clinical data suggest that changes in intrathoracic impedance and fluid accumulation in the thoracic cavity or lungs are correlated inversely; thus a decrease in impedance below a threshold can serve as a useful marker for volume overload,[37] but false-positive results have been a concern. Additional measures that can help in heart failure include measurement changes in average heart rate over time, heart rate variability, and patient activity levels.

Advancements in CIED Programming: Options and Best Programming

Rate Drop/Sudden Bradycardia Response

Rate drop or sudden bradycardia response is an algorithm designed to preempt the onset of asystole or marked bradycardia in patients with neurocardiogenic cardioinhibitory syncope. The algorithm detects a programmable drop in rate compared to the previous heart rate or R-R cycle and activates atrial or DDD pacing at a rate typically 20 beats higher than the detection rate for a programmable period, thus preventing recurrent syncope in these patients.

Advanced Sensor Technology

One major goal of modern rate-adaptive pacemakers is to mirror the chronotropic response of a normal sinus node by appropriately increasing the heart rate of patients with chronotropic incompetence in response to physical activity and emotional need.[38,39] Activity-based rate-adaptive sensors (i.e., vibration sensors) have been widely used since 1983. However, these have drawbacks[40,41] as a physiologic heart rate is modulated by complex autonomic inputs to the sinus node.

There remained a need for combining a fast-reacting, low-specificity, activity-based sensor with a high-specificity, slower-responding physiologic sensor to better simulate normal sinus rhythm.[41] Dual-sensor devices, commercially available over the last 2 decades, combine an accelerometer-based activity sensor and a minute ventilation–based physiologic sensor; they have shown promise in preserving physiologic rate response.[42] Recent data in CRT patients suggest that, in properly selected patients, activity and minute ventilation–based sensor technology to increase heart rates can be associated with improved survival.[43,44] Closed-loop stimulation (CLS), a sensor developed by Biotronik, determines the heart rate based on an autonomic response. This self-learning algorithm establishes heart rate by assessing changes in intracardiac impedance, a measure of right ventricular contractility, and the heart rate is regulated to various metabolic demands by this autonomic response.[45]

Avoidance of Right Ventricular Pacing

The negative impact of a high percentage of right ventricular pacing on LV function and ventricular synchrony is well known.[46] Minimizing right ventricular pacing in a patient with intact AV conduction is paramount. Several algorithms have been developed to promote intrinsic AV conduction and can be collectively labeled as *positive AV hysteresis*.[47] This term generally involves lengthening of AV delay to search for intrinsic conduction. The extended AV delay persists until there is lengthening of the PR interval beyond preset limits, at which point the originally programmed AV delay kicks in. Several versions of this algorithm are available with the goal to minimize adverse consequences from right ventricular pacing. Another algorithm is a mode switch algorithm; the pacemaker primarily works in an AAI(R) mode (i.e., no AV delay) but switches to a DDD(R) mode with a preset AV delay if there is evidence for AV block.[48]

Avoiding Inappropriate and Unnecessary Implantable Cardioverter-Defibrillator Shocks

Although ICDs can save lives, shocks can be painful, can cause psychological distress, and may be delivered when not necessary.[49,50] Shocks can be given when they are not necessary. Atrial fibrillation with rapid ventricular response, sinus tachycardia, atrial tachycardia or atrial flutter with rapid conduction, and other supraventricular tachycardias are the most common arrhythmias causing such inappropriate therapy.

Many ventricular arrhythmias will self-terminate. Intervening with antitachycardia pacing or shocks may not be necessary, but both may be effective for sustained ventricular tachycardia (and shocks for ventricular fibrillation). The development of antitachycardia pacing is a double-edged sword, however. While it can reduce the need for shocks, especially in primary prevention devices, it may be potentially proarrhythmic. Strategies to reduce inappropriate and unnecessary therapy using device programming rely on several factors, including keeping the lowest tachycardia detection zone rate cutoffs higher (unless ventricular tachycardia rates are known to be slow) and delaying the time to ICD therapy. Algorithms developed to discriminate supraventricular arrhythmias from ventricular tachycardia are adjunctive interventions.[49,51,52]

Optimal programming has taken on great importance in the management of patients with ICDs.[52] Ultimately, like all medical therapies, patients should be considered as individuals. Patients with known ventricular tachycardia will require individualized programming, and patients with more progressive heart failure also may benefit from considerations of lower rate and/or shorter detection times than patients with less severe heart failure. For instance, ventricular tachycardias at lower rates may be less well tolerated by a Class IV patient than the same-rate ventricular tachycardia in a Class II patient.

Revolutionary Technologies

Leadless Pacing

Despite advances in lead performance, the transvenous lead and device pocket remain a weak link, increasing the risk of infection, lead fracture, dislodgement, and serious vascular complications. Although lead extraction technology has advanced, risks of lead extraction remain and thus elimination of the lead altogether may be optimal. This has led to the development of percutaneously implanted leadless devices. Leadless pacemakers finally have become a reality and may soon have widespread impact.

At the time of this writing, two such systems, Nanostim from St. Jude Medical and Micra from Medtronic, have been studied. The Micra is a leadless, transcatheter, single-chamber ventricular pacemaker, which is hermetically sealed in a capsule (volume, 0.8 cm^3; length, 25.9 mm; and weight, 2.0 g). The capsule has splines at the end of it that help anchor the device to the right ventricular endocardium. Functionally, Micra is similar to a transvenous pacemaker, offering rate-responsive single-chamber pacing and auto-capture features. The device is connected to a steerable catheter and inserted through a 23 Fr femoral venous sheath. The Nanostim transcatheter device is similar to the Micra in that it is a fully contained device sealed in a capsule (length, 42 mm; diameter, 5.99 mm) with similar pacing capabilities, but it differs from Micra in having an active-fixation mechanism. Studies have confirmed their efficacy and safety, but long-term data are lacking.[53,54]

Subcutaneous Implantable Cardioverter-Defibrillator

The subcutaneous ICD is the first truly novel ICD technology. It consists of a pulse generator implanted in the subcutaneous axillary space and a solid-core lead with both sensing electrodes and the shocking coil, which is tunneled subcutaneously under the skin of the chest. The shocking coil is placed on the lead such that it is parallel to the sternum in the subcutaneous space and provides a shocking vector to the active can implanted into the midaxillary subcutaneous space. The data from the FDA investigational device exemption (IDE) trial and the European Effortless Registry are promising, showing that the device can be used with a high degree of confidence that appropriate arrhythmias will be terminated with ICD shocks and that the safety profile is encouraging.[55]

This device is a viable alternative for many patients, including primary prevention patients who simply need protection from sudden death and patients who are at high risk for the complications associated with transvenous leads. Developed originally by Cameron Health and specifically by Gust Bardy, this technology is currently manufactured and marketed by Boston Scientific. The device is compatible with dedicated bipolar pacemakers because it does not provide antibradycardia pacing capabilities. It also does not have antitachycardia pacing capability, although future capsule pacing systems being developed are likely to be compatible with the subcutaneous ICD and overcome this limitation.

TABLE 1.4 Main Modifications Introduced in MRI-Conditional Pacing Systems

Modification	Purpose
Reduction in ferromagnetic components	Reduce magnetic attraction and susceptibility artifacts
Replacement of reed switch by Hall sensor	Avoid unpredictable reed switch behavior
Lead coil design and insulation	Minimize lead heating and electrical current induction
Filter circuitry	Prevent damage to internal power supply
Dedicated pacemaker programming	Prevent inappropriate pacemaker inhibition Prevent competing rhythms

MRI, magnetic resonance imaging.
Modified from Ferreira AM, Costa F, Tralhao A, et al. MRI-conditional pacemakers: current perspectives. *Med Devices (Auckl)*. 2014;7:115–124.

MRI-Conditional Pacemakers and Implantable Cardioverter-Defibrillators

Magnetic resonance imaging (MRI)-conditional pacemakers and, more recently, ICD systems have become available. Until recently, an MRI scan has been contraindicated in patients with CIEDs. This has been secondary to various factors such as (1) movement and/or vibration of the pulse generator or leads; (2) temporary or permanent modification of device function; (3) inappropriate sensing, triggering, or activation of the device; (4) excessive heating of the leads; (5) induced currents in the leads; and (6) electromagnetic interference.

These limitations prompted a series of modifications in generator and lead engineering designed to minimize interactions that could compromise device function and patient safety (Table 1.4). MRI-conditional pacemakers were first introduced in 2008 and have demonstrated safety in the 1.5-T MRI environment, as well as similar lead and device performance when compared to non-MRI-conditional devices.[56]

On the other hand, a large and growing body of evidence exists on the safety of MRI scanning in patients with modern, non-MRI-conditional (not FDA labeled) pacemakers and ICDs. Contemporary devices have less ferromagnetic materials compared with devices manufactured before 2007. Investigators such as those at Johns Hopkins have suggested protocols to perform these procedures safely.[57]

Final Thoughts and an Eye Into the Future

The path to present CIEDs has been spectacular—and short. There have been many fits and starts and innovations that have not yet seen the light of day or have been abandoned. Technological advancements, a large number of which are not mentioned here owing to space constraints, have led to the present highly capable devices, leads, and ancillary equipment. Pacemakers have evolved remarkably. ICDs have also progressed rapidly. CRT implantation has been streamlined, and more options are now available. Other advances include smaller devices, better leads, greater device longevity, MRI compatibility, remote monitoring, and novel technology minimizing endovascular components, among others. What is next?

Tremendous technological improvements will likely occur over the next several years. We may not need leads at all. Next may be a totally leadless, fully capable, subcutaneous pacemaker/ICD/CRT system. Promising early work on using electromagnetic waves to wirelessly power implanted devices provides hope that rechargeable CIED batteries (used even in early CIEDs) may not be too far in the future.[58]

"Smart" CIEDs with better discrimination between ventricular tachycardia, supraventricular tachycardia, and noise may communicate with the patient's smartphone, download information, and allow programming from a distance. Perhaps new physiologic sensors will be able to better detect position and physiologic needs. Rate histograms and more sophisticated arrhythmia monitoring may be incorporated to predict death, ICD shocks, hospitalization, and poor outcomes.

Studies in rats using optogenetic technology, whereby adeno-associated virus 9 is used to express the channel rhodopsin-2 transgene at various sites in the rat ventricle, allowed pacing at different frequencies with blue-light illumination. Optogenetic technology may thus have great potential to modulate in vivo cardiac electrical activity and may have applications in cardiac resynchronization.[59]

Experimentation with biologic pacing may pave the way to solving problems of conduction system disease without the need for implantable hardware.[60] Regrowth of sinus and AV node cells or even His-Purkinje cells may represent the holy grail of cardiac pacing.[61]

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3

Surgical Techniques and Tools

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Introduction

Mastering surgical judgment and skills is paramount in providing patients with a safe and successful surgical procedure. This can be a challenge for both trainees and recent graduates from cardiac electrophysiology (EP) training programs. Several factors are responsible for these challenges. Basic anatomy and surgical training for medical students continues to be compressed into more abbreviated courses, while medical knowledge continues to grow at what seems to be an exponential rate. Electrophysiology fellows beginning their training are often far removed from the basic surgical training acquired in medical school. After completing an internal medicine residency and general cardiology fellowship, the individual may not have performed a surgical procedure for 5 years or more. Furthermore, upon entering an EP fellowship, his or her surgical training may be limited depending on the skillset of his or her mentors and the focus of the training program, especially with today's emphasis on ablations.

Acquiring surgical skills is easier when utilizing appropriate instruments and practicing techniques that are efficient and mechanically sound. When surgical skills turn into a repetition of mistakes or cumbersome or inaccurate maneuvers, they may require further steps or maneuvers to compensate and unnecessarily prolong the procedure and risk to the patient. Moreover, correction of poor technique requires substantially more effort than learning sound techniques from the beginning. This chapter provides an overview of common surgical instruments and their use, including sutures and needles, knot tying, suturing, and dressings. Such knowledge will assist the implanting physician to adapt his or her techniques based on individual patient needs and not rely on one single method.

Surgical Leadership

Control of the surgical procedure and patient safety require situational awareness and maintaining control of all activities and personnel present throughout the course of the procedure. Although the operating physician is ultimately responsible for the procedure's outcome, control through intimidation, albeit simpler than encouraging team participation, is not as safe or effective as leading by example. The successful operating physician will gain confidence and support from his or her team by instilling enthusiasm, enhancing each team member's performance, and emphasizing that his or her success depends on theirs. Careful attention to one's own behavior is critical to developing and keeping a loyal and efficient team. Personnel who are familiar with the implanting physician's personal preferences and idiosyncrasies and who are briefed on his or her plans for the procedure immediately beforehand will respond quickly when unexpected findings or complications are encountered during the course of the operation.

Surgical/Procedural Safety Checklist

The introduction of a safety checklist in the operating room/laboratory has its origins in the airline industry.[1] It is called by various names (Timeout, Huddle, Surgical Clinical Outcomes Assessment Program [SCOAP], etc.) in different institutions, but its purpose is the same: to make certain that one is operating on the right patient for the right procedure on the correct site for the right reason, and to ensure one's team and all present know each other and their roles.[2,3] In each step of the safety checklist, all music, conversation, and distractions are halted. Before induction of conscious sedation or general anesthesia, the patient's identity is confirmed by two personnel, the correct surgical site and side are marked if appropriate, a signed consent form is identified, and allergies are reviewed. Anesthesia equipment is documented to be present and fully functional. Airway/aspiration risk is assessed, the patient is positioned, and a plan to address deep vein thrombosis (DVT) and pulmonary embolus (PE) is confirmed. If the patient is at increased risk for the procedure, equipment sufficient to address the risk should be available and a plan for its use reviewed.

Once the patient is prepped and draped and before skin incision (see Chapter 6), a second confirmation of the patient, consent, surgical site, and procedure is performed and directed by the operating physician. Team members announce their names and roles, and personnel exchanges, if anticipated, are discussed. The anesthesia team reviews concerns, including special medications (insulin, β-blockers, etc.), relevant allergies, and issues that may affect recovery. The operating physician reviews a brief description of the procedure, anticipated difficulties, critical aspects of the procedure, and plans to address

them. The anticipated duration of the procedure, estimated blood loss, and special instruments needed are discussed, and essential imaging requirements are confirmed. Nursing or surgical assistant personnel confirm that the surgical prep has been completed, a fire safety risk assessment is complete, equipment is available and functional, and a management system for sharps (needles, knife blades, etc.) is in place. A process control led by the operating physician is performed to confirm that antibiotic prophylaxis has been administered within 60 minutes of procedural start time, a redosing plan is in place if the procedure is anticipated to exceed the standard dosing schedule, an active warming plan is in place, and serum glucose monitoring and an insulin protocol are initiated if applicable. Finally, all members are encouraged and expected to "speak up" and address any concerns they may have before or at any time during the course of the procedure.

Once the cardiac rhythm device procedure is complete and before wound closure, the operating physician should perform a visual and physical inspection of the generator pocket to confirm that the generator and lead(s) are appropriately positioned and there are no foreign objects (sponges, etc.) present. Fluoroscopy may also be used; however, it does not replace a visual and physical inspection. All conversation, music, and distractions are stopped while sponges, needles, and instruments are counted to confirm that everything is present and accounted for. Following wound closure, unexpected findings or equipment issues encountered during the procedure are addressed and a response plan is formulated. Key plans for recovery (β-blockers, pain management, insulin, anticoagulation, etc.) are reviewed, and a postoperative conversation is conducted with the patient's family. An example of such a checklist is illustrated in Fig. 3.1.

Instruments

Surgical instruments of importance to the physician who is implanting cardiac rhythm devices include those designed for cutting, grasping, retracting, clamping, and suturing.

Instruments for Cutting

The scalpel, consisting of a handle and a blade, is the most basic of cutting instruments and is synonymous with surgeons and the art of surgery. The most common handles come in two sizes, Bard Parker (BP) #3 (most common) and BP #4. Most modern scalpel handles have a locking mechanism for safe blade placement and removal (Fig. 3.2). The BP #3 handle can accommodate smaller blade sizes from #10 to #15, while the BP #4 handle is designed for larger blades and is not typically used in device implantation cases. Commonly used scalpel blades include #10, #11, and #15. Some operators may use a #12 blade; however, this is unusual. An illustration of scalpel blades is shown in Fig. 3.3. The #10 blade is similar in shape to the #15 blade but larger. Both are used to incise the skin and for sharp dissection. The #15 blade, being smaller, allows for more precise incisions. The #11 blade is pointed and is used primarily for making a press cut or stab incision. Device implanters typically use press cuts when cutting sutures from an anchoring sleeve on the lead. The #12 blade is hooked with the cutting edge on the concave portion of the blade. Its most common use in device implantation is cutting suture sleeves away from surrounding tissue without disturbing the anchoring sleeve. The scalpel handle may be held in two ways: a pencil grip for short, fine incisions or backhand cutting, and the fingertip grip, which allows for a maximum amount of cutting edge contact for greater control when making longer incisions (Fig. 3.4). When making an incision, the knife is drawn from left to right (right-handed surgeon) or from away toward the operator. When incising the skin, three points of tension are provided with the fingers laterally and the knife blade longitudinally to provide a stable cutting surface and allow precise control of cutting depth (Fig. 3.5). Additionally, the knife blade must be held perpendicular to the skin to avoid beveling (Fig. 3.6).

If the scalpel defines the surgeon, the scissors are arguably the most versatile of all the instruments. They may be used for tissue dissection, undermining skin to relieve tension when closing, cutting sutures or other surgical material, or spreading or opening tissue planes for such tasks as generator pocket formation, or as a blunt dissector if the blades are closed. There are two basic types of scissors, blunt tipped for dissection and sharp tipped for cutting. Curved blunt-tipped scissors such as Metzenbaum, McIndoe, or Mayo are designed for dissecting and have an advantage over other instruments as they allow for both blunt and sharp dissection without having to change instruments (Fig. 3.7). The curved blade also provides directional mobility, visibility, and flexibility in the angle of approach to the tissue, as well as allowing lifting and palpating of tissue compared to straight scissors. They are most effective when dissecting along tissue planes. Straight blunt-tipped scissors provide a greater mechanical advantage when dissecting through scar and tough tissue, but their use in device implantation is limited. Straight sharp-tipped scissors are typically used for cutting sutures or other ligatures (Fig. 3.8). They allow rapid and accurate positioning of the scissor tips before cutting. Regardless of the type of scissors used, pronation to neutral hand positions provides the greatest maneuverability of the instrument in all directions.

Instruments for Grasping

Forceps are nonlocking grasping tools that function as an extension of the thumb and opposing fingers in the assisting hand to augment the instrument in the operating hand. Their primary purpose is to grasp, retract, or stabilize tissue. They may also be used to pack or extract sponges, pass ligatures, and stabilize and manipulate needles during suturing. Forceps are categorized by the presence and type of teeth that are designed for the specific tissue they are intended to hold. Fine-toothed forceps such as DeBakey forceps are considered atraumatic and are designed for soft tissue and vessels (Fig. 3.9). They may be used on leads without damaging the insulation if used gently. Toothed forceps include those with teeth designed for dense tissue (Russian), with a single tooth on the end (rat-tooth), and

UW Medicine Surgical/Procedural Safety Checklist

1 Before Induction: (ALL MUSIC, CONVERSATION & DISTRACTIONS HALTED AT EACH STEP)

With Patient Confirm: ❑ Identity (2 identifiers) ❑ Site and side marked (or NA) ❑ Procedure ❑ Consent ❑ Allergies

Anesthesia Confirms:
- ❑ Anesthesia Machine ready
- ❑ Patient Position
- ❑ Airway/aspiration risk assessment completed
- ❑ Increased risk, needed equipment available, plan described
- ❑ DVT / PE chemoprophylaxis plan in place

2 Briefing – Prior to Skin Incision; * After Prep and Drape* (All Team Members)

- ❑ Team members announce name and role.
- ❑ Attending Surgeon, Anesthesia, Nursing/ Surgical Tech: Confirm Patient, Consent, Site & Procedure
- ❑ Personnel exchanges discussed (timing of and plan for announcing exchanges)
- ❑ Covering surgeon identified (if the Attending Surgeon needs to leave during the procedure)

Anesthesia Team Reviews

- ❑ Concerns (Airways, special meds (beta blockers) relevant allergies, conditions affecting recovery, etc)

Attending Surgeon Reviews

- ❑ Brief description of procedure, anticipated difficulties and critical event
- ❑ Expected duration of procedure;
- ❑ Expected blood loss
- ❑ Need for instruments/supplies/IV access beyond those normally used for the procedure
- ❑ Essential imaging displayed; right & left confirmed; has position changed and is marked site visible

Nursing / Surgical Tech Team Reviews

- ❑ Surgical prep completed
- ❑ Fire Safety Risk Assessment (e.g. skin prep dry, no pooling of prep solutions, laser precautions….)
- ❑ Equipment issues (e.g. instruments ready and trained on, requested implant available, gas tanks full)
- ❑ Sharps management plan reviewed
- ❑ Neptune/suction setting verified.

Process Control – Prior to Skin Incision (Surgeon Leads)

- ❑ Antibiotic prophylaxis given in last 60 minutes
- ❑ Active warming in place
- ❑ Glucose checked as required; Insulin protocol initiated if needed
- ❑ Re-dosing plan for antibiotics
- ❑ Specialty-specific checklist
- ❑ Other concerns (ALL MEMBERS EMPOWERED TO "SPEAK UP" AND ADDRESS CONCERNS)

3 Prior to closure

Attending Surgeon:
- ❑ Perform methodical visual & physical sweep of wound & report

Nursing:
- ❑ All music, conversation & distractions halted
- ❑ Circulator & Scrubs perform preliminary count of needles/sponges/instruments and report

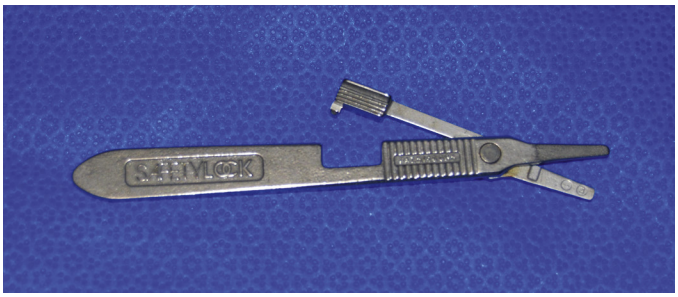
Attending Surgeon & Nursing:
- ❑ Confirm instrument, sponge, and needle count are correct

4 Debriefing, Care transition

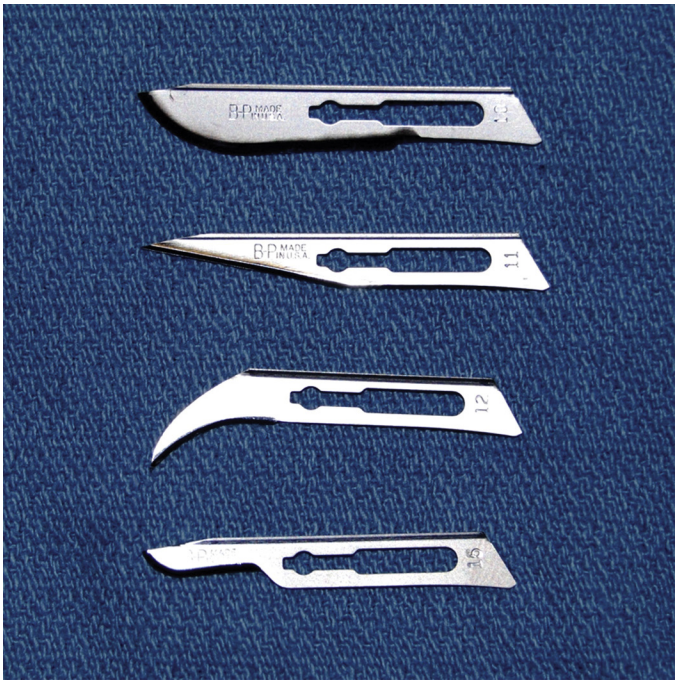
Attending Surgeon or Designee & Nursing:
- ❑ Confirm no retained objects to include visual inspection of sponges.
- ❑ Specimen, confirm label, laterality & instructions
- ❑ Confirm name of procedure
- ❑ Equipment or other issues to be addressed? Response plan formulated? (Who/When)
- ❑ Key concerns for recovery (plan for pain management, nausea/vomiting, insulin, anticoagulation)
- ❑ If no Foley: Bladder scan / I&O Cath arranged
- ❑ For patient on beta blocker, post-op plan formulated
- ❑ Discussion of family location and contact for surgeon post-op conversation

9/2014

• **Fig. 3.1** University of Washington safety checklist: Surgical Clinical Outcomes Assessment Program (SCOAP).



• **Fig. 3.2** Bard Parker #3 locking scalpel handle.



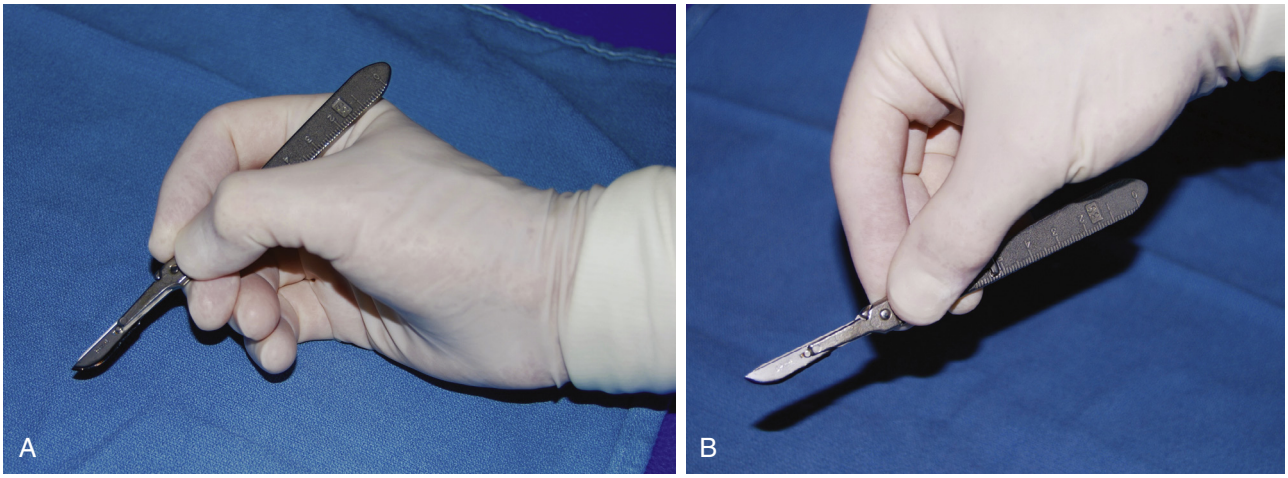
• **Fig. 3.3** Scalpel blades (top to bottom: #10, #11, #12, #15).

smaller versions designed for closing skin (Adson) (Fig. 3.10). Forceps with teeth should never be used to grasp a pacemaker or defibrillator lead as the teeth may compromise the integrity of its outer insulation. The choice of forceps should be dictated by the amount and nature of the tissue being handled. Forceps are held such that they become extensions of the thumb and index finger. A pencil-grip position provides the widest range of maneuverability. Holding the shank of the forceps in the palm of the hand significantly limits its use and requires extreme flexion of the wrist and should be avoided (Fig. 3.11).

Instruments for Retracting

A key component for safe, efficient surgery is good exposure. For that reason, a number of instruments have been designed for this purpose and fall into one of two categories: manual (held by the surgeon or assistant) and self-retaining. Good exposure requires thorough planning and thoughtful execution. If a procedure is particularly difficult, it is likely that inadequate exposure played a significant role.

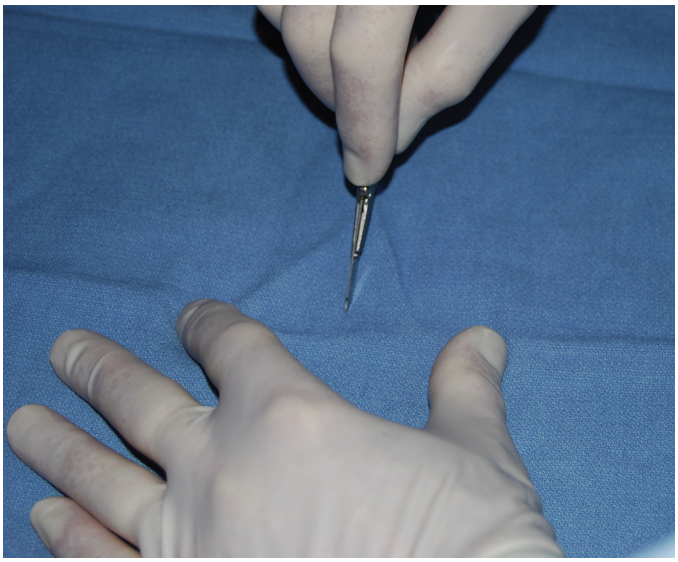
Skin hooks are manual retractors with one or more small, sharp hooks designed to hook into the dermis without trauma to the epidermis and are used primarily for wound retraction (Fig. 3.12). They are useful for undermining dermis to reduce or remove tissue tension from the skin closure, but must be used with extreme caution as they may inadvertently pierce the insulation of an indwelling lead if not positioned under direct visualization. The Senn retractor (Fig. 3.13) is an alternative to the skin hook and is preferred by many operators as it consists of a rake on one end and a right-angled flat blade on the other. For visualization while creating the pocket for the device generator and lead(s), manual retractors such as the Army-Navy, small Deaver, Richardson, and Goulet may be used (Fig. 3.14). These instruments can provide traction if one is employed and traction/countertraction if more than one is used. Manual retractors allow for dynamic tissue retraction as they may be continuously adjusted based on the needs of the surgeon. The specific retractor chosen for device implantation depends on tissue depth, the width of the incision, and surgeon preference.



• **Fig. 3.4** Holding the scalpel: (A) pencil grip; (B) fingertip grip.

Care must be taken to avoid traumatizing the tissues with overaggressive traction. It is preferable to have a variety of retractors on the surgical table or readily available to be able to adapt to various types of body habitus.

Self-retaining retractors such as the Weitlaner, cerebellar, and Gelpi (Fig. 3.15) provide both traction and countertraction with a locking mechanism located on the handle. This is advantageous as it provides long-term continuous exposure and frees the hands of the surgeon or assistant to perform other functions without losing exposure. For most patients, the Weitlaner is sufficient to provide exposure to the anterior fascia of the underlying muscle; however, in obese patients and those with large breasts, the cerebellar retractor with its angled arms can provide deeper exposure without impeding the surgeon's progress. The Gelpi retractor has single-pointed tips on the ends and is useful for small incisions such as those performed for tunneling a subcutaneous defibrillator lead (see Chapter 10). Care must be taken when using the Gelpi retractor as its pointed ends may damage the insulation of the lead or tear the surgeon's glove.



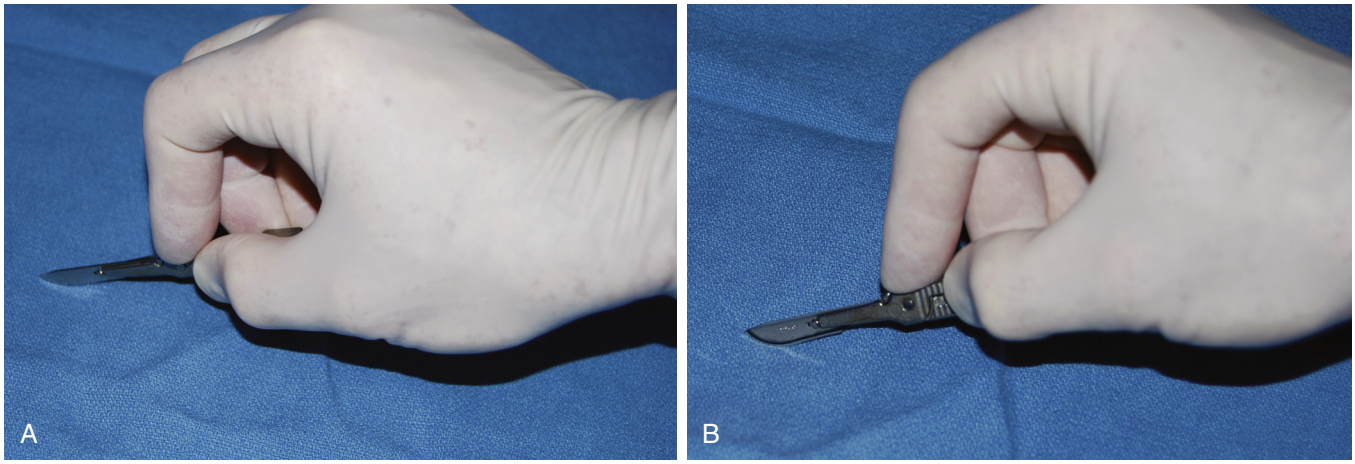
• **Fig. 3.5** Incising with three points of tension. The surgeon's free hand provides two points with the thumb and index finger, while the scalpel blade provides the third point. This provides an undistorted surface for the incision.

Instruments for Clamping

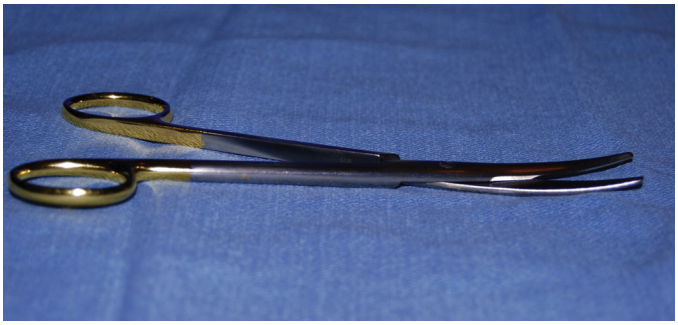
Clamps are useful tools for occluding, gripping, dissecting, and retracting. The most commonly encountered clamp for physicians implanting cardiac rhythm devices is the hemostat, also known as a "snap" or a "mosquito." It is a small ratchet-locking clamp designed to grasp or occlude blood vessels before cutting. It is also useful during dissection when bleeding is encountered. When used in this manner, care must be taken to clamp the culprit bleeding vessel and avoid clamping excessive surrounding tissue. Hemostats may be curved, be straight, or have a right angle at the tip (Fig. 3.16), which is advantageous for fine dissection around vessels such as the cephalic vein. Because clamps hold tissue and ligatures securely, they may also be used as retractors to maneuver layers of tissue or change the angle of a vessel such as the cephalic vein to provide easier access.

Instruments for Suturing

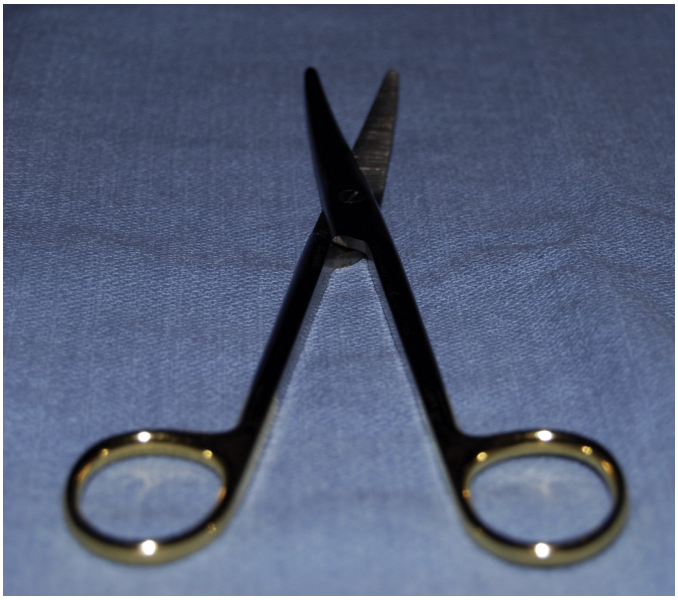
Instruments for holding and driving needles through tissue are referred to as either needle holders or needle drivers. The art of suturing is easier and more efficient when the needle holder is used correctly. Needle holders are available in different lengths with jaw sizes designed to accommodate needles of specific sizes and shapes (Fig. 3.17). Specialty needle holders such as the Gilles (Fig. 3.18) possess jaws to hold the needle and scissors designed to cut sutures built into a single instrument and are favored by plastic surgeons. The most common needle holders used by physicians implanting cardiac rhythm devices have a ratchet-locking mechanism designed to hold the needle firmly in place once positioned and either serrated or tungsten carbide–impregnated jaws to prevent needle slippage



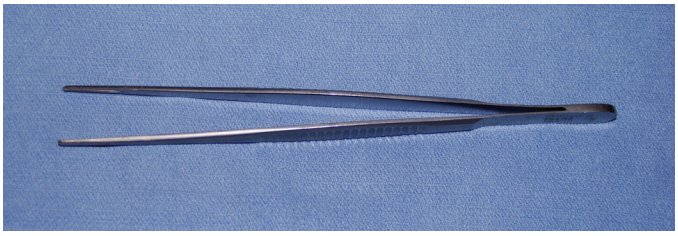
• **Fig. 3.6** Holding the knife blade: (A) 90 degrees perpendicular to the skin; (B) angle less than 90 degrees, resulting in beveled incision.



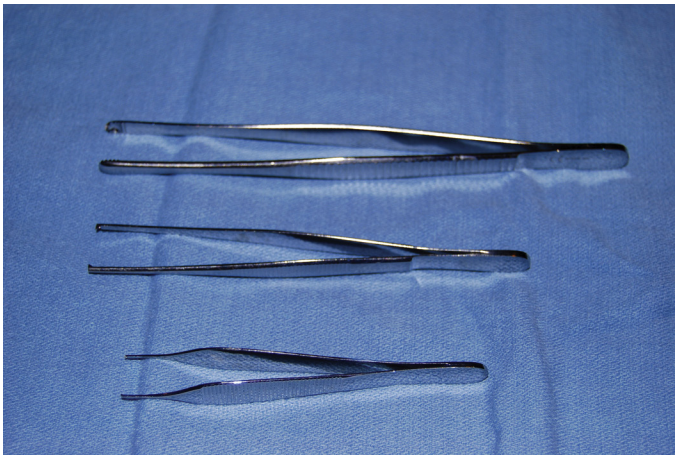
• **Fig. 3.7** Curved, blunt-tipped scissors for dissecting.



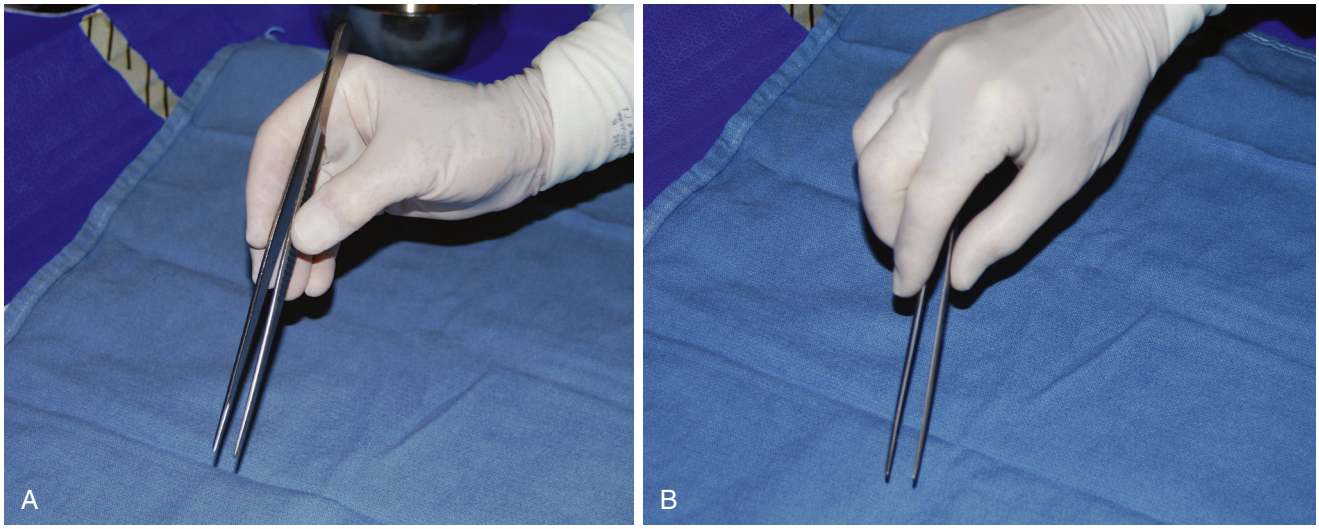
• **Fig. 3.8** Straight, sharp-tipped scissors for cutting.



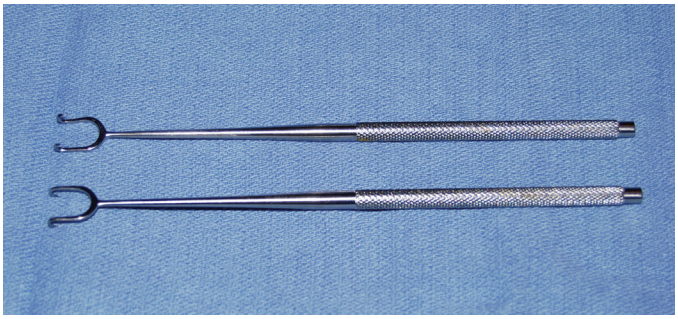
• **Fig. 3.9** DeBakey forceps.



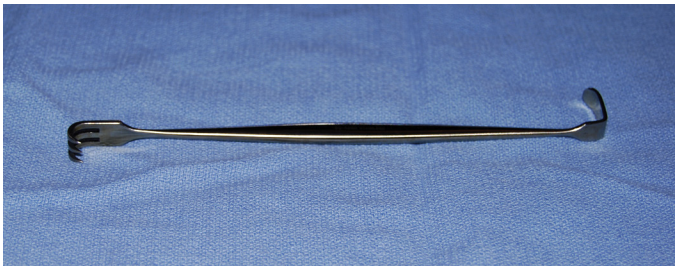
• **Fig. 3.10** Toothed forceps (top to bottom: Russian, rat-tooth, Adson).



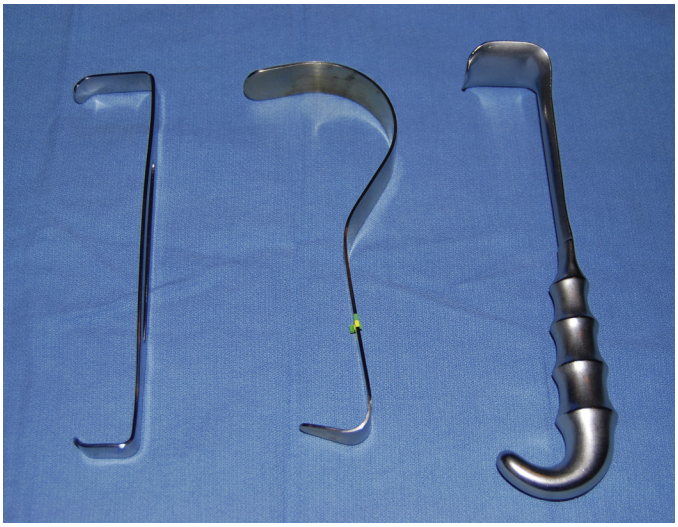
• **Fig. 3.11** Holding forceps: (A) pencil grip; (B) incorrect position, limiting use.



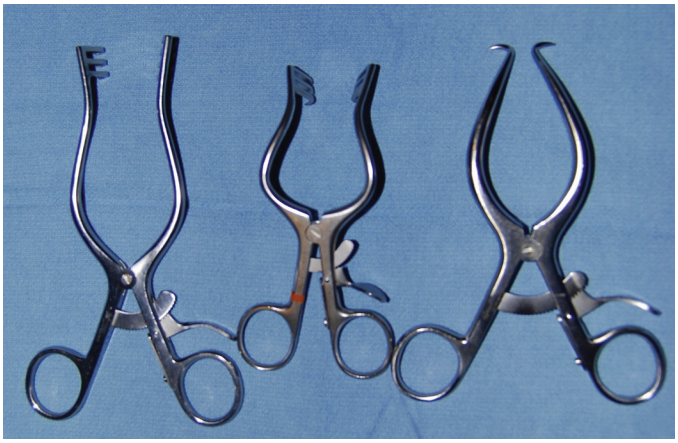
• **Fig. 3.12** Skin hooks.



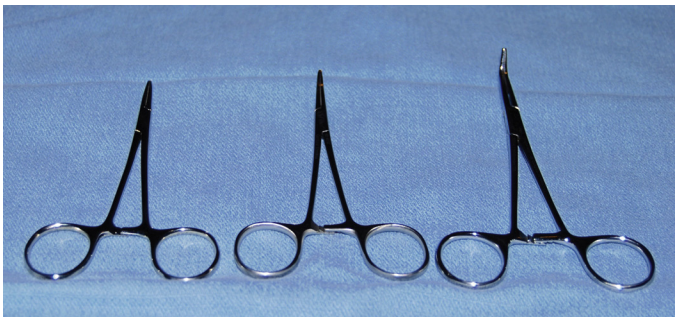
• **Fig. 3.13** Senn retractor.



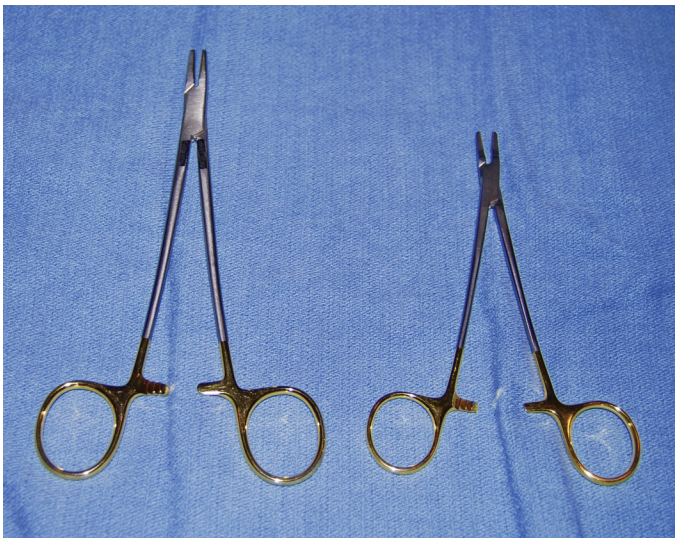
• **Fig. 3.14** Manual retractors (left to right: Army-Navy, small Deaver, Richardson).



• **Fig. 3.15** Self-retaining retractors (left to right: Weitlaner, cerebellar, Gelpi).



• **Fig. 3.16** Hemostats (left to right: curved, straight, right-angled).



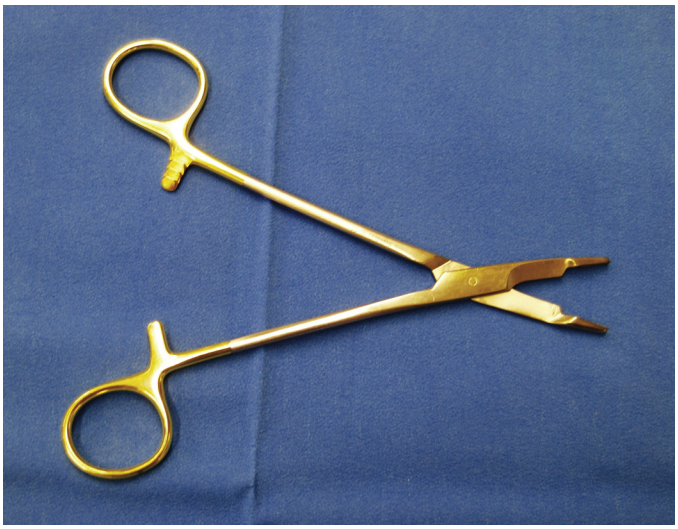
• **Fig. 3.17** Large and small needle drivers.

as it is driven into tissue (Fig. 3.19). The needle holder is positioned in the hand in a manner similar to that with scissors. Curved needles must be driven through tissue along its curved shape or the needle will bend or break. For forehand suturing, the needle point is directed toward the nondominant hand and pointing upward, and the forearm is supinated to drive the tip of the needle through tissue in a plane matching that of the curve of the needle. For backhand suturing, the direction of the needle is reversed in the needle holder and the forearm is pronated, again matching the curve of the needle.

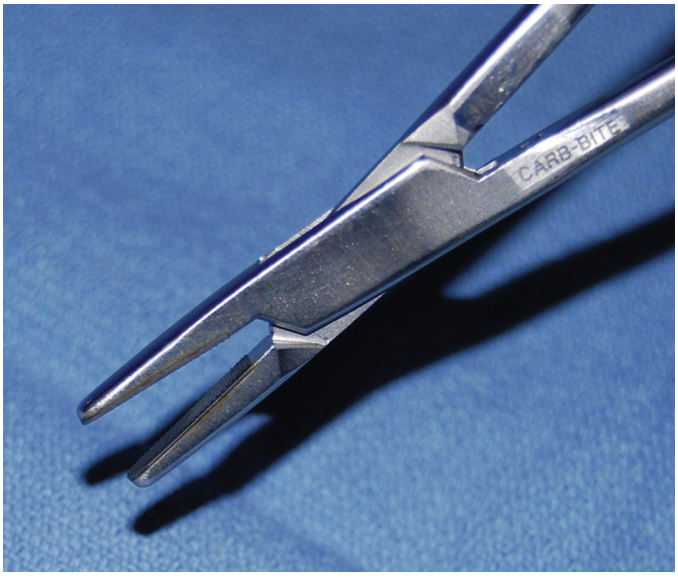
Electrocautery

Modern electrocautery represents a continuum from ancient medicine when heated metal was used to cauterize bleeding wounds in battle. Today, electrocautery is primarily used to cut tissue and coagulate bleeding vessels within open wounds. Several electrocautery manufacturers exist with units capable of different functions; however, the principles remain the same.

Electrocautery delivers alternating current in a sine wave pattern at frequencies from 500,000 to 3,000,000, far faster than

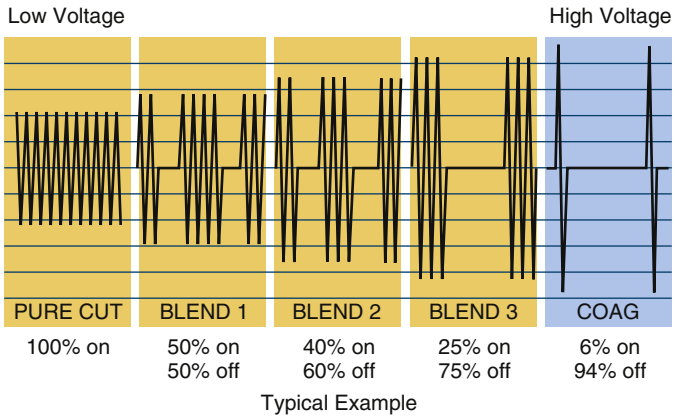


• **Fig. 3.18** Gilles needle driver.



• **Fig. 3.19** Carbide-tipped jaws of needle driver. These prevent needle from slipping or spinning when driven into tissue.

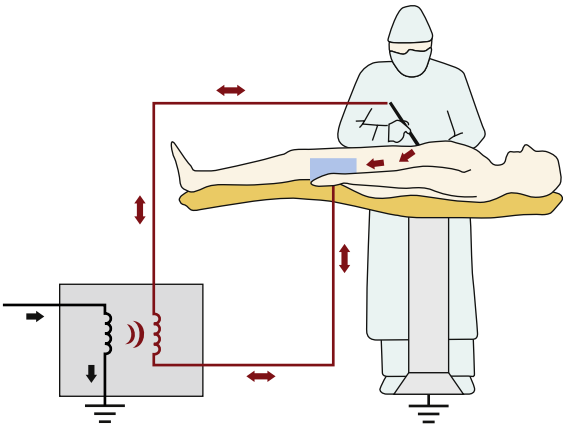
electrical current, which can stimulate muscles and nerves.[4] The energy is typically displayed in watts, with 35 to 45 W being the most commonly used. Three modes of energy are deliverable: cut, coagulation, and blend (Fig. 3.20). Cutting generates a continuous low-voltage current and generates more intense heat at the tip of the electrode, producing instantaneous steam in the cells in contact with the electrode, disrupting those cells before heat can be transmitted to deeper tissue. The intense heat produced within the cells causes them to vaporize, with subsequent smoke formation.[5] As a result, the cutting mode produces a localized cutting surface without significant thermal spread or formation of char on the tip of the electrode. While desirable for tissue dissection, this property can generate heat sufficient to melt a lead's polypropylene or silicone insulation; hence, it should not be used whenever dissecting near leads. The coagulation setting produces an intermittent (e.g., 6% on, 94% off) voltage current over a wider area than does the cut



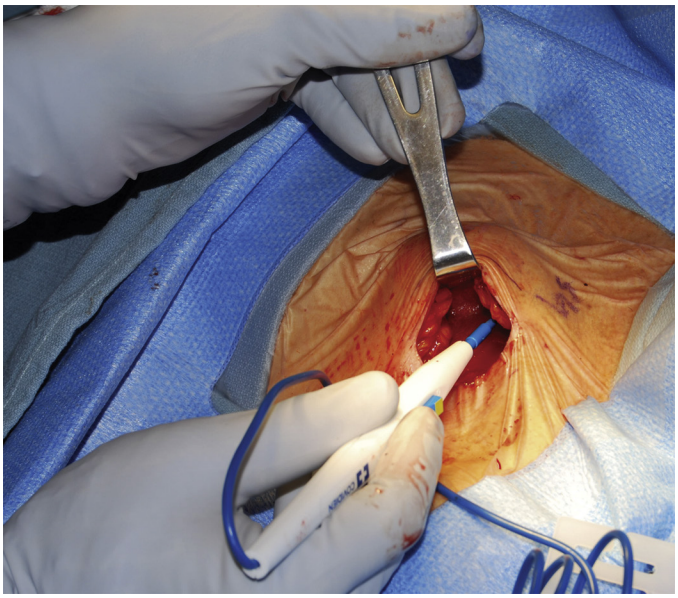
• **Fig. 3.20** Relationship of electrocautery energy settings. (From Massarweh NN, Cosgriff N, Slakey DP. Electrosurgery: history, principles, and current and future uses. *J Am Coll Surg.* 2006;202[3]:520–530.)

setting. This causes gradual loss of cellular water without steam disruption resulting in dehydration and coagulation. Because the current required is higher when it is intermittent, the tissue heats more slowly, resulting in broader thermal spread and char formation on both tissue and the electrode.[5] Coagulation current should never be applied to the skin owing to broad thermal spread. Paradoxically, coagulation is preferable if tissue dissection surrounding pacemaker or defibrillator leads is required, as thermal spread can help dissipate the amount of heat applied to the insulation. If electrocautery is used near a lead, the energy output should be decreased (e.g., 35 W down to 25 W) and applied parallel and never perpendicular to the lead to further avoid focal thermal injury. The best use for coagulation for the device-implanting physician is in tissue with poor conductivity, such as subcutaneous fat, where greater tissue penetration is beneficial. Some electrocautery units have the ability to blend both cut and coagulation by adding a 120-Hz ripple to the output power to combine the benefits of both settings. Combining more frequent current (cut) with higher output (coagulation) improves the electrode's ability to coagulate small vessels and maintain hemostasis during dissection. When applying current to a clamp or forceps holding a vessel, cutting or blend settings will cauterize the vessel more quickly than the coagulation setting.

Most cautery units are equipped to function with either monopolar or bipolar electrodes. Monopolar electrodes are the most commonly used in device implantation and other surgical settings, consisting of an electrode (pen) and an indifferent electrode (grounding pad) placed in contact with the patient's skin (Fig. 3.21). The electrode is activated by one of two buttons (cut, coagulation) on the pen's shaft. The pen is held like a pencil with the index finger free to push the appropriate button that activates the selected type of current and delivers it to the tissue in contact with the tip of the electrode (Fig. 3.22). Current is concentrated at the tip of the electrode and diminishes very quickly until it exits the patient into the grounding pad (indifferent electrode). The large size of the grounding pad dissipates heat to prevent skin burns; however, if the grounding pad becomes wrinkled or is applied over relatively avascular tissue, over hair, or near a metal prosthesis, the density of current



• **Fig. 3.21** Isolated electrocautery circuit. Energy enters from the electrode and exits from the indifferent electrode (grounding pad). (From Massarweh NN, Cosgriff N, Slakey DP. Electrosurgery: history, principles, and current and future uses. *J Am Coll Surg.* 2006;202[3]:520–530.)



• **Fig. 3.22** Electrocautery pen held like a pencil, with index finger access to both cut and coagulation buttons.

can increase significantly and result in skin burns. Bipolar electrodes eliminate the need for an indifferent electrode by incorporating each electrode onto one instrument, typically forceps, and they are activated by a foot switch. Bipolar electrodes are more effective in cauterizing in wet environments such as active bleeding or the presence of irrigation solutions or serous fluid because the electrodes are far closer together (1 mm) when current is applied. Unfortunately, this same property precludes their use as a cutting tool, which is why they are rarely used in device implantations.

The use of electrocautery provides unique challenges to the management of patients with pacemakers and defibrillators. In addition to insulation and/or lead damage, the device generator may sense the current as electromagnetic interference (EMI) inhibiting pacemaker output, which in a pacemaker-dependent patient can be dangerous, trigger the noise reversion mode (asynchronous pacing), alter the pacing

• **Fig. 3.23** Plasma knife (PEAK PlasmaBlade).

function (trigger mode switch), or, rarely, revert the patient's pacemaker generator back to preimplant settings (power-on reset). If the generator battery is at the end of life, application of electrocautery to the pacemaker generator can deplete the remaining battery power with abrupt cessation of pacing. For implantable defibrillators, application of electrocautery will have the same effects on pacing as with pacemakers, but also can result in inappropriate implantable cardioverter-defibrillator (ICD) shocks when EMI is detected falsely as ventricular fibrillation/tachycardia. The risk for these adverse effects of electrocautery diminish when electrocautery is used below the umbilicus and the pacemaker or ICD generator is in the upper chest.

Plasma Knife

While modern electrocautery has been utilized for decades, its use can be associated with thermal damage to tissues and potentially delayed wound healing. In an effort to minimize the amount of collateral damage from electrocautery, the PEAK (pulsed-electron avalanche knife) PlasmaBlade (Medtronic) was developed (Fig. 3.23). Unlike electrocautery, it uses brief, high-frequency pulses of radiofrequency energy to form electrical plasma alongside an insulated electrode. The working temperature remains between 40°C to 100°C, resulting theoretically in less thermal damage to tissue. In a porcine model, the PEAK PlasmaBlade reduced acute thermal injury depth 7- to 10-fold, decreased the inflammatory response, increased wound burst strength, and resulted in a superior scar.[6] More recently, Ruidiaz et al. confirmed decreased thermal injury depth, inflammatory response, and scar width in humans as well.[7] Moreover, the use of the PlasmaBlade has been shown to decrease the risk of lead damage at the time of device replacement, was associated with shorter hospital stays, and was cost effective.[8] This technology is promising, and further research will be needed to fully ascertain its utility.

Suture Materials and Needles

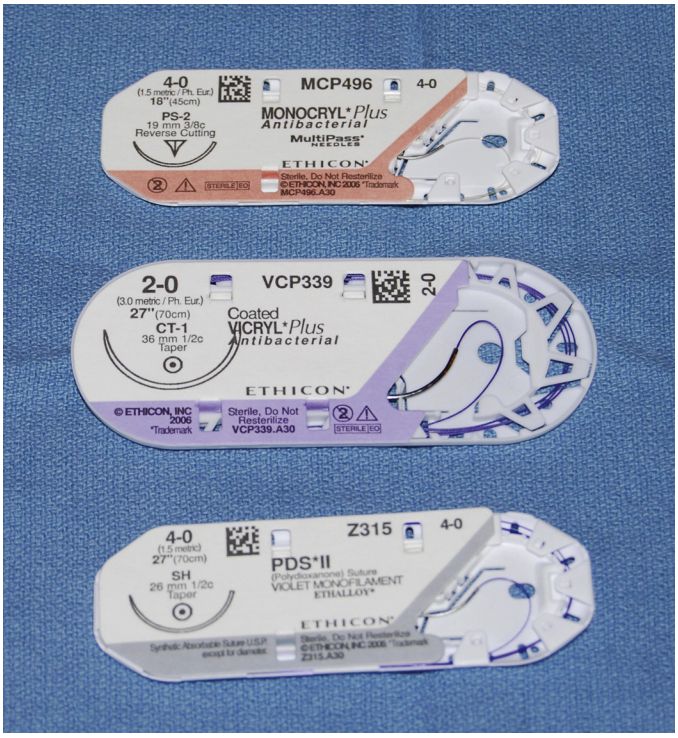
Suture Characteristics

Many sutures exist to help close incisions; these can be distinguished by certain unique characteristics. For example, sutures will differ based on tensile strength, or its ability to resist breakage. Elasticity refers to a suture's ability to contract back to its original length after deformation. Memory relates to a suture's tendency to retain its shape after deformation. Finally, sutures will differ in their coefficient of friction, or resistance to passing through tissue. Tissue reactivity is yet another characteristic that plays into choice of suture. There is no single ideal suture; rather, the appropriate suture can be selected for its desired purpose based on these characteristics.

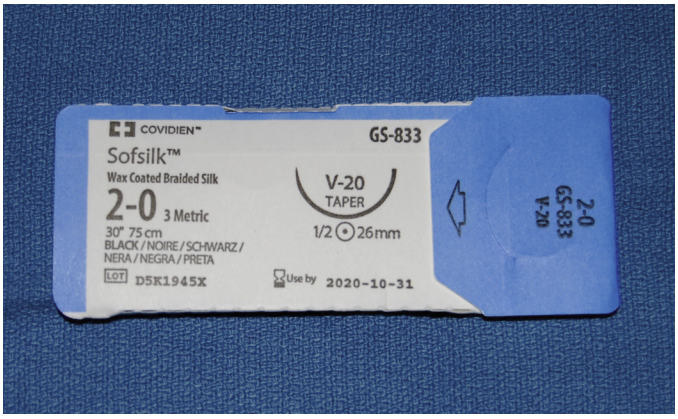
Suture sizes are defined by the United States Pharmacopeia (USP). This classification is based on tensile strength, with #5 possessing the greatest tensile strength, decreasing down to #0, and then further decreasing from 2-0 all the way down to 11-0, which has the lowest tensile strength and is typically used in fine ophthalmologic applications. In practicality, the majority of sutures used in wound closure from a cardiology perspective would include #0, 2-0, 3-0, and 4-0.

Sutures can also be subdivided based on the materials used to make them. In general, sutures can be either monofilament or braided. Monofilament suture, as its name implies, is composed of a single filament of suture that thus passes through tissue easily and has a low coefficient of friction. As it has less surface area and is smoother than braided sutures, it is particularly useful in potentially contaminated settings. It also induces less tissue reaction. Braided suture, on the other hand, consists of multiple filaments of sutures weaved together. It has a greater coefficient of friction and incites a greater inflammatory response. However, it requires fewer throws to make a secure knot than monofilament sutures.

Another classification for sutures refers to their permanence: they can be either "absorbable" or "nonabsorbable." Absorbable sutures are degraded by the body and lose their structural integrity with time. The half-life of a suture refers to the time it takes for an absorbable suture to decrease its tensile strength by half in vivo. The longer the half-life, the longer the suture remains around. Absorbable sutures have traditionally been divided into gut sutures or synthetic sutures. Gut sutures are typically derived from sheep intestinal submucosa or beef intestinal serosa, and are composed mainly of purified collagen. Examples include plain gut, which rapidly loses its tensile strength in 7 to 10 days, and chromic gut, which is treated with chromium salts to prolong degradation. Gut sutures are degraded by enzymatic processes, whereas synthetic absorbable sutures are broken down by hydrolysis. Examples of commonly used synthetic absorbable sutures include poliglecaprone (Monocryl), polyglactin (Vicryl), and polydioxanone (PDS) (Fig. 3.24). All three types can be used in a layered closure for deep wounds, including PDS or Maxon for Scarpa fascia, 3-0 Vicryl for deep dermal sutures, and a continuous running subcuticular Monocryl suture. Nonabsorbable sutures are divided by the USP into three different classes: class I, silk; class II, cotton derived; and class III, stainless steel. The gold standard remains silk, which has superior

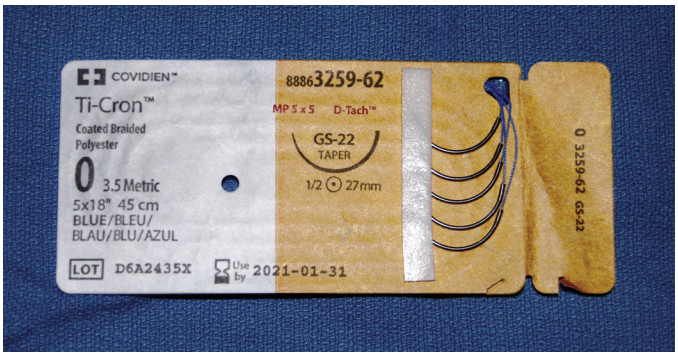


• **Fig. 3.24** Synthetic absorbable suture (top to bottom: poliglecaprone [Monocryl], polyglactin [Vicryl], and polydioxanone [PDS]).



• **Fig. 3.25** Silk suture.

handling characteristics (Fig. 3.25). Silk suture is composed of an organic protein (fibroin) derived from the silkworm species *Bombyx mori*. Because it is an animal protein, it may produce a transitory local inflammation or incite an allergic response in patients with known sensitivities to silk products.[9] It is typically braided and dyed black for ease of visibility in tissues. It is also commonly used by electrophysiologists for securing devices owing to its ease of handling. Although classified as nonabsorbable, silk sutures have been shown in vivo to lose their tensile strength with time, and at 1 year will have lost almost all its tensile strength. After 2 years, it may not even be detectable in tissue. Ti-Cron (Medtronic/Covidien) is a synthetic (coated polyester) braided suture with handling properties very similar to silk, and is preferred by many electrophysiologists as it does not lose its tensile strength and is hypoallergenic (Fig. 3.26).



• **Fig. 3.26** Synthetic (coated polyester) braided suture (Ti-Cron; Medtronic/Covidien).

Suture Needles

Just as there are many different types of sutures available, there are different types of needles as well. Two broad classes are cutting needles and tapered needles. In general, cutting needles are designed to cut through skin and thickened, tough connective dermal tissue. Cutting needles can further be divided into conventional cutting, which have a third cutting edge on the inner curve of the needle, and reverse cutting, which have a third cutting edge on the outer curve of the needle. Reverse-cutting needles are designed to minimize the risk of tissue cutout while adding strength to the needle. In contrast, tapered needles are designed to pierce and separate tissue without cutting it. The needle point for tapered needles is sharp and then transitions into an oval or rectangle in the body of the needle. It is typically used for internal soft tissue including viscera, blood vessels, and subcutaneous layers.

Needle size is determined by its various dimensions. These include needle length, the distance from point to end, and chord length, the straight-line distance from point to swage. The radius is the distance from the center of a circle formed by the curve of the needle to the body of the needle. Diameter refers to the actual thickness of the needle itself. Fig. 3.27 illustrates the various types of needles available, while Fig. 3.28 illustrates a typical suture package with category of use, size, characteristics, names, generic names, number and length of strands, needle description, and manufacturer.

Hemostasis

Meticulous hemostasis is critical to a procedure's success; careless hemostasis can lead to postoperative hematomas, which can be a potential nidus for infection. The key to hemostasis is recognizing the more substantial blood vessels before dividing them, so they can be safely cauterized or ligated before puncturing or transecting them inadvertently. Before incision, local anesthetic with dilute epinephrine may be used to decrease the amount of bleeding along the incision line. Pressure and/or packing remains effective for small amounts of venous ooze. Electrocautery remains the most common means to achieve hemostasis and is typically all that is required. However, topical agents can be used as adjuncts to help with hemostasis if needed.

Currently there is a wide variety of topical agents that exist for hemostasis, and they are particularly useful on raw surfaces with diffuse ooze, which does not respond well to conventional electrocautery. They can be classified into four categories: mechanical barrier agents, biologically active agents, flowable sealants, and fibrin sealants.[11]

The mechanical barrier agents achieve hemostasis by physically occluding blood flow and relying on the patient's own coagulation system to function. Examples include porcine gelatin, bovine collagen, oxidized regenerated cellulose (Surgicel), and polysaccharide spheres. Gelfoam is an example of a gelatin sponge, which is a water-insoluble, nonelastic product that can absorb up to 45 times its weight in blood. It enhances clot formation and provides a matrix to give structural support to forming clot. The collagen agents activate platelets locally to generate a platelet plug and favor the formation of fibrin (Helistat, Avitene).

In contrast, biologically active agents are thrombin derivatives that bypass the majority of the patient's coagulation cascade, enabling direct activation of fibrinogen to fibrin. These exist in liquid or spray forms or can be combined with other agents such as gelatin or fibrinogen (fibrin sealant). Flowable sealants consist of thrombin mixed with other hemostatic agents to form a more viscous, pastelike consistency, which can flow and cover irregular surfaces with crevasses that can be difficult for mechanical barrier agents to reach. An example of this would be Floseal. Fibrin sealants combine high concentrations of fibrinogen with thrombin immediately before use, often through a dual-syringe system. They exist in both liquid and aerosolized form (Tisseel), as well as absorbable patches.

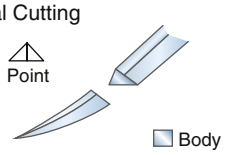
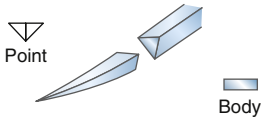
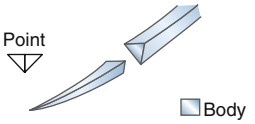
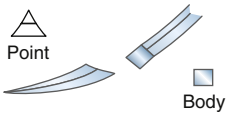
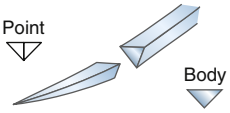
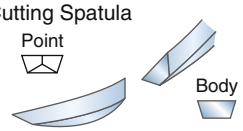
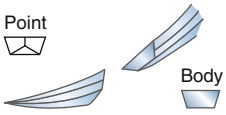
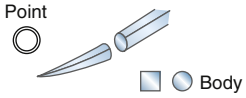
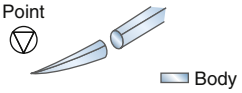
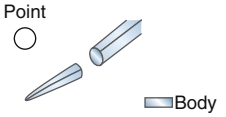
Although many hemostatic products exist, there is very limited data advocating one product over another. Consideration should be given to whether a patient has an intact coagulation cascade or not, as the mechanical barrier agents would not function well in those with coagulation defects.

Knot Tying

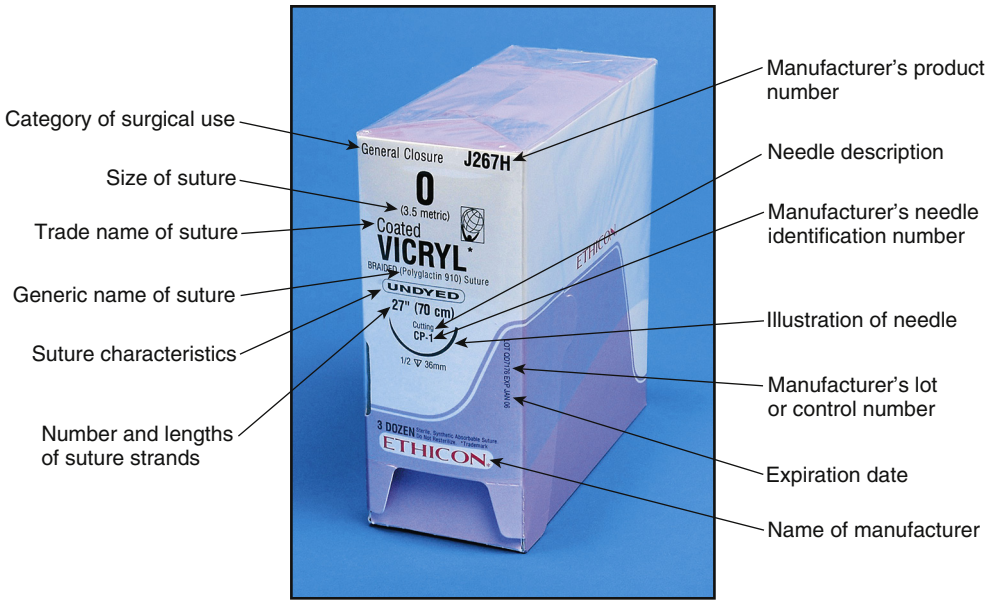
No matter how accurately a suture is placed, it will be useless if a secure knot is not placed correctly to hold the tissues in approximation. Knots can be tied either one or two handed, or with the use of a needle driver, as is the case with instrument ties. Instrument ties can be useful in tight spaces, such as the mouth or nose, or when working with very fine sutures, as in microsurgery. However, any method is acceptable provided a secure knot is formed.

Knots

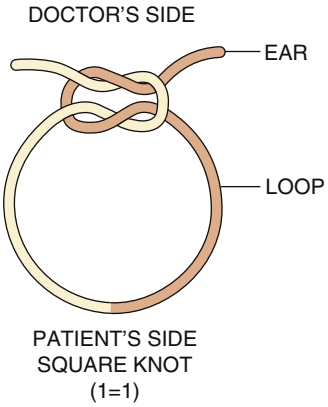
The standard knot used in surgery is a square knot, also known as a reef knot (Fig. 3.29). Historically, this knot was a binding knot used to secure a rope around an object. One throw is laid down with the right strand brought over the left, and then a subsequent throw is performed with the left strand going over the right. It must be secured in place with one additional throw, with right again crossing over left.

SHAPE	APPLICATION
Conventional Cutting 	skin, sternum
Reverse Cutting 	fascia, ligament, nasal cavity, oral mucosa, pharynx, skin, tendon sheath
Precision Point Cutting 	skin (plastic or cosmetic)
PC PRIME* Needle 	skin (plastic or cosmetic)
MICRO-POINT* Reverse Cutting Needle 	eye
Side-Cutting Spatula 	eye (primary application), microsurgery, ophthalmic (reconstructive)
CS ULTIMA* Ophthalmic Needle 	eye (primary application)
Taper 	aponeurosis, biliary tract, dura, fascia, gastrointestinal tract, laparoscopy, muscle, myocardium, nerve, peritoneum, pleura, subcutaneous fat, urogenital tract, vessels, valve
TAPERCUT* Surgical Needle 	bronchus, calcified tissue, fascia, laparoscopy, ligament, nasal cavity, oral cavity, ovary, perichondrium, periosteum, pharynx, sternum, tendon, trachea, uterus, valve, vessels (sclerotic)
Blunt 	blunt dissection (friable tissue), cervix (ligating incompetent cervix), fascia, intestine, kidney, liver, spleen

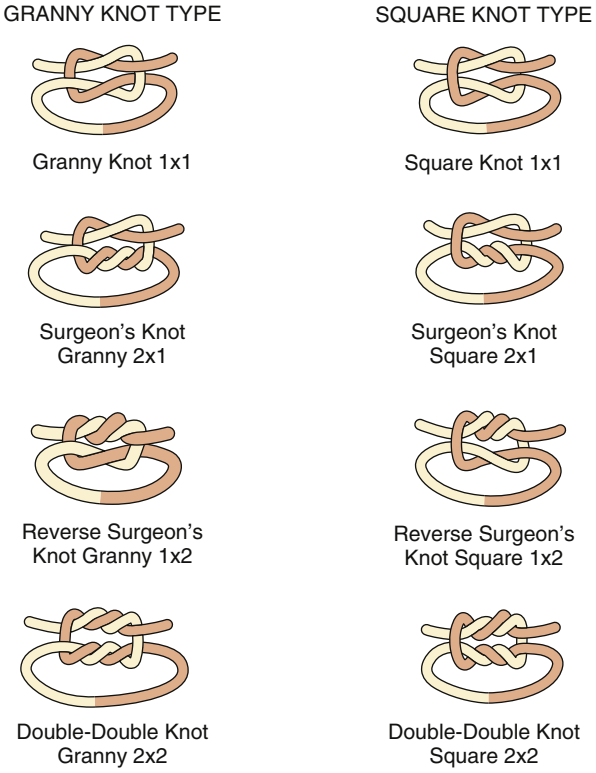
• **Fig. 3.27** Needle shapes and structure. (Courtesy Ethicon.)



• **Fig. 3.28** Typical suture package including category of use, size, characteristics, names, generic names, number and length of strands, needle description, and manufacturer. (From Pieknik R. *Suture and Surgical Hemostasis: A Pocket Guide*. Philadelphia: Elsevier; 2006.)



• **Fig. 3.29** Square knot. (From Pieknik R. *Suture and Surgical Hemostasis: A Pocket Guide*. Philadelphia: Elsevier; 2006.)



• **Fig. 3.30** Granny knot. (From Pieknik R. *Suture and Surgical Hemostasis: A Pocket Guide*. Philadelphia: Elsevier; 2006.)

A granny knot, also known as a slip knot, is started the same way as a square knot, with a right-over-left throw (Fig. 3.30). However, the second throw is a repeat of the first, where the right-hand strand again is brought over the left-hand strand. The final throw brings the left-hand strand over the right. The granny knot is not as secure as a square knot.

The surgeon's knot is useful in situations of high tension (Fig. 3.31). On the first throw of a surgeon's knot, the right strand is passed twice through the loop formed by the left, and then the second throw is completed like a square knot. When the first throw is tied down, it has less tendency to slip than a single half-hitch.

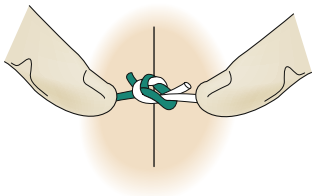
Suturing and Wound Closure

A comprehensive guide to suturing is beyond the scope of this chapter; becoming facile at suturing takes years of experience practicing and honing skills. However, many of the important principles will be reviewed.

Body Mechanics

The operator should be in a comfortable position in the operating room. Feet should be resting slightly wider than shoulder width. The spine should be erect, shoulders should be relaxed, and the lower spine's natural curvature should be maintained. The table height should be adjusted at a position that feels

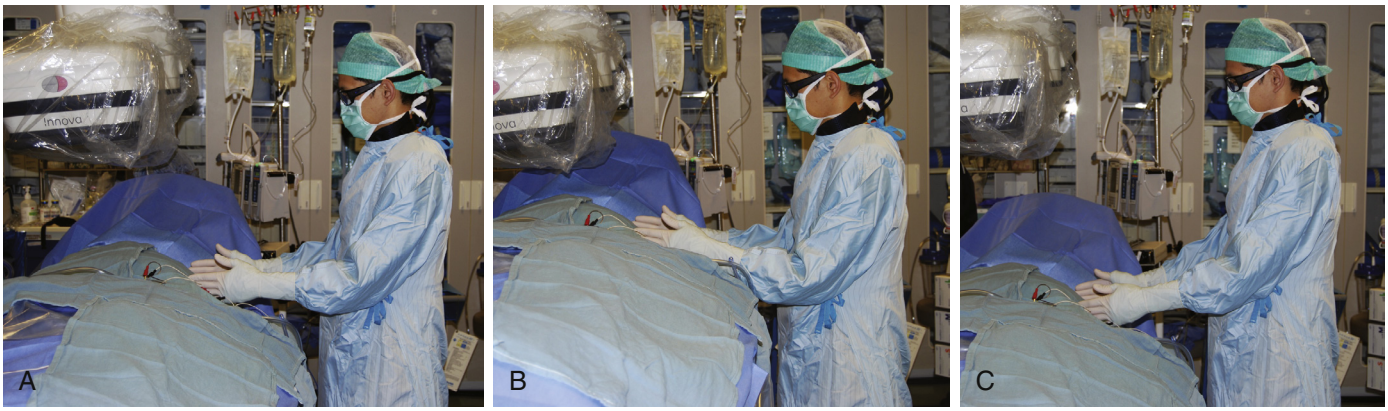
natural for the surgeon, typically around elbow height (Fig. 3.32). The table should be at a distance at which visualization of the surgical field is easy and without strain. Operating room lights should be adjusted to focus the maximum brightness on the surgical field and positioned in a manner to prevent the operator from casting his or her shadow over the surgical field (Fig. 3.33).



• **Fig. 3.31** Surgeon's knot. (From Pieknik R. *Suture and Surgical Hemostasis: A Pocket Guide*. Philadelphia: Elsevier; 2006.)

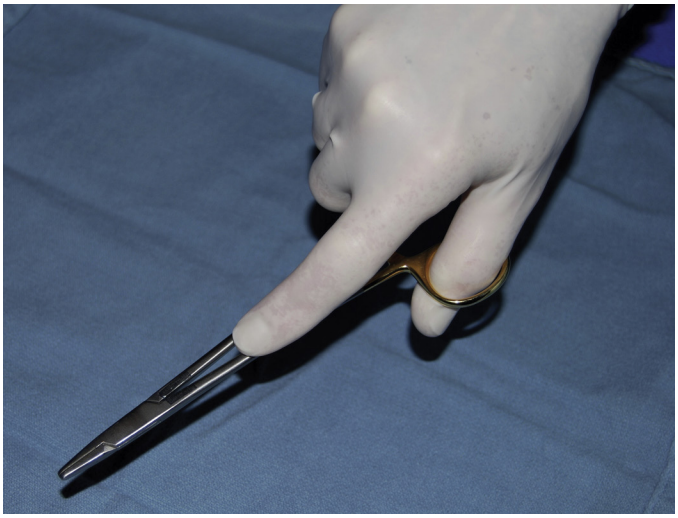
Holding the Needle Driver

The needle holder should be held comfortably in the hand. The thumb rests gently on the edge of one of the rings; the ring finger passes through the other ring. The index and middle fingers stabilize the shaft of the needle driver (Fig. 3.34). An alternative method of holding the driver is "palming" it, where the thumb does not go through a ring but rather rests on the shaft itself (Fig. 3.35). Likewise, the ring finger doesn't pass through the ring, but wraps around it with the middle and fifth digit. When palming, the thenar eminence is used to engage and disengage the ratchet mechanism of the needle driver. Either method is acceptable, but one should feel comfortable enough that it becomes second nature.

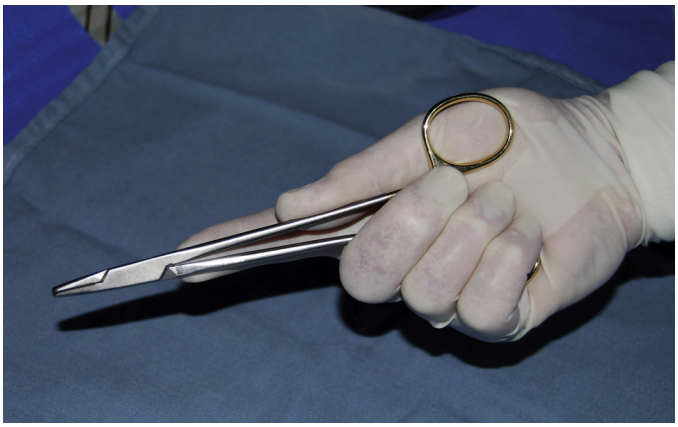


• **Fig. 3.32** Table height adjusted to surgeon. (A) Proper height with forearms parallel to ground; (B) table too high, resulting in fatigue to surgeon's arms and hands; (C) table too low, risking inadvertent contamination.

• **Fig. 3.33** Operating room light position. (A) Correct placement with two sources; (B) incorrect placement with surgeon obstructing light and creating a shadow over the surgical field.



• **Fig. 3.34** Needle holder in surgeon's hand. The distal thumb rests in one of the rings of the needle driver while the ring finger passes through the other. Note that the index finger stabilizes the shaft of the needle driver.



• **Fig. 3.35** "Palming" the needle holder. The thumb does not go through a ring but rather rests on the shaft itself. The thenar eminence is used to engage and disengage the ratchet.

Loading the Needle

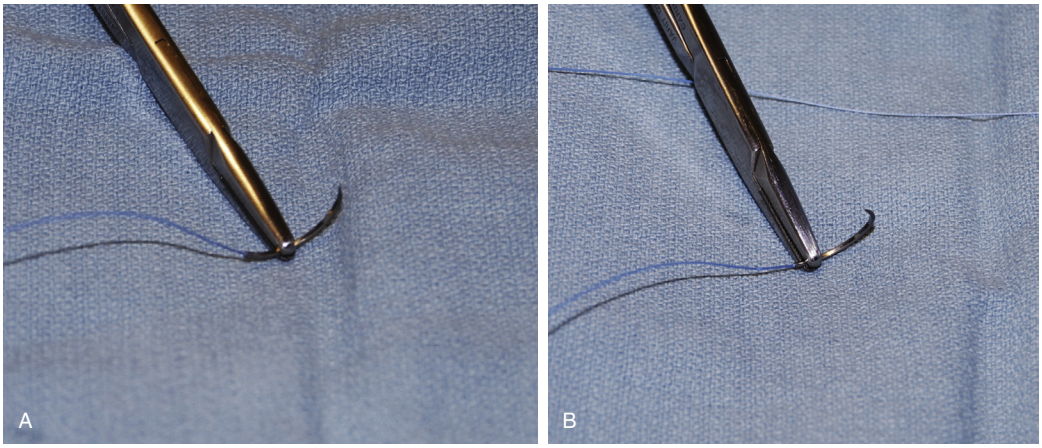
The needle should be grasped with the tip of the needle driver. The needle should be held approximately at the junction of its anterior two-thirds and posterior one-third. If the needle is grasped near its attachment with the suture (the "swage"), the suture may release from the needle unintentionally (Fig. 3.36). The needle should be at 90 degrees with the needle driver shaft, or even angled slightly away from the surgeon, as this is more ergonomic.

Suturing

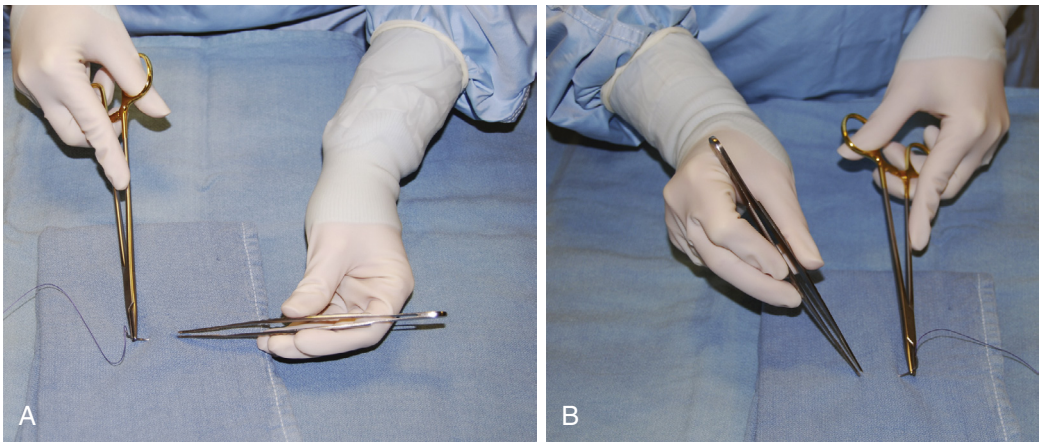
Sutures can be placed forehand or backhand, depending on the intended direction of the needle. This correlates with pronation and supination of the forearm, respectively (Fig. 3.37). Care should be taken to enter the tissue perpendicularly and to follow the curve of the needle to minimize trauma to the surrounding tissues. The needle should pass through the tissue easily and without resistance. The opposite hand should be in use, gently retracting the tissue to make passage of the needle easier. It is useful to have a fine-toothed retractor like Adson forceps in the opposite hand when working on skin, or rat-tooth forceps in deeper areas. Both hands should always be in use and constantly working in concert with each other (Fig. 3.38). Once the suture has been placed, the needle can be grasped with either the forceps or the needle driver, but care must be taken not to grasp the needle at its tip, which can blunt or deform it. When removing the needle from the tissue, care must be taken to follow the curve of the needle. After a secure knot is tied, suture scissors are used to cut the suture. The scissors should be held similarly to the needle driver. Braided sutures can be cut closer to the knot than monofilament sutures.

Common Suturing Techniques

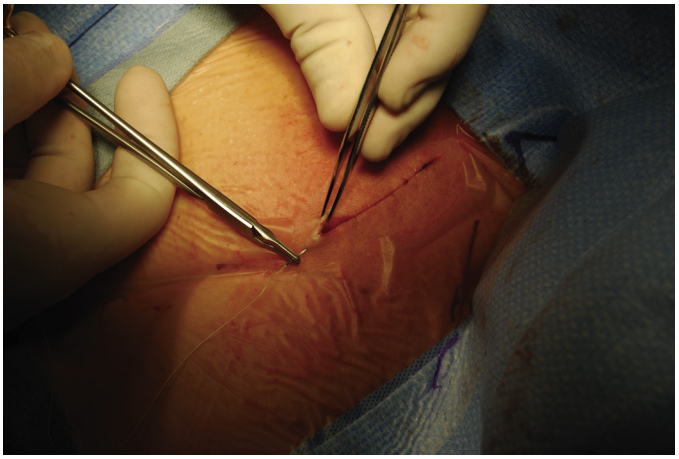
Perhaps the simplest technique is the simple interrupted suture, in which the needle is passed through one edge of the incision, across the wound, and through the other edge of the incision.



• **Fig. 3.36** Suture needle loaded on needle holder. (A) Proper location; (B) too close to swage, which may cause the suture to unintentionally release from the needle.



• **Fig. 3.37** Forearm movement during application of suture needle into tissue. (A) Pronation in forehand stitch; (B) supination in backhand stitch.



• **Fig. 3.38** The "two-handed" surgeon. The right hand directs the needle through the tissue while the left hand gently retracts the tissue with forceps to make passage of the needle easier.

A knot is then tied, typically with three to four throws (Fig. 3.39). This can be used for skin with an external nonabsorbable monofilament stitch like nylon; the suture can be removed in 7 to 10 days. Another example would be a buried internal suture through dermis (deep dermal) with an absorbable suture like Vicryl.

Unlike the simple interrupted suture, which requires one pass of the needle across the incision, vertical and horizontal mattress sutures require two passes of the needle—one across and one back (Fig. 3.40). The vertical mattress consists of a wider pass across the incision (far-far), and then one back that is closer to the wound edges (near-near). This allows precise apposition of the skin edges with good eversion. In contrast, the horizontal mattress utilizes one pass of the needle across, and then another equal pass back adjacent to the previous pass. This, in effect, allows a broader length of incision to be approximated with a single suture. It is useful for areas where tissue quality is poor and prone to tearing, as well as areas of high tension. If tied too tight, it can lead to tissue ischemia.

A continuous suture technique uses a single suture to run the length of the incision (Fig. 3.41). At the skin, a simple interrupted suture is placed, but rather than cutting the suture, multiple passes of the needle in the same direction across the incision are made until the length of the incision is covered. A knot is then tied at the end of the incision. If this suture is done at the skin level, sutures should be removed within 7 to 10 days to avoid track marks from the sutures. Another form of continuous suture is the continuous subcuticular suture, where a single suture is passed through the superficial dermis in a direction parallel to the incision (Fig. 3.42). A similar bite is then taken on the opposite side, backtracking slightly before placing the bite. Multiple passes are then made, alternating sides, until the length of the incision is covered. If these passes are made through a constant depth along the length of the incision, the opposite skin edges will be level and good edge-to-edge apposition will be obtained.

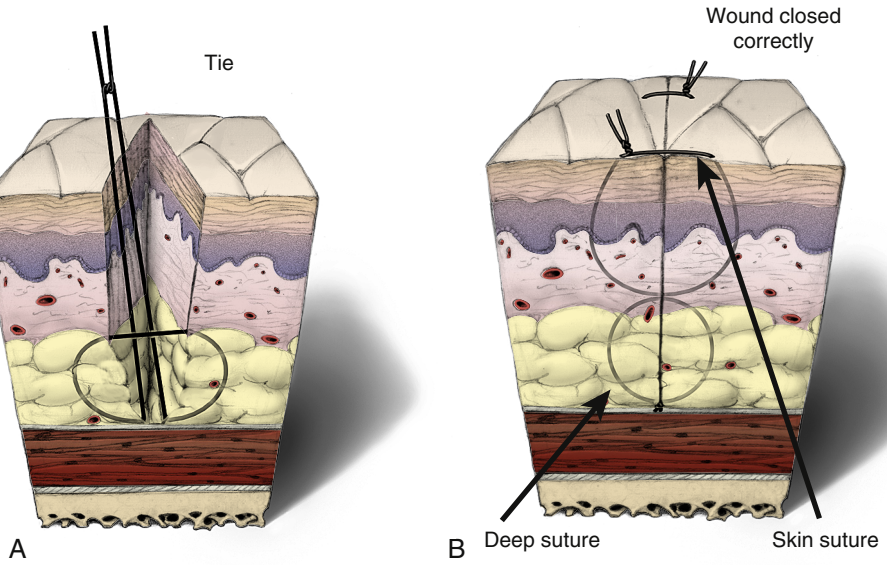
Staples can also be used in lieu of a subcuticular or superficial closure (Fig. 3.43). Good eversion is typically obtained; however, railroad track marks can commonly occur. This can be minimized by removing the staples early, within 5 days. As with any superficial closure, the healing tends to be better if a deep dermal layer of sutures is used in conjunction with the staples.

For rare incisions under minimal tension, surgical glue (Dermabond) can be used to achieve edge apposition (Fig. 3.44). It can be used along with deep dermal sutures, but not in place of them. The glue consists of 2-octyl cyanoacrylate, and a strong bond is formed that rapidly provides wound strength equivalent to that seen at 7 days with conventional subcutaneous suture. It also forms a seal that in theory helps prevent water penetration or bacterial contamination of the healing wound.

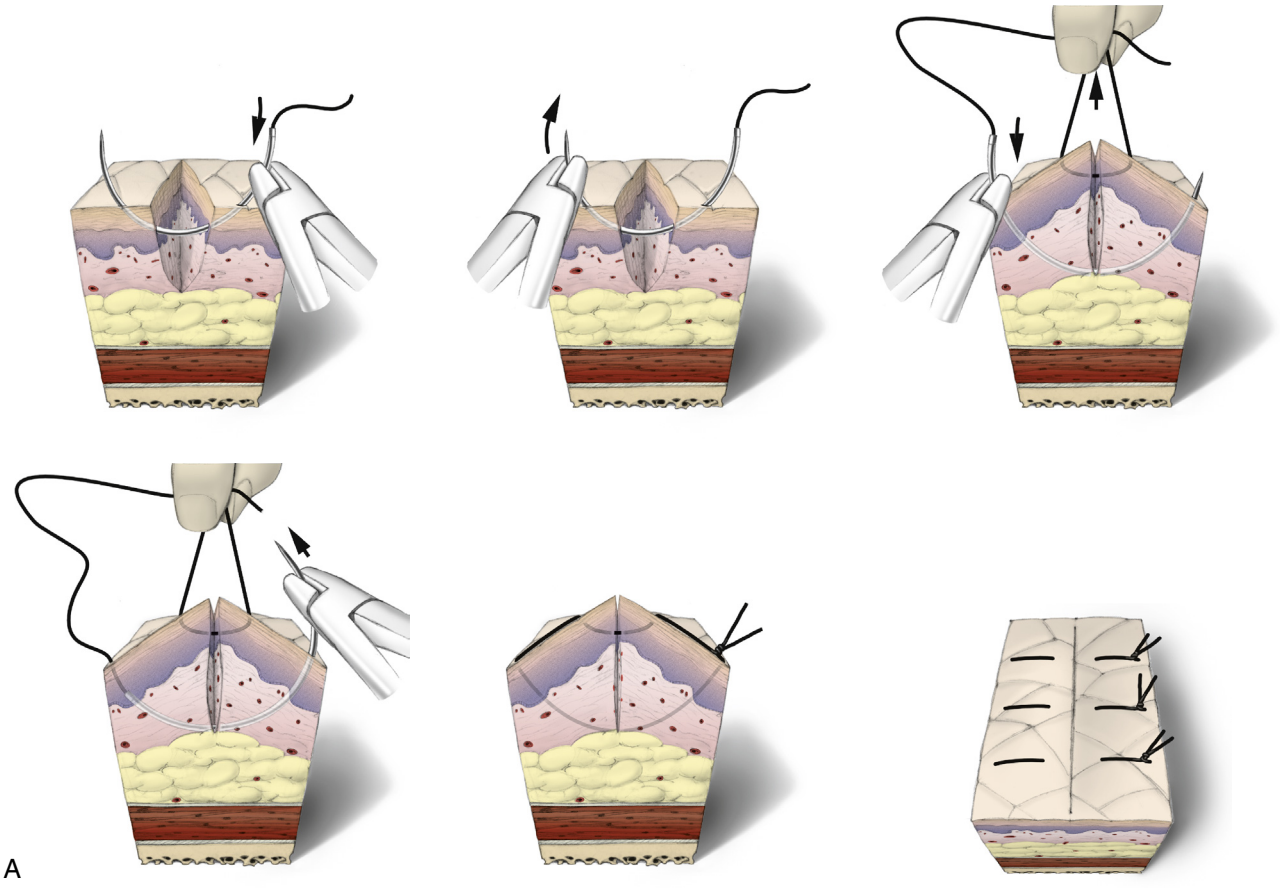
Adhesive strips (Steri-Strips) are made of porous, nonwoven elastic material with adhesive backing, and can be used to reinforce superficial closure (Fig. 3.45). Adhesive strips can potentially lead to better cosmesis and are cost effective compared with surgical glue.

Goals for Wound Closure

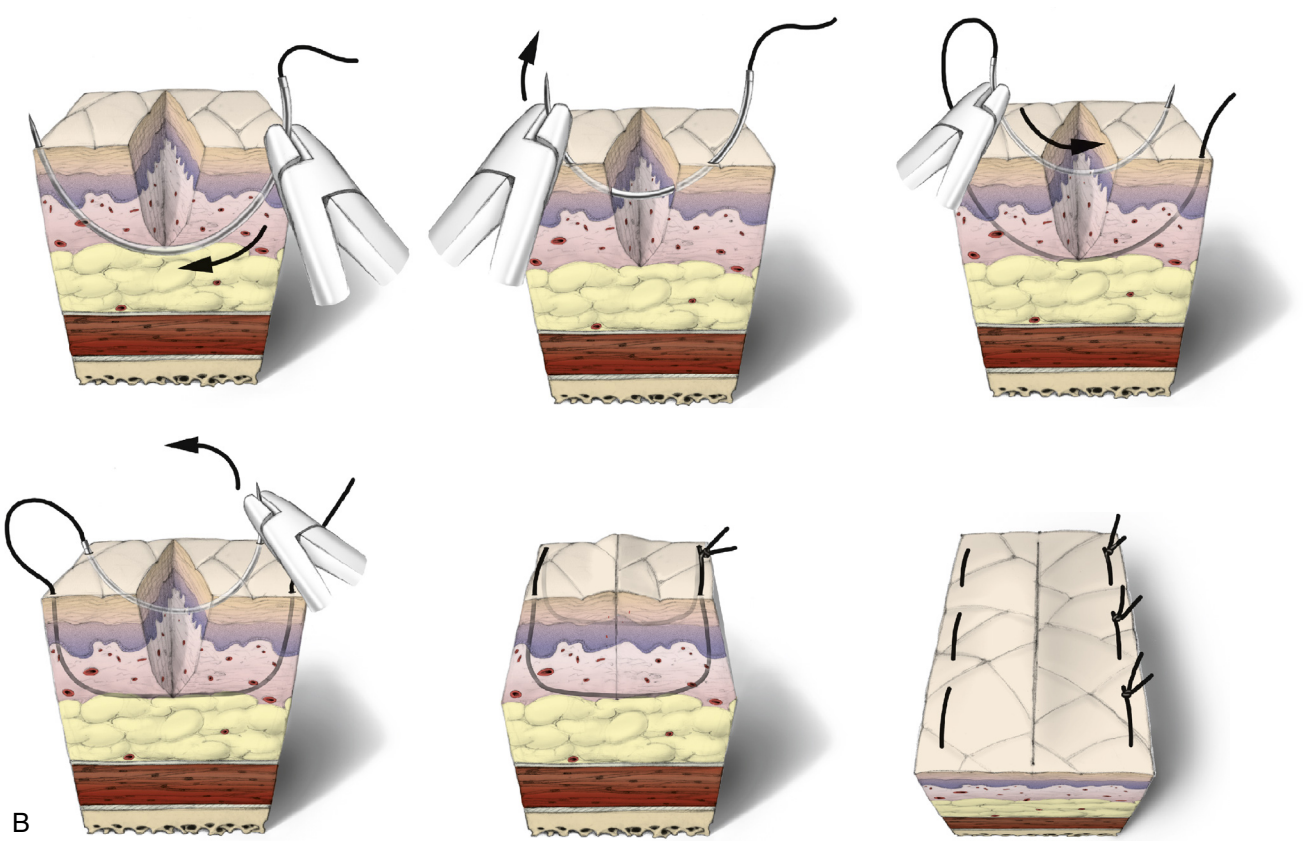
When closing wounds, one should adhere to certain key principles. Meticulous hemostasis should be achieved to prevent postoperative hematoma and minimize the risk of infection. Irrigation with antibiotic solution can be used if a medical device is being manipulated. Care must be taken to minimize



• **Fig. 3.39** Interrupted suture: (A) superficial; (B) deep dermal.



• **Fig. 3.40** (A) Vertical mattress suture.



• **Fig. 3.40, cont'd** (B) Horizontal mattress suture.

traumatic handling of tissues including during retraction and suturing. Dead space should be eliminated, which can be achieved by a meticulous layered closure. The Scarpa layer should be reapproximated if possible. The final incision should have good edge-to-edge apposition, and be slightly everted. Finally, a dry sterile dressing should be applied to minimize the risk of postoperative infection.

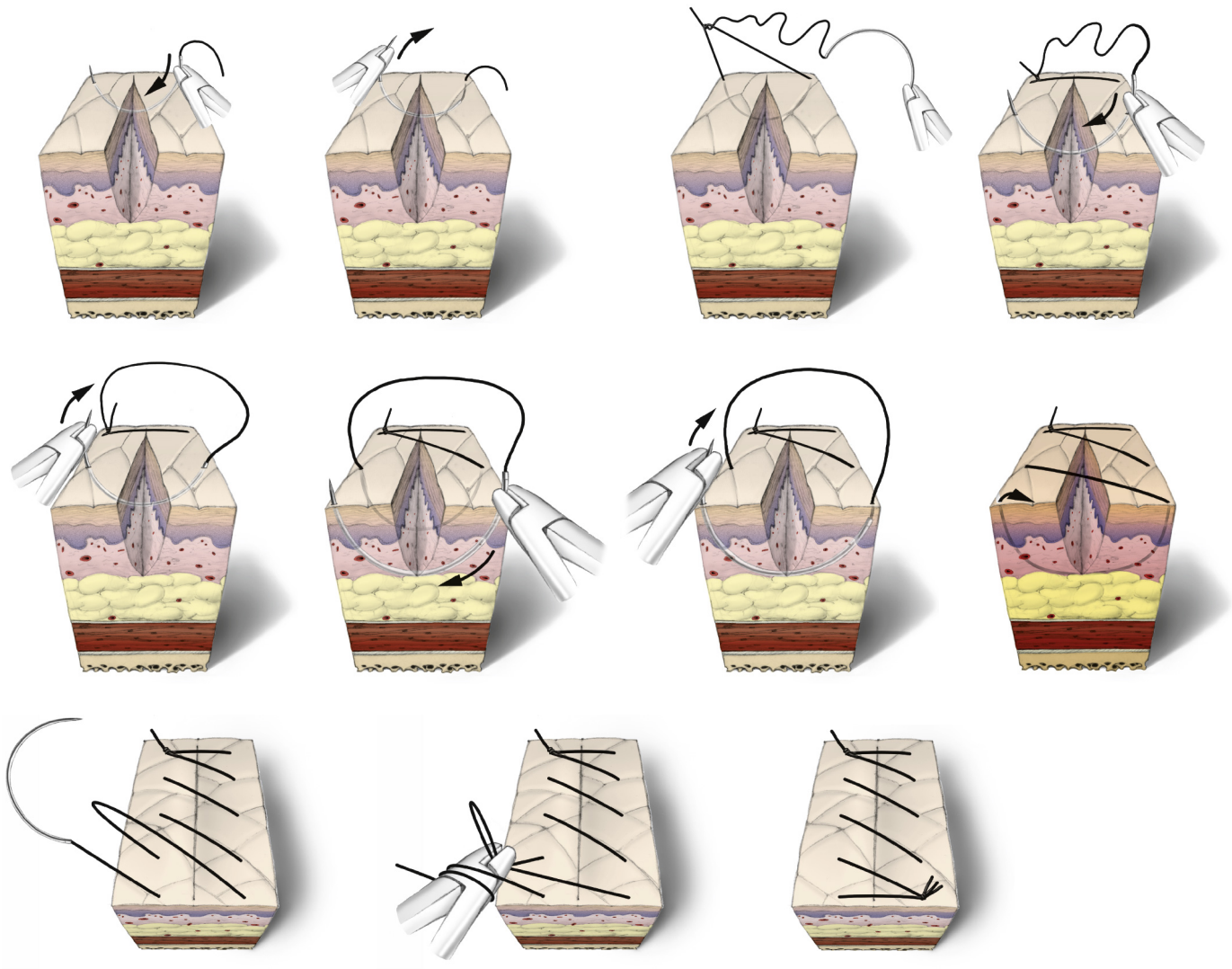
Wound Healing

Once the procedure is complete, the process of wound healing commences. Ideally, the wound will heal by primary intention. Healing of a surgical wound occurs in three distinct phases: (1) inflammatory, (2) proliferative, and (3) maturation/remodeling (Fig. 3.46).

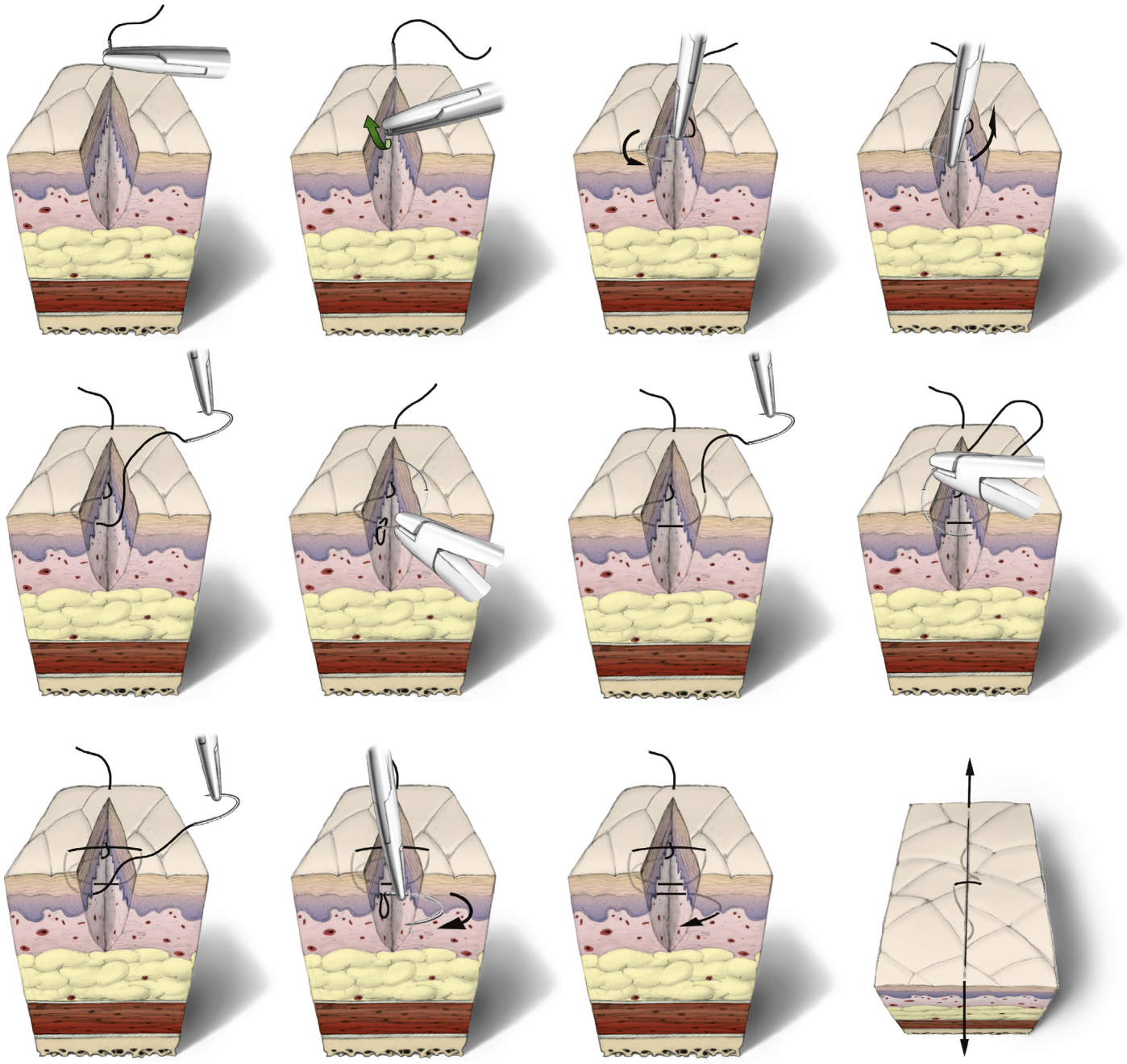
The inflammatory stage lasts anywhere from 3 to 7 days. Once hemostasis is achieved, vasodilation occurs, bringing an influx of inflammatory cells including neutrophils, monocytes, and macrophages. Numerous growth factors (platelet-derived growth factor, transforming growth factor-β) and cytokines are released, initiating angiogenesis, duplicating vascular smooth muscle cells, and preparing the cellular milieu for fibroblasts.

The proliferative phase occurs from 7 to 28 days and consists of multiple processes, including fibroplasia, matrix deposition, angiogenesis, and reepithelialization.

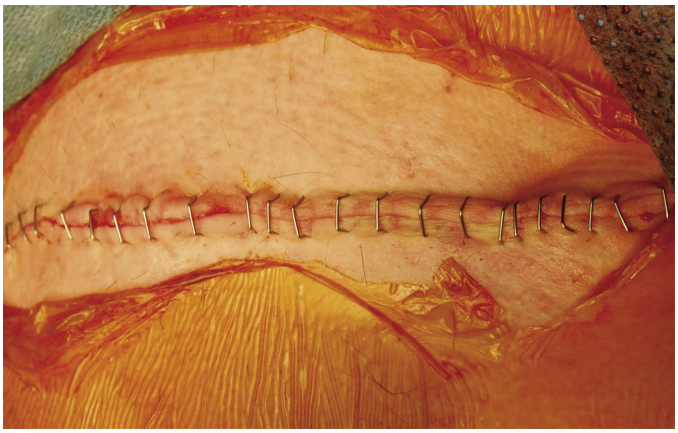
The final phase, remodeling, occurs after the third week and continues for up to a year. Collagen deposition and remodeling is one of the hallmarks of this phase. In addition, wound contraction is being mediated by myofibroblasts, and maximal tensile strength can be expected to occur by 3 months postoperatively. Scar appearance, however, will continue to evolve over the course of a year. Therefore, efforts to revise scars should be postponed until full maturation of the scar has occurred.



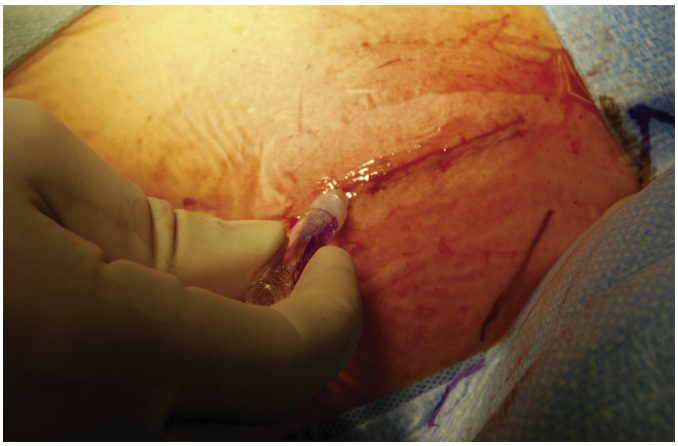
• **Fig. 3.41** Continuous running (over-and-over) suture.



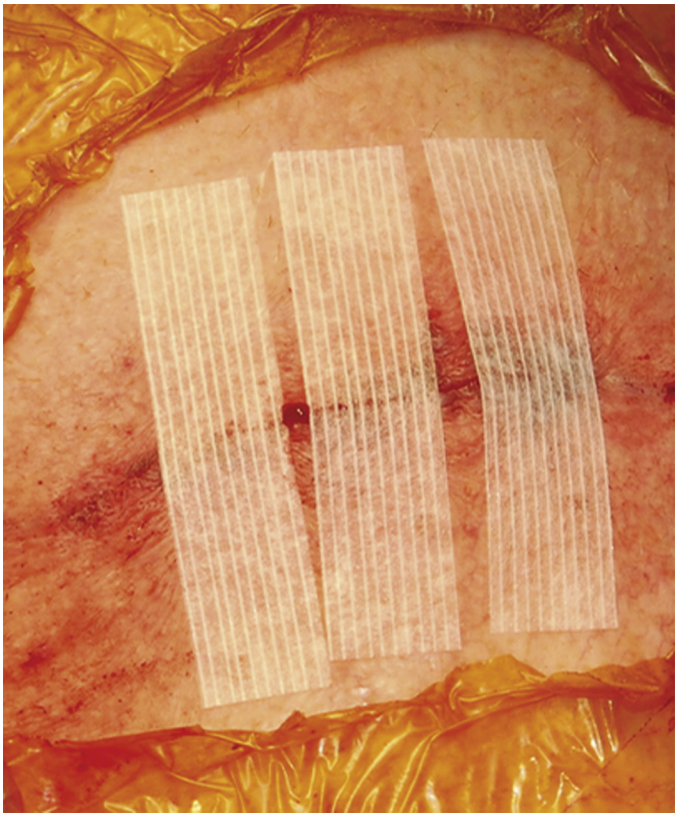
• **Fig. 3.42** Continuous subcuticular suture.



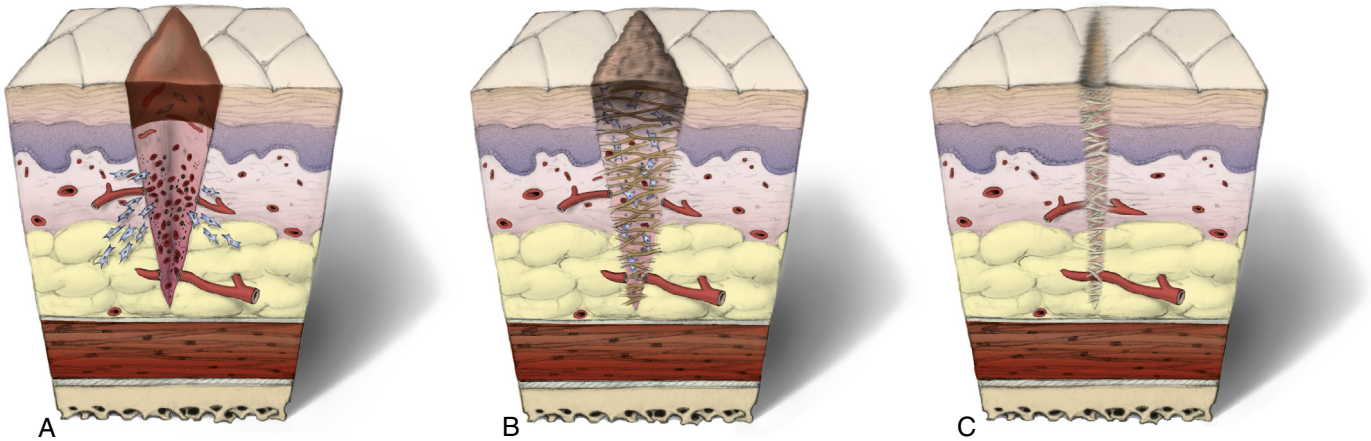
• **Fig. 3.43** Skin staples.



• **Fig. 3.44** Two-octyl cyanoacrylate surgical glue (Dermabond) used to approximate skin edges that are not under tension. It may also be used as an occlusive barrier following a subcuticular closure.



• **Fig. 3.45** Adhesive strips (Steri-Strips) used to alleviate tension from the incision following closure.



• **Fig. 3.46** Three phases of wound healing. (From Sherris DA, Kern EB. *Essential Surgical Skills*, ed 2. Philadelphia: Elsevier; 2004.)

Summary

Mastering surgical skill and technique is an ongoing process that may take years to develop. Careful attention to judgment, situational awareness, and leadership are equally as important as using the proper instruments, sutures, and needles. Moreover, focus on posture and sound biomechanics are just as important as adequate exposure and illumination of the surgical field. Anticipating when deviations from the typical procedure may occur will provide the patient with the best surgical outcome in a safe and efficient manner.

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4

Anesthesia for Interventional Cardiology

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Introduction

The basic goals of providing anesthesia are to decrease the pain and anxiety resulting from invasive medical procedures. Various methods are available, ranging from rendering a patient completely unresponsive to only providing infiltration of local anesthetics to the area where the procedure will be done. It is important to realize that all these procedures, including "just local," have the potential for complications and should only be done after a preoperative evaluation by trained personnel. The technique that is used is determined by the type of surgical procedure, the anatomic site of the procedure, the expected duration of the procedure, risk factors for that particular patient, the efficacy and safety of the technique, and patient acceptance. The main options available can be classified as minimal, moderate, or deep sedation; regional anesthesia; and general anesthesia. Emergency equipment and medications should be available to manage any cardiorespiratory or other events.

Preoperative Evaluation

The preoperative evaluation should include documentation of comorbid illness, clinical examination and reduction of patients' anxiety through education, and assurance that preexisting medical conditions have been optimally managed. When necessary, selective referrals should be made to medical specialists, relevant investigations ordered, and interventions intended to decrease risk initiated. Aspects of perioperative care need to be discussed, including arrangements for appropriate postoperative care. If any health concerns are identified, such as poorly controlled diabetes or hypertension, it may be appropriate to delay, modify, or cancel the surgical procedure.[1]

Key Points in the Preoperative Evaluation

History

- Presenting problem: Note the reason for the proposed cardiovascular procedure with relevant investigations and response to previous treatments.
- Coexisting medical problems: Significant respiratory, gastrointestinal, renal, neurologic, hematologic, or endocrine diseases may complicate the surgical and anesthetic course.
- Allergies and drug reactions: True allergies should be differentiated from known drug side effects. Specific drugs that are more often associated with problems include antibiotics, intravenous (IV) contrast, succinylcholine, latex, heparin, and protamine.
- Anesthesia history: Important problems are adverse reactions to sedative and anesthetic agents, vascular access, ease of mask ventilation, difficulty with laryngoscopy and intubation, intraoperative awareness, significant postoperative nausea and vomiting (PONV), and prolonged emergence.
- Family history: Malignant hyperthermia, myopathies, and pseudocholinesterase deficiencies have significant implications for anesthesia care if the patient has any of these conditions.
- Social history: Chronic obstructive pulmonary disease (COPD) increases the risk of respiratory complications. Patients with COPD tend to wake up coughing, which can cause hematoma formation after venous or arterial access procedures. Alcohol and recreational drug use should be noted. Chronic opioid and benzodiazepine use causes tolerance to anesthetic and sedative drugs. Moderate sedation in this latter group of patients is challenging.
- Current medications: β-Blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, antiarrhythmics, anticoagulants, diabetic medications, and recent steroid use should particularly be noted.
- Fasting guidelines: Except for emergent procedures, all patients should stop clear liquids 2 hours before the procedure and light meals 6 hours before the procedure. An 8-hour or more delay may be appropriate after a large, fatty meal.

Review of Systems

- Upper airway pathology that will affect airway management, such as prior radiation therapy, neck masses, or previous maxillofacial surgery
- Respiratory conditions such as upper respiratory tract infection, asthma or COPD, productive cough, orthopnea, or sleep apnea
- Cardiac disease, including any implanted cardiac devices
- Endocrine disease: Diabetes, thyroid
- Gastrointestinal disease: Particularly risk factors for aspiration such as gastroparesis, severe reflux due to a hiatal hernia, or autonomic neuropathy due to diabetes

Physical Examination

- Vital signs should be taken, including oxygen saturation and temperature, height, and weight.
- Airway: The two basic questions are whether there are any predictors for difficult mask ventilation and difficult laryngoscopy.
  - Difficult mask ventilation: (1) A full beard and a lack of teeth make it difficult to get a good seal around the face; (2) macroglossia (higher Mallampati grade); (3) history of snoring or obstructive sleep apnea (OSA)—yes to more than four questions in Table 4.1 signifies a high risk for OSA; (4) high body mass index.
  - Difficult laryngoscopy: (1) Limited mouth opening; (2) thyromental distance: a receding chin allows less space to displace the tongue anteriorly during direct laryngoscopy; (3) Mallampati classification: this assesses the relative size of the tongue when the mouth is fully opened—classes III (only the soft palate and base of uvula are visible) and IV (only the hard palate is visible) are predictors of difficult laryngoscopy, particularly in the presence of other risk factors such as a receding chin (Fig. 4.1); (4) loose or chipped teeth should be noted—protruding teeth can complicate laryngoscopy; (5) restricted neck extension; (6) pathology involving the neck such as masses, deviations, and venous congestion are important—management of a patient with superior vena cava syndrome or intrathoracic masses (e.g., retrosternal goiter) compressing the trachea is very challenging.
- Respiratory examination: Evidence of lower respiratory infection, pulmonary edema, wheezing, or signs of respiratory distress should be noted.
- Cardiovascular examination: Exercise tolerance is a useful predictor of how the patient's physiology will cope with the hemodynamic challenges due to anesthesia drugs and the particular procedure. Patients who can sustain 4 metabolic equivalents (METS) or greater generally have enough cardiopulmonary reserve to tolerate anesthesia and a surgical procedure.
- Abdominal examination: Abdominal distention or ascites may compromise ventilation and increases the risk for aspiration.

TABLE 4.1 STOP-BANG Scoring Model

S	Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?
T	Do you often feel tired, fatigued, or sleepy during the daytime?
O	Has anyone observed you stop breathing during your sleep?
P	Do you have or are you being treated for high blood pressure?
B	Body mass index >35 kg/m^2?
A	Age older than 50 yr old?
N	Neck circumference >40 cm?
G	Male gender?

From Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. *Anesthesiology*. 2008;108:812–821.

- Neurologic examination: Existing deficits should be noted and the patient's ability to cooperate with monitored anesthesia care assessed. Elderly patients with existing neurologic deficits may become disinhibited and uncooperative in an unfamiliar environment, particularly when sedation is used.

Laboratory Studies

Routine studies are not indicated beyond what the physicians order for the procedure being performed. Selected hematologic, coagulation, and serum chemistry studies should be done when indicated based on the presence of comorbidities and current medication use (e.g., diuretics, digoxin). Potassium abnormalities are particularly important in patients with cardiac pathology. Blood should be cross-matched if the patient is anemic or there is a risk of significant blood loss, especially if the patient has antibodies to unusual antigens.

Cardiology patients usually do not require further cardiac investigations in addition to those indicated for their cardiac procedure. In selected patients a chest radiograph and pulmonary functions may be indicated.

Preoperative Considerations

Selecting the Appropriate Anesthesia Management

The type of perioperative sedation/anesthesia used should be based on the preoperative evaluation, the type and duration of procedure that will be done, and, to some extent, patient preference. The options are:

- Sedation only for a minimally invasive procedure
- Tissue infiltration of a local anesthetic, with or without sedation
- Regional anesthesia, with or without sedation
- General anesthesia

Relevant Definitions

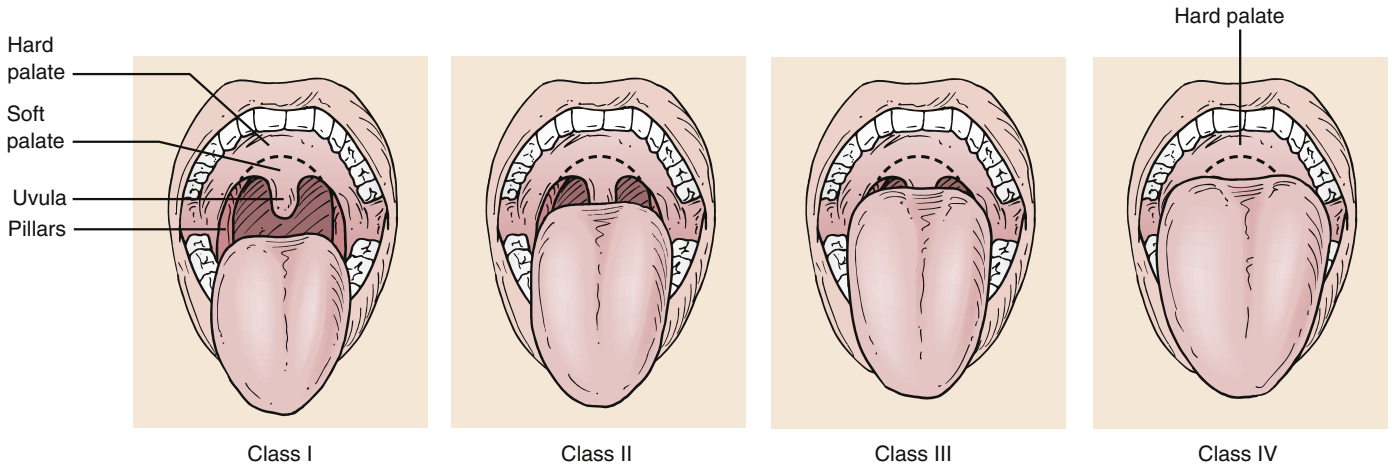
These different degrees of sedation and anesthesia are not always clearly defined, and patients may progress from one level to a lighter or deeper level during the course of the procedure. Each level has associated respiratory and cardiovascular implications, as described in Table 4.2. The American Society of Anesthesiologists (ASA) defines the levels of sedation and anesthesia as follows.

Sedation

Sedation inhibits transmission of nerve impulses between the cerebral cortex and limbic system. This inhibits both anxiety and the creation of long-term memories. Sedation is a continuum from anxiolysis to general anesthesia but is frequently divided into several categories, as shown in Table 4.2. Sedation can be used by itself or as a supplement to local anesthetic infiltration or regional anesthesia.

General Anesthesia

General anesthesia renders the patient completely unresponsive to painful stimulation.[2] This implies a loss of protective



• **Fig. 4.1** The Mallampati score is based on the number of pharyngeal structures seen. Class III or IV may indicate the potential for a difficult laryngoscopy particularly in the presence of other risk factors, such as a receding chin. (From Whitten CE. *Anyone Can Intubate,* ed 5. Copyright 2015 Mooncat Publications.)

TABLE 4.2 Definition of General Anesthesia and Levels of Sedation

	Minimal Sedation Anxiolysis	Moderate Sedation/ Analgesia (Conscious Sedation)	Deep Sedation/ Analgesia	General Anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful[a] response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

From American Society of Anesthesiologists. Continuum of depth and sedation: definition of general anesthesia and levels of sedation/analgesia. Committee of Origin: Quality Management and Departmental Administration; 2014. Available at: http://www.asahq.org/~/media/sites/asahq/files/public/resources/standards-guidelines/continuum-of-depth-of-sedation-definition-of-general-anesthesia-and-levels-of-sedation-analgesia.pdf.

Sedation is a continuum; it should always be anticipated that a patient may progress to a deeper level than intended, even in the hands of experienced practitioners, owing to variations in the response to anesthetic drugs. The appropriate monitoring, drugs, equipment, and expertise should be available to resuscitate a patient at all times.

[a]Reflex withdrawal from a painful stimulus is NOT considered a purposeful response.

Monitored anesthesia care (MAC) describes a specific anesthesia service in which an anesthesiologist has been requested to participate in the care of a patient undergoing a diagnostic or therapeutic procedure.

airway reflexes, although the patient may still be breathing spontaneously as long as the airway is not obstructed. It may also be accompanied by various degrees of cardiovascular depression.

Regional Anesthesia

Transmission of nerve impulses between a targeted part of the body and the spinal cord is blocked, resulting in loss of sensation in a specific area. A patient under regional anesthesia remains fully conscious. Two categories of regional anesthesia exist. A peripheral blockade inhibits sensory perception in a body part, such as numbing a tooth for dental work or administering a nerve block to stop sensation from an entire limb. A central blockade administers the anesthetic around the spinal cord, which suppresses all sensation below the block. Examples of central blockade include epidural and spinal anesthesia.

Identifying High-Risk Patients

It is important to identify patients who are at higher risk for complications during moderate sedation so that more experienced care by anesthesia providers can be arranged. This may even be necessary for unstable patients having minimal or no sedation, but where rapid resuscitation may be needed. Each institution should have detailed patient selection guidelines for moderate sedation. The ASA classification of physical status is often used as a guide (Table 4.3). However, this classification is open to interpretation, even among anesthesiologists. To minimize confusion and possible conflict when selecting patients

TABLE 4.3 ASA Classification Clarification Table

Class	Physical Status	Examples	Presence of Anesthesia Provider
I	Healthy patient No disease outside surgical procedure	Healthy without medications	Not needed unless any absolute contraindications present
II	Medically well-controlled mild to moderate systemic disease No functional limitation	Well-controlled disease states including: Diabetes Hypertension Asthma Epilepsy Obesity Thyroid disorders	Not needed unless any absolute contraindications present
III	Severe systemic disease Definite functional limitation	Poorly controlled: Diabetes ± end-organ disease Hypertension (diastolic BP >100) Asthma/COPD History of stable coronary artery disease	Consider anesthesia consult
IV	Severe incapacitating disease Constant threat to life	Unstable angina Severe CHF Severe COPD on home oxygen End-stage renal failure on dialysis End-stage hepatic failure Morbid obesity with OSA on oxygen therapy	Consider anesthesia consult with reference to Tables 4.4 and 4.5
V	Moribund patient not expected to survive 24 hr without the procedure	Septic shock Polytrauma Massive PE Ruptured aortic aneurysm Head injury with increased ICP	Mandated anesthesia consult

ASA, American Society of Anesthesiologists; BP, blood pressure; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; ICP, intracranial pressure; OSA, obstructive sleep apnea; PE, pulmonary embolism.

TABLE 4.4 University of Washington Moderate Sedation Patient Selection Guidelines: Absolute Contraindications

ASA class V
(see unit-specific policy for cardiac parameters)

Elective procedure with a full stomach (not fasted)

Previous adverse reaction to sedation/anesthesia

Morbid obesity, body mass index >45 kg/m^2

Known/anticipated difficult airway for ventilation or intubation

Cardiomyopathy with EF <30%

Severe aortic/mitral stenosis
(valve area ≤1 cm^2)

Complex congenital heart disease

Pulmonary hypertension with PA systolic pressure >40 mm Hg or cor pulmonale

Pregnancy >16 weeks' gestation

Neurologic or neuromuscular disorders compromising respiratory or swallowing function

TBI with GCS score <10

From University of Washington Medical Center. *Moderate sedation/analgesia for procedures by non-anesthesia care providers*. Seattle, WA: 2014.

ASA, American Society of Anesthesiologists; EF, ejection fraction; GCS, Glasgow Coma Scale; PA, pulmonary artery; TBI, traumatic brain injury.

suitable for sedation by clinicians who are not anesthesiologists, our institution uses the absolute and relative contraindications shown in Tables 4.4 and 4.5, respectively, in addition to specific cardiac laboratory guidelines. These guidelines were developed at the University of Washington Medical Center in consultation with each specialty to ensure that anesthesia providers were taking care of the sickest patients who are most at risk for complications. Table 4.6 sets out the specific guidelines for the cardiology care area. Since the introduction of these guidelines, there has been a significant reduction in the number of cases that have required anesthesia intervention partway through a procedure.

Additional Risk Factors to Consider in Planning Sedation for Procedures

The presence of any of the below risk factors may place the patient at a higher risk for complications related to sedation/analgesia that are separate from those that may occur as a result of the procedure itself. If two or more risk factors are present, particularly anatomic factors related to the airway, consultation with an anesthesia care provider is recommended.[3]

Patient Factors

- Anatomic
  - Airway and craniofacial abnormalities, which may pose significant difficulty and risk if emergent

TABLE 4.5 University of Washington Moderate Sedation Patient Selection Guidelines: Relative Contraindications

Condition	Advice to Practitioner
Patients with OSA who are noncompliant with CPAP treatment	Also consider short-stay admission
BMI 40–45 and STOP-BANG score >4 (see Table 4.1)	
Advanced lung disease on home oxygen (except for bronchoscopy cases done by pulmonary/critical care physicians)	
End-stage liver disease	Consult if encephalopathy requiring treatment *or* two of the following: Child-Pugh score of C, MELD >15, Na <128 mEq/L
End-stage renal disease	Consult if any of the following are present: Known OSA Obesity (BMI >30 kg/m^2) Diabetes treated with an insulin infusion
Traumatic brain injury	Unintubated patients with: GCS score 13–15 → proceed with caution GCS score 10–12 → seek anesthesia advice

From University of Washington Medical Center. Moderate sedation/analgesia for procedures by non-anesthesia care providers. Seattle, WA: 2014.

BMI, body mass index; CPAP, continuous positive airway pressure; GCS, Glasgow Coma Scale; MELD, Model for End-Stage Liver Disease; OSA, obstructive sleep apnea.

TABLE 4.6 University of Washington Cardiac Labs Unit–Specific Policy for Cardiac Parameters of Relative Contraindications for Moderate Sedation for Procedures

	Cardiac Care Area		
Cardiac Subgroup	EP Lab	Cardiac Catheterization Lab	Echcocardiography Lab
Cardiomyopathy	EF <20%: consider anesthesia consult	EF <20%: consider anesthesia consult	EF <20%: consider anesthesia consult
Severe mitral stenosis (valve area ≤1 cm^2)	OK	OK	OK
Severe aortic stenosis	Consult anesthesia if ≤1.0 cm^2	Consider anesthesia consult if ≤0.8 cm^2	Consult anesthesia if ≤0.8 cm^2
Pulmonary hypertension (PAS >50 mm Hg)	PAS >50 mm Hg: consider anesthesia consult	PAS >50 mm Hg: consider anesthesia consult	PAS >50 mm Hg: consider anesthesia consult
Congenital heart disease	Consult anesthesia for complex cyanotic CHD	Consult anesthesia for therapeutic procedures Consider anesthesia consult for diagnostic procedures	Consult anesthesia for complex cyanotic CHD
LVAD	Consider anesthesia consult	Consider anesthesia consult	Consider anesthesia consult
ASA class V	Consult anesthesia	OK	Consult anesthesia

From University of Washington Medical Center. Moderate sedation/analgesia for procedures by non-anesthesia care providers. Seattle, WA: 2014.

ASA, American Society of Anesthesiologists; CHD, congestive heart disease; EF, ejection fraction; EP, electrophysiology; LVAD, left ventricular anterior displacement; PAS, pulmonary artery stenosis.

intubation is required and may also cause problems with bag/mask ventilation
- History of difficult intubation, snoring, stridor, or sleep apnea
- Mallampati grade III or greater
- Tonsillar hypertrophy, nonvisible uvula
- High or arched palate
- Large tongue
- Small mouth opening (<5 cm)
- Significant malocclusion of jaw, lockjaw
- Unable to bite upper lip with lower incisors (upper lip bite test)
- Protruding incisors
- Edentulous (difficult mask ventilation), loose or capped teeth
- Receding chin
- Short chin-to-thyroid cartilage distance (<6 cm)
- Neck or oral mass

  - Short neck and/or limited neck extension
  - Cervical spine injury, arthritis, or trauma limiting head, neck, or jaw range of movement
  - Significant obesity, especially involving head, neck, and facial structures
- Medication factors
  - Long-acting medications, which may produce prolonged duration and excessive effect
  - Effects of medications when used in combination: Consider additive or synergistic effects
  - Patients with chronic pain taking large doses of narcotics: Potential need for drug dosing beyond recommended maximums
- Psychosocial
  - Current emotional state
    - Extreme anxiety or agitation
    - Belligerent or uncooperative
    - Intoxication with drugs or alcohol
  - Previous experience or history
    - Profound claustrophobia
    - History of problems with a similar procedure
- Physiological
  - Underlying medical conditions
  - ASA classification I to V (see Table 4.3)
  - Chemical dependency/high tolerance to opioids
  - Allergies or sensitivities to local anesthetics, sedatives, or analgesics
  - Hemodynamic instability
  - Age (consider both chronological and physiologic age)
  - Cognitive function
    - Inability to follow directions
    - Profound confusion or delirium

Environmental Factors

- Location of procedure area: Availability of additional emergency support may be limited in remote locations.
- Skill level of personnel available: At least one person involved must be Advanced Cardiovascular Life Support (ACLS) certified. Personnel at the University of Washington Medical Center use a set of guidelines (see Table 4.6) that are specific for interventional cardiology. The patients usually have significant cardiac comorbidities that the cardiology team is capable of managing.

Procedural Factors

- Prone patient position
- Prolonged procedure: Patients may not be able to remain in one position for an extended period (consider effects of cumulative drug dosing)
- Highly complicated or new procedure (may be prolonged and require deeper sedation)
- Emergent procedure (patient may have a full stomach, increasing risk for aspiration)

Intraoperative Care

Guidelines for Non–Operating Room Anesthesia

The following is a summary of the ASA requirements for monitored care and anesthesia outside of the operating room.[4]

Environment

- Oxygen supply: A reliable source available for the duration of the procedure and a backup supply
- Suction
- Equipment for positive-pressure ventilation capable of administering at least 90% oxygen
- Anesthesia drugs and equipment depending on the type of anesthesia
- Monitoring equipment as defined by the ASA "Standards for Basic Anesthetic Monitoring"
- Electrical outlets
- Adequate illumination of the patient, anesthesia equipment, and monitoring equipment
- Sufficient space for equipment and personnel and to allow easy access to the patient
- "Code cart" immediately available
- Reliable two-way communication to request assistance
- Appropriate staff and facilities to provide postanesthesia care

Anesthesia Equipment

- Anesthesia machine (for general anesthesia care)
- Suction catheters
- Intubation equipment, oral/nasal airways, endotracheal tubes
- Intravenous pumps
- Warming devices: Forced-air warming and fluid warming

Equipment for Transport to Postanesthesia Care Unit or Intensive Care Unit

- Oxygen delivery (face mask, nonrebreathing mask, or transport ventilator)
- Oxygen tanks
- Portable monitors
- External defibrillator/pacemaker for specific patients

Intraoperative Monitoring

Early detection of impaired respiratory function or cardiovascular depression is imperative. This allows for interventions to prevent drug- or procedure-related complications.

Standard Monitors for Non–Operating Room Anesthesia[5]

- Oximetry
- Blood pressure
- Electrocardiograph (ECG)
- Capnography
- Temperature

Assessment of Ventilation

- Respiratory depression due to an overdose of sedating agents was responsible for 21% of claims related to monitored anesthesia care (MAC). Over half of these events were believed to be preventable with better monitoring.[6]
- Pulse oximetry[7]: This is an accurate monitor of arterial oxygen saturation. Desaturation is a late sign of insufficient ventilation when supplemental oxygen is used.
- Capnography[7]: This is a useful tool to measure respiratory rate and to detect episodes of apnea early. The use of capnography in combination with pulse oximetry results in decreased episodes of apnea and hypoxemia. The ASA's "Standards for Basic Anesthetic Monitoring" was updated in 2011 to include mandatory end-tidal carbon dioxide monitoring during moderate and deep sedation.

Additional Monitoring

High-risk patients or procedures that may cause hemodynamic compromise may benefit from the following:

- Invasive arterial blood pressure measurement. This should be considered for the following:
  - Severe cardiac disease (ischemic, valvular, and heart failure)
  - Procedures that may cause sudden changes in blood pressure
  - Situations where a noninvasive blood pressure (NIBP) monitor is contraindicated or may not be accurate:
    - Arrhythmias
    - Morbid obesity
    - Continuous flow blood from a ventricular assist device
    - Severe coagulopathy (risk of intramuscular hematoma)
    - Profound hypotension or hypertension
    - Intraaortic balloon pump (IABP)
- Use of potent cardiovascular drugs such as nitroprusside
- Pulmonary arterial pressure and cardiac output: This may be appropriate to guide management in patients with severe pulmonary hypertension and cardiogenic or vasodilatory shock
- Transesophageal echocardiography: This may be useful as a monitoring tool or to identify the cause for unexplained cardiovascular collapse
- Central venous pressure
- Cerebral oximetry or mixed venous saturation: This may be useful in patients with a ventricular-assist device who do not have enough pulsatility for reliable pulse oximetry

Moderate Sedation

Risks and Complications

Non–operating room anesthesia (NORA) is associated with challenges owing to the type of patient, the environment, and the procedures. It is easy to underestimate the risks posed by this unique situation. This is supported by a closed-claims review by Robbertze et al.,[8] which showed that 60% of claims relating to severe complications (death and permanent brain damage) were due to MAC or general anesthesia (GA) in NORA compared with only 30% in the operating room.

There is still risk involved even if the procedure is "just" done under MAC. A review of closed claims relating just to MAC showed that the severity of injury was similar to that which occurred during general anesthesia. A significant number of claims were due to death or permanent brain damage. The most common cause for injury was respiratory compromise due to oversedation.[6]

Patients presenting for cardiac procedures may have significant cardiac disease and in some cases may be managed nonsurgically because they are considered too high risk for surgical intervention. The environment may pose a challenge owing to lack of access to the patient and emergency equipment; for example, a difficult-airway cart may not be readily available or may not be regularly inspected and maintained. The room may be dimly illuminated to make viewing of the video display easier for the proceduralist. Sufficiently trained staff also may not be available to help in the event of an emergency.[5]

An ongoing quality improvement program should be in place to reduce morbidity and mortality related to NORA sedation. Potential risks or areas of deficiency should be identified to prevent adverse outcomes. Changes should be made where necessary and the results of these changes should be monitored. Guidelines or policies should be in place to ensure that staff are aware of the recommended methods of clinical practice. A system should be in place to allow voluntary and anonymous reports of critical incidents. Complications should be reviewed and systems errors identified and corrected.

Training Requirements for Moderate Sedation for Nonanesthesia Clinicians (MDs, PAs, and ARNPs)

Each institution should have its own policy and curriculum for moderate sedation training. The curriculum components vary considerably across the country, but most institutions require practitioners performing the procedure and prescribing the sedation/analgesia to:

- Have privileges both for the procedure and the sedation/analgesia
- Be familiar with the dosage and possible side effects of any medication he or she chooses to use
- Be able to manage drug-related complications including airway management, ventilation, and cardiovascular rescue (ACLS)
- Specifically prescribe and direct drug use

Medications for Sedation and Anesthesia

Minimal Sedation

Minimal sedation usually involves low doses of a single anxiolytic agent such as midazolam given either orally or intravenously. Our institution allows the use of up to 50 μg of

fentanyl as an alternative. Occasionally, low-dose analgesics may be given in addition to an anxiolytic.

Moderate Sedation

Typically, a combination of intravenous midazolam and fentanyl is used for moderate sedation, with recommended maximum doses (institution dependent) to minimize the incidence of adverse events.

Propofol is frequently used by anesthesiologists for light sedation through to general anesthesia. The use of propofol by nonanesthesia clinicians is controversial owing to concerns about its narrow therapeutic window and higher incidence of complications when administered by less experienced providers.

SEDASYS (Ethicon Endo-Surgery) was developed to deal with these concerns. This system integrates data from the ECG, noninvasive blood pressure, capnography, respiratory rate, pulse oximetry, and patient responsiveness to verbal stimuli to guide drug delivery. This allows nonanesthesia providers to administer mild to moderate propofol sedation. The ASA has recommended that it should be used for patients over age 18 years with ASA I and II physical status who are undergoing colonoscopy or esophagogastroduodenoscopy procedures. It should only be used in situations where anesthesia support is immediately available if required. It is not licensed for other procedures at this time.

Deep Sedation, Monitored Anesthesia Care, and General Anesthesia

These may ONLY be administered by anesthesia providers. However, all nonanesthesia clinicians should be prepared to resuscitate a patient whose level of sedation exceeds that intended.

Benzodiazepines

Examples of benzodiazepines include midazolam, diazepam, and lorazepam. These drugs provide anxiolysis, amnesia, sedation, and, in higher doses, anesthesia, in addition to antiepileptic and muscle-relaxant properties. The combination of midazolam and an opioid such as fentanyl is widely used to provide moderate sedation. Midazolam has the shortest duration of action and is the most appropriate benzodiazepine to use, particularly for shorter procedures. Patients typically recover within 2 hours, but it may take up to 6 hours.[9]

These drugs act by binding to the γ-aminobutyric acid A ($GABA_A$) receptor, similar to barbiturates, and depress the reticular activating system in the brainstem. This effect is antagonized by flumazenil, which reverses the central nervous system (CNS) effect of benzodiazepines.

These drugs can be given orally, intramuscularly, and intravenously. The intranasal dose of midazolam is 0.2 to 0.3 mg/kg and the sublingual dose is 0.1 mg/kg. These routes as well as oral dosing have not been approved by the U.S. Food and Drug Administration (FDA); however, they have been used for preoperative pediatric sedation. The dose range of intravenous midazolam for sedation is 0.01 to 0.1 mg/kg depending on the level of sedation required, the concurrent use of other drugs, and the age of the patient. A total dose of more than 5 mg should not be necessary in healthy adults. Patients older than 60 years or debilitated patients should not require more than 3.5 mg. Note that the full effect may require at least 2 minutes.

Other options for oral sedation are diazepam (0.2–0.5 mg/kg in pediatric patients and 5–10 mg in adult patients 1 hour before the procedure) and lorazepam (0.05 mg/kg in pediatric patients and 2–3 mg in adults 1 hour before the procedure). However, these drugs have long metabolic half-lives and are particularly poor choices for elderly patients, who have slower drug metabolism. The biggest problem with oral agents occurs when they are administered shortly before the procedure, in which case their peak effect may occur after the patient has left the medical center.

Benzodiazepines depend on hepatic biotransformation to inactive metabolites. Diazepam has an elimination half-life of 30 hours (even longer in older patients), compared with 15 hours for lorazepam and only 2 hours for midazolam. Note that a secondary peak occurs with diazepam, resulting in sedation 6 to 12 hours after administration owing to enterohepatic circulation.

Drug Interactions

- Erythromycin: Inhibits metabolism of midazolam and increases duration of action
- Opioids: Synergism leads to increased risk of apnea and may potentiate cardiovascular depression
- Cimetidine: Binds cytochrome P450 and decreases the metabolism of diazepam

Pharmacodynamics

- Cardiovascular: These drugs have minimal cardiovascular depressant effects. Blood pressure and peripheral vascular resistance may decrease owing to anxiolysis and reduced sympathetic tone. The depressant effect is more pronounced when opioids are used concurrently.
- Respiratory: These drugs depress ventilatory response to CO_2. Patients rarely become apneic if benzodiazepines are used as the only sedative. The addition of other respiratory depressants, such as opioids, increases the risk of apnea significantly.
- CNS: These drugs are anxiolytic and sedative and provide antegrade amnesia. They increase the seizure threshold and provide mild muscle relaxation mediated at the spinal cord level. Benzodiazepines do not provide analgesia.

Reversal

Flumazenil may be required in selected patients who become obtunded or hypoxic after receiving benzodiazepines. The initial maximum dose is 3 mg. Patients who respond can receive additional doses up to 5 mg. Up to 3 mg/hr can be used if the patient becomes resedated. Reversal of acute benzodiazepine overdosage with flumazenil may cause seizures in patients using benzodiazepines on a continuing basis.

Propofol

This drug is primarily used to induce general anesthesia but is also useful as a sedative. The primary advantage is faster recovery and faster time to discharge compared to alternative drugs such as midazolam. Owing to relatively complex infusion

pharmacokinetics and a higher incidence of respiratory and cardiovascular side effects compared to midazolam, the use of propofol in the endoscopy suite by nonanesthesiologists is controversial and currently subject to local regulation. Personnel using this drug should be able to resuscitate a patient whose level of sedation becomes deeper than intended.

It is formulated as a 1% oil-in-water emulsion, which includes egg lecithin and soybean oil. This solution supports the growth of bacteria. An opened vial or syringe should be discarded within 6 hours if agents such as disodium edetate or sodium metabisulfite have not been added to it.

Induction doses range from 1 to 2 mg/kg depending on what other drugs are used concurrently as well as the patient's age and coexisting medical conditions. Sedation can be achieved with smaller increments such as 10 to 30 mg. The typical maintenance infusion rate for sedation is 25 to 75 μg/kg per minute, versus 100 to 150 μg/kg per minute for anesthesia.

Propofol has a rapid onset and a short duration of action. The initial distribution half-life is 2 to 8 minutes. With repeated doses or with a continuous infusion, this distribution half-life will increase.

Pharmacodynamics

- Cardiovascular: Propofol causes a drop in arterial blood pressure owing to a decrease in systemic vascular resistance and a decrease in sympathetic activity. Propofol inhibits the baroreceptor response to hypotension. This effect is more pronounced with:
  - Larger doses
  - Rapid injection
  - Old age
  - Impaired left ventricular function
  - Hypovolemia
  - Concurrent use of β-blocking drugs
- Respiratory: Propofol is a profound respiratory depressant. The respiratory response to hypercarbia and hypoxia is depressed. Upper airway reflexes are depressed to a greater extent than with other induction drugs. This facilitates placing a laryngeal mask airway. However, propofol can readily cause upper airway obstruction when used for sedation. Personnel using propofol for sedation should be appropriately trained to manage airway obstruction.
- CNS: Propofol causes anxiolysis, sedation, and amnesia. Excitatory effects such as myoclonus may occur on induction. It increases the seizure threshold and can be used to treat status epilepticus. Propofol has antiemetic and antipruritic properties. Intravenous injection often results in localized pain.

Opioids

Opioids bind specific receptors in the CNS as well as other tissues. Although four opioid receptor types have been identified, analgesia and respiratory depression are provided by the μ receptors. Drugs acting on these receptors can be full agonists (morphine, fentanyl), partial agonist-antagonists (nalorphine, butorphanol), or antagonists. Agonist-antagonists are typically avoided in anesthesia because they may antagonize the effects of full agonists. Examples of opioids include fentanyl, remifentanil, morphine, and meperidine (pethidine).

Fentanyl has a rapid onset and short duration of action, making it suitable for shorter procedures. It has the additional advantage of causing very little histamine release and being cardiovascularly stable. Opioids are respiratory depressants and can cause respiratory arrest. Benzodiazepines work synergistically with opioids, and the combination can result in apnea and cardiac arrest.[10] Meperidine is 7–10 times less potent than morphine but it relaxes smooth muscles and produces mild euphoria.

Remifentanil is a unique ultra-short-acting opioid that is hydrolyzed by nonspecific esterases in the blood. The terminal elimination half-life is less than 10 minutes. This makes remifentanil particularly useful for interventional cardiology procedures because it significantly decreases the amount of other more cardiovascularly depressant anesthesia drugs required to keep a patient asleep without the risk of accumulation after a prolonged infusion.

Drug dosages and their duration of action are shown in Table 4.7. The listed loading dose will likely produce a significant effect that may be excessive in small, frail, or elderly patients.

Pharmacodynamics

- Cardiovascular: Morphine and meperidine cause histamine release, which decreases systemic vascular resistance and venous tone. Fentanyl and its derivatives cause less histamine release and tend to be more cardiovascularly stable. Blood pressure may be decreased owing to reduced medullary sympathetic outflow. Meperidine depresses myocardial contractility.

TABLE 4.7 Dose, Peak Effect, and Duration

Opioid	Typical Loading Dose (mg)	Peak Effect (min)	Duration (hr)
Morphine	10 (in 1–2 mg increments)	20–30	3–4
Hydromorphone	1.5 (in 0.2–0.4 mg increments)	15–30	2–3
Fentanyl	0.1 (in 25–50 μg increments)	3–5	0.5–1
Remifentanil	0.1 (in 25–50 μg increments)	1.5–2	0.1–0.2

Modified from Levine WC. *Handbook of Clinical Anesthesia Procedures of the Massachusetts General Hospital*, ed 8. Philadelphia: Lippincott Williams & Wilkins; 2016.

- Respiratory: Opioids depress respiration. The initial effect is a decreased respiratory rate followed by decreased tidal volumes at higher dosages. The ventilatory response to hypercarbia is depressed, and opioids suppress the cough reflex.
- Gastrointestinal system: Opioids cause decreased motility with an increased tone and secretions. Tolerance does not develop to this side effect, and chronic opioid users should be considered to have a full stomach. Peripheral opioid antagonists (methylnaltrexone and alvimopan) have been developed to manage these side effects of chronic opioid therapy.
- CNS: Opioids are primarily used for analgesia; however, higher doses cause sedation and anesthesia. Meperidine has an active metabolite (normeperidine) that can result in seizures when large doses are used. Euphoria, dependence, and addiction may occur. Repeated dosing of opioids will result in tolerance to the analgesic, sedative, and respiratory depressant effects. Opioids are approximately twice as potent in the elderly brain.
- Other: Muscle rigidity of the chest and abdomen can impair ventilation and may require treatment with neuromuscular blocking drugs. This is more common when larger doses are used for general anesthesia. Opioids may cause urinary retention. This is complicated by a decreased drive to urinate. Nausea and vomiting are common.

Reversal

Opioids can be reversed with naloxone; 20- to 80-μg increments should be given until the patient responds. The duration of action is 30–60 minutes. This may be shorter than the effect of the opioid agonist, and patients should be monitored for recurrent respiratory depression. Other complications include pain, hypertension, tachycardia, and pulmonary edema. Chronic opioid users may experience acute withdrawal symptoms.

Other Drugs Used by Anesthesiologists for Sedation

Dexmedetomidine

Dexmedetomidine was approved by the FDA in 1999 for sedation in the intensive care unit. This was extended in 2008 for procedural sedation in nonintubated patients. Dexmedetomidine produces dose-dependent anxiolysis and sedation. Patients can be woken up even from deep levels of sedation and cooperate with instructions. This "cooperative sedation" can be useful during certain procedures. Dexmedetomidine does have some analgesic effects. The main advantage is the lack of respiratory depression. It can be used as a single agent or in combination with opioids or benzodiazepines.

The main disadvantages are slow onset and recovery, as well as the potential for bradycardia and hypotension. It can be used to treat postoperative shivering.

Ketamine

This drug has dissociative properties, provides analgesia, and has limited respiratory depression compared to other sedatives. This dissociative state may make patients unresponsive to verbal commands. Ketamine may cause hypertension, tachycardia, and recovery agitation. These effects can be minimized with anxiolytics such as propofol or benzodiazepines.

General Anesthesia

General anesthesia adds to the cost and duration of the procedure. Before the procedure starts, consideration should be given regarding whether general anesthesia may be a safer alternative than using regional anesthesia and/or sedation.

Indications for General Anesthesia

- Respiratory distress or orthopnea
- High risk for hemodynamic instability
- Transesophageal echocardiography required for ongoing monitoring
- Inability to cooperate, such as pediatric, delirious, or severely cognitively impaired patients
- Long procedures, particularly if movement would compromise the procedure
- Chronic pain problems, which may complicate positioning for the procedure, such as chronic back pain
- Patients with risk factors for sedation such as morbid obesity or severe obstructive sleep apnea, or patients with severe reflux who require moderate sedation
- When the site cannot be adequately anesthetized with local infiltration, such as when a subpectoral pocket will be used for a pacemaker or implantable cardioverter-defibrillator

Management of Anesthetic Care

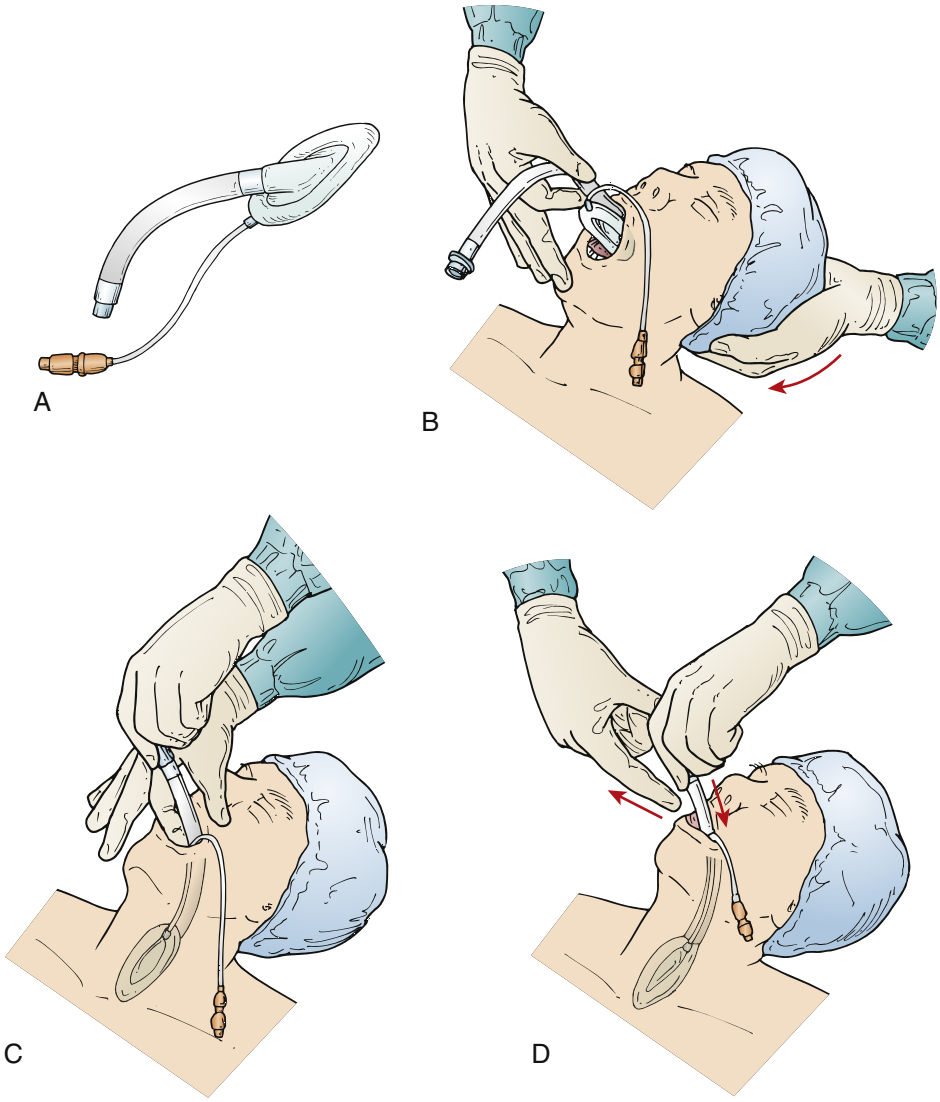
Induction and Airway Management

Patients can be intubated with an endotracheal tube, or the airway can be maintained with a laryngeal mask airway (LMA). The LMA has several advantages over endotracheal intubation: less anesthetic is required for placement, no laryngoscopy is required, and it can be placed quickly even by inexperienced personnel (Fig. 4.2). Patients have reduced anesthetic requirements during maintenance, less coughing during emergence, and a lower incidence of sore throat. The main disadvantage is that the seal around the larynx is less secure. Gastric insufflation may occur with positive-pressure ventilation. If gastric reflux occurs, it can lead to laryngospasm and/or aspiration pneumonia.

Maintenance

The anesthetic is maintained by a combination of anesthetic drugs (either inhalational or intravenous), opioids, and neuromuscular blocking drugs. The cardiovascular side effects of these drugs are managed with fluids and vasopressors such as phenylephrine, ephedrine, or vasopressin.

Volatile anesthetic agents suppress atrioventricular nodal reentrant tachycardia, and these agents are therefore best avoided. Volatile anesthetic agents also suppress inducible



• **Fig. 4.2** A laryngeal mask airway may be used to secure an airway in an emergency. It can be placed quickly and easily, even by inexperienced personnel. (A) The laryngeal mask ready for insertion. The cuff should be deflated tightly with the rim facing away from the mask aperture. There should be no folds near the tip. (B) Initial insertion of the laryngeal mask. Under direct vision, the mask tip is pressed upward against the hard palate. The middle finger may be used to push the lower jaw downward. The mask is pressed forward as it is advanced into the pharynx to ensure that the tip remains flattened and avoids the tongue. The jaw should not be held open once the mask is inside the mouth. The nonintubating hand can be used to stabilize the occiput. (C) By withdrawing the other fingers and with a slight pronation of the forearm, it is usually possible to push the mask fully into position in one fluid movement. Note that the neck is kept flexed and the head extended. (D) The laryngeal mask is grasped with the other hand and the index finger withdrawn. The hand holding the tube presses gently downward until resistance is encountered. (Courtesy Teleflex Medical Incorporated.)

ventricular arrhythmias. Intravenous anesthesia or, alternatively, deep sedation, at least during the mapping phase, can be used.

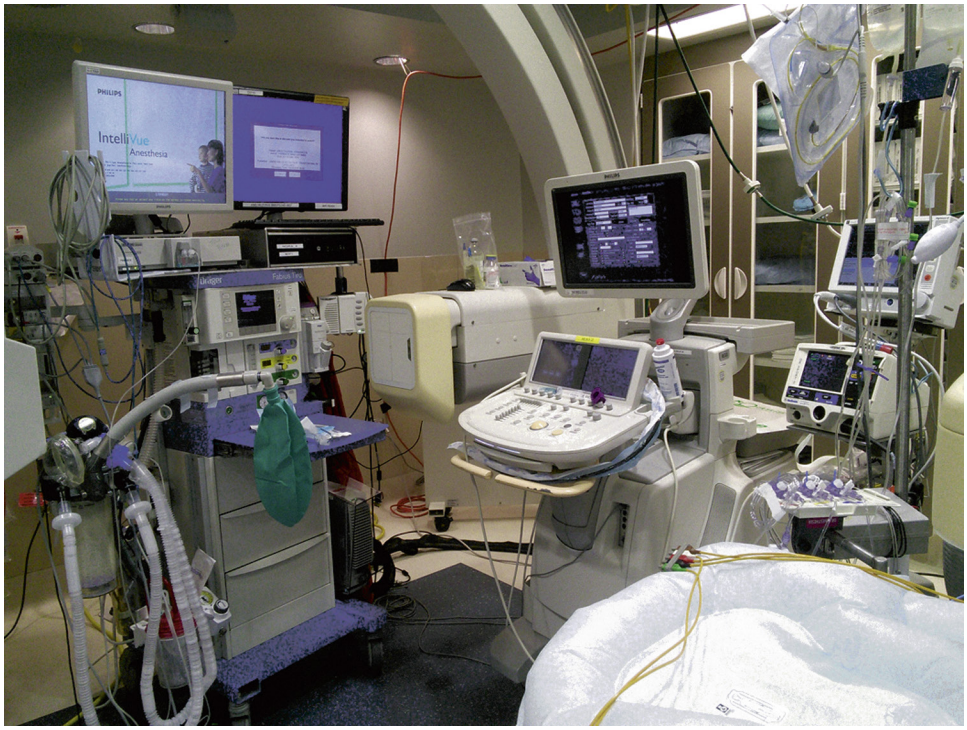
Emergence

At the end of the procedure the anesthetic drugs are discontinued, the neuromuscular block is reversed, and the patient is extubated once criteria are met, including a minute ventilation that supports adequate oxygenation as well as adequate carbon dioxide elimination, and being awake enough to protect the patient's own airway from aspiration.

Factors Complicating Care for Patients in Interventional Cardiology

Out of Operating Room Environment

Space is often restricted by imaging equipment (Fig. 4.3). This complicates access to the patient for airway management and



• **Fig. 4.3** Anesthesia equipment for a complex case requiring invasive monitoring and transesophageal echocardiography in the imaging laboratory.

cardiopulmonary resuscitation. This also complicates bringing in additional equipment such as a transesophageal echo machine in the event of hemodynamic instability. Anesthesiologists should be involved during the construction or remodeling of a facility where general anesthesia may be required, such as with hybrid operating rooms.

Manipulation of the C-arm or image intensifier and movement of the table can dislodge the breathing circuit or monitoring lines, or even inadvertently extubate the patient. Extensions for monitoring lines, IV lines, and breathing circuits help to avoid this.

Dimly lit procedure rooms improve the view of the video screens but impede access to the patient. It can be hazardous negotiating around wires and equipment. Tripping can be avoided and the cords can be protected by covering them, or moving them out of the way of the anesthesia work space if possible.

The imaging table may also not allow for a range of positioning options. For instance, the reverse Trendelenburg position may not be available for placing jugular central venous lines or in the event of aspiration. An additional wedge or pillow may be required for patients with orthopnea, which then can alter the fluoroscopic landmarks, and the implanting physician should be aware of this situation.

When required, additional equipment may need to be brought in from the operating room for specific cases. This includes blood administration sets, fluid warmers, rapid infusion devices, and additional airway equipment such as a fiberoptic bronchoscope or a GlideScope. Rarely used anesthesia emergency equipment, such as the "difficult-airway cart" or the "malignant hyperthermia cart," may not be readily available. Also, cardiology staff may not be familiar with anesthesia equipment and procedures.

If waste gas scavenging is not available, total intravenous anesthesia with propofol and remifentanil may be performed to avoid contaminating the room with volatile agents.

Patient Comorbidities

Anesthesia drugs used for induction and maintenance cause variable degrees of cardiovascular depression. Some (such as etomidate and midazolam) have a better safety profile than others. However, even with these drugs, there is a reduced sympathetic outflow. Additionally, hypoxia and hypercarbia may complicate the intubation. Patients with severe cardiac disease such as valvular disease, notably aortic stenosis, coronary arterial disease, cardiogenic shock, or pulmonary hypertension, may not tolerate these changes and may quickly reach a point where resuscitation may not be effective. If indicated, supportive measures such as an intraaortic balloon pump should be readily available or should be done before induction.

Patients with congenital heart disease present a unique challenge. It is important for the anesthesiologist to have a clear understanding of the underlying anatomy and the physiologic effects of anesthesia in each particular patient.

Anesthesia complications such as unanticipated inability to intubate or ventilate, hypotension on induction, anaphylactic reactions, and aspiration will have much more severe consequences in patients with significant cardiac comorbidities.

Procedures

Prolonged hypotension due to induced arrhythmias can be treated with vasoactive and inotropic agents to maintain an acceptable mean arterial blood pressure. Electrolytes and glucose should be checked during isoproterenol infusions and K^+ corrected as required.

Arrhythmias, hemorrhage, tamponade, myocardial ischemia, or bradycardia may complicate interventional cardiac procedures. The anesthesia team needs to be aware of the likely complications of the planned procedure so that they have the appropriate emergency equipment and drugs available. The anesthesia team also needs to know when drugs such as heparin or isoproterenol are given. Anesthesia personnel may not be aware of the amount of IV fluid given during certain electrophysiology procedures such as radiofrequency ablation, and this can lead to fluid overload if additional fluid is being given as part of the anesthesia management.

Communication between the proceduralist and the anesthesiologist is essential. Otherwise it is very difficult for personnel not directly familiar with electrophysiology procedures to discern if there has been a complication. Communication during the procedure is critical. If the proceduralist is concerned about a potential complication, communication about anticipated changes in vital signs needs to be verbalized to the anesthesia team. Conversely, if the anesthesiology team is concerned about hypotension (especially if vasopressors are being considered), heart rate changes, or oxygen saturation changes, these concerns need to be verbalized to the proceduralist.

The occurrence of complications may impact how the extubation is managed and the type of postoperative care and monitoring that will be required.

Unanticipated Request for Anesthesia Support During a Case

Occasionally anesthesia support may be required for cases in which it was not initially indicated. This may happen because of cardiovascular or respiratory compromise or because a patient became acutely confused and uncooperative.

The initial problem is that anesthesia personnel may not be immediately available. Anesthesiologists need rapid access to an anesthesia machine, equipment, and medications to effectively resuscitate such a patient.

It is critical that an anesthetic machine and an anesthetic cart with drugs and airway equipment be stored in proximity to procedure rooms. This facilitates fast and effective patient care.

Access to current vital signs may not be readily visible from the head end of the bed when an anesthesiologist arrives for an emergent case. This makes it difficult to deal with the hemodynamic effects of doing an emergency intubation. The anesthesiologist may also not be aware of coexisting medical conditions and adverse drug reactions that may impact anesthesia care. The airway management can be challenging since there may not be time to evaluate the potential for a difficult airway, positioning may be suboptimal, and there may not be a full range of airway devices available.

Local Anesthesia

Pharmacology

Local anesthetics impair propagation of action potentials in a nerve axon. This is mediated by blocking voltage-gated Na channels, which prevent membrane depolarization.

Chemistry

The two main classes of local anesthetic drugs are esters and amides. Esters (cocaine, procaine, and chloroprocaine) are rapidly metabolized by plasma pseudocholinesterase. One of the products of this metabolism is *p*-aminobenzoic acid (PABA). Amides (lidocaine, bupivacaine, and ropivacaine) are metabolized in the liver by P450 enzymes.

Bupivacaine is a racemic mixture of S(–) and R(+) isomers. The latter are more likely to produce cardiac and CNS side effects. Ropivacaine is similar to bupivacaine in terms of the onset and duration of the block. It produces less of a motor block. The main advantage over bupivacaine is that it is formulated as a pure S(–) isomer to reduce toxicity.

Both amides and esters are weak bases and become ionized in an acidic environment. Drugs with a pKa (the pH at which the ionized [BH^+] and the un-ionized [B] fraction are in equilibrium) closer to physiologic pH are less ionized and more lipid soluble.

The onset of action depends on lipid solubility and the relative amount of ionized compared to un-ionized drug. Lower-potency and less lipid-soluble drugs have a faster onset. More lipid-soluble drugs have a longer duration of action.

Additives

- Epinephrine: This prolongs the duration of action and reduces systemic absorption. This is less effective for longer-acting agents. The duration of action of bupivacaine is not significantly altered, but the duration of lidocaine is extended by 50% or more.
  - Typical amounts are 5 μg/mL (1:200,000 dilution).
  - Commercial solutions are pH adjusted to 4. This maintains the chemical stability of the epinephrine. Epinephrine can be added just before use to avoid this; 0.1 mL of a 1:1000 epinephrine added to 20 mL produces a 1:200,000 solution.
- Sodium bicarbonate: This shortens the onset and improves the quality of the block, particularly of commercial epinephrine-containing solutions. The alkaline pH increases the non-ionized free base, which results in faster diffusion. It may also decrease pain on injection.
- Lidocaine: 1 mL (1 mEq) of 8.4% concentration added to 10 mL lidocaine or mepivacaine.
- Bupivacaine: 0.1 mL of 8.4% concentration added to 10 mL of bupivacaine. Larger amounts may cause the bupivacaine to precipitate.
- Liposomal microspheres: These are multivesicular liposomes, which contain bupivacaine. The local anesthetic is slowly released from the liposome, which increases the duration of action significantly and delays the peak plasma concentration of bupivacaine. Pain scores are reduced for up to 72 hours.[11,12] Unfortunately, they are

only approved by the FDA for local infiltration after bunionectomy and hemorrhoidectomy.

Systemic Absorption

Systemic absorption determines the likelihood of toxicity. Plasma concentrations are determined by the total dose, the time over which the drug is given, and the vascularity of the site of the injection (absorption from IV administration > tracheal > intercostal > epidural > regional block > subcutaneous).

As noted, vasoconstrictors only reduce absorption for less lipid-soluble drugs. Toxicity should be considered as additive when local anesthetic drugs are mixed or used concurrently. Toxicity is increased by[13]:

- Advanced age
- Low cardiac output/heart failure
- Underlying cardiac disease such as ischemic heart disease and conduction abnormalities
- Liver disease
- Low plasma protein concentration
- Acidosis: either metabolic or respiratory
- Other medications that inhibit Na channels (e.g., class I antiarrhythmic drugs)

Children with right-to-left shunts are more prone to side effects if lidocaine is given IV (lung tissue absorbs significant amounts of the local anesthetic).

Adverse Reactions

- Allergic reactions: The PABA metabolite of esters can rarely cause anaphylaxis. Although anaphylaxis to amide local anesthetics is exceedingly rare, preservatives such as methylparaben can cause hypersensitivity in patients who are allergic to PABA.
- Methemoglobinemia: Prilocaine is metabolized to o-toluidine, which can cause methemoglobinemia. Benzocaine (commonly used in topical sprays) can also cause this side effect. Treatment consists of methylene blue, which reduces methemoglobin (Fe^{3+}) to hemoglobin (Fe^{2+}). The usual dose is 1 to 2 mg/kg of 1% methylene blue over 5 minutes.
- Systemic toxicity
    - Intravascular injection: Much lower doses will produce systemic toxicity. Intravascular epinephrine will cause tachycardia and hypertension. This can be a useful warning sign of inadvertent IV injection and can be avoided by aspirating before injecting and using smaller incremental volumes.
    - CNS: Initial symptoms are circumoral and tongue numbness, metallic taste, tinnitus, and blurred vision. This progresses to excitatory signs such as agitation and restlessness. Muscle twitching precedes grand mal seizures, coma, and eventually respiratory arrest. CNS toxicity is exacerbated by metabolic and respiratory acidosis as well as hypoxia. Higher-potency lipid-soluble drugs (such as bupivacaine) result in toxicity at lower doses.
    - Cardiovascular: Local anesthetics depress myocardial contractility, automaticity, and conduction owing to the class I effect on fast Na channels. These drugs (with the exception of cocaine) cause vasodilation at higher doses. Signs of CNS toxicity precede cardiac side effects, which occur at higher concentrations in awake patients. Cardiovascular collapse or arrhythmias may be the presenting sign in anesthetized patients. Bupivacaine causes more pronounced cardiac conduction abnormalities and ventricular arrhythmias than equivalent doses of lidocaine because of the high degree of tissue binding. This is exacerbated by acidosis. Prolonged resuscitation may be required.

Treatment of Local Anesthetic Systemic Toxicity

Local anesthetic systemic toxicity (LAST) presents initially with the CNS symptoms and signs described previously.[13] However, in a patient who is sedated or under general anesthesia, the cardiovascular effects may be the first indication of toxicity. The management of a cardiac arrest in this situation differs from the ACLS guidelines for cardiac arrest:

1. Call for help.
2. Initial focus
    a. Airway management: Ventilate with 100% oxygen.
    b. Seizure suppression: Benzodiazepines are preferred; AVOID propofol if the patient has cardiovascular instability.
    c. Alert the nearest facility that has cardiopulmonary bypass capability. Hypoxemia and acidosis potentiate the cardiovascular toxicity of local anesthetics and make these patients refractory to treatment. Cardiopulmonary bypass has been effective in this setting.[14]
3. Management of cardiac arrhythmias
    a. Basic life support and ACLS will require adjustment of medications and perhaps prolonged effort.
    b. AVOID vasopressin, calcium channel blockers, β-blockers, and local anesthetics (do not use lidocaine for ventricular arrhythmias).
    c. REDUCE individual epinephrine doses to less than 1 μg/kg.
    d. Lipid emulsion (20%) therapy (values in parentheses are for 70-kg patient)
        - Bolus 1.5 mL/kg (lean body mass) intravenously over 1 minute (~100 mL)
        - Continuous infusion 0.25 mL/kg per minute (~18 mL/min)
    e. Repeat bolus once or twice for persistent cardiovascular collapse.
    f. Double the infusion rate to 0.5 mL/kg per minute if blood pressure remains low.
    g. Continue infusion for at least 10 minutes after attaining circulatory stability.
    h. Recommended upper limit: Approximately 10 mL/kg lipid emulsion over the first 30 minutes.
4. Post LAST events at www.lipidrescue.org.[15]

Frequently Used Local Anesthetics

The maximum dosage and duration of action of frequently used local anesthetics are listed in Table 4.8.

TABLE 4.8 Local Infiltration

Anesthetic	Concentration (%)	Duration (min)[1] Plain	Duration (min)[1] With Epinephrine	Maximum Dose (mg/kg)[19]
Procaine	0.5–1.0	20–30	30–45	12
Lidocaine[a]	0.5–1.0	30–60	120	4.5
Ropivacaine	0.5	120–240	180–240	3
Bupivacaine	0.25–0.5	120–240	180–240	3

Modified from Levine WC. *Handbook of Clinical Anesthesia Procedures of the Massachusetts General Hospital*, ed 8. Philadelphia: Lippincott Williams & Wilkins; 2016.
[a]Maximum does of lidocaine with epinephrine is 7 mg/kg.

Postoperative Analgesia After Local Infiltration

Patients may require oral analgesia once the local anesthetic has worn off for procedures such as pacemaker placements. Options are acetaminophen with or without opioids, and nonsteroidal antiinflammatory medications such as ibuprofen or celecoxib.

Postoperative Care and Monitoring

Postanesthesia Care Unit Facilities

Patients should be observed after administration of sedation and anesthesia. Emergencies are rare but can happen suddenly and may be life threatening. Once patients are fully awake and complications of the sedation/anesthesia and surgical procedure have been dealt with, they can be discharged home, to a step-down unit, or to the cardiology ward. Patients who are unstable and need to remain intubated may be transported directly to the intensive care unit.

The ASA has a set of guidelines in place to ensure optimal care following a surgical procedure. These apply to patients who have just received general anesthesia, regional anesthesia, or moderate/deep sedation.

Summary of ASA Guidelines[16]

Periodic Patient Assessment and Monitoring

- Airway patency, respiratory rate, and oxygen saturation until the patient has recovered from the effects of the anesthetic drugs
- Pulse and blood pressure
- Neuromuscular function (only if nondepolarizing neuromuscular blocking drugs were used or in patients with preexisting neuromuscular dysfunction)
- Mental status/level of consciousness
- Temperature
- Pain
- Nausea and vomiting
- Postoperative hydration status
- Urine output
- Drainage, bleeding, or hematoma formation due to the surgical procedure

Treatment During Emergence and Recovery

- Supplemental oxygen should be provided during transportation and in the recovery room.
- Normothermia should be maintained and forced-air warming systems should be available.
- Postoperative shivering can be managed with meperidine HCl (Demerol).
- Antagonists for sedative, analgesic, and neuromuscular blocking agents can be given: flumazenil, naloxone, neostigmine (used for the reversal of residual neuromuscular blockade).
- Pacemaker or intracardiac defibrillator: Postanesthesia care unit staff should be informed of the type of device as well as the features of the device.

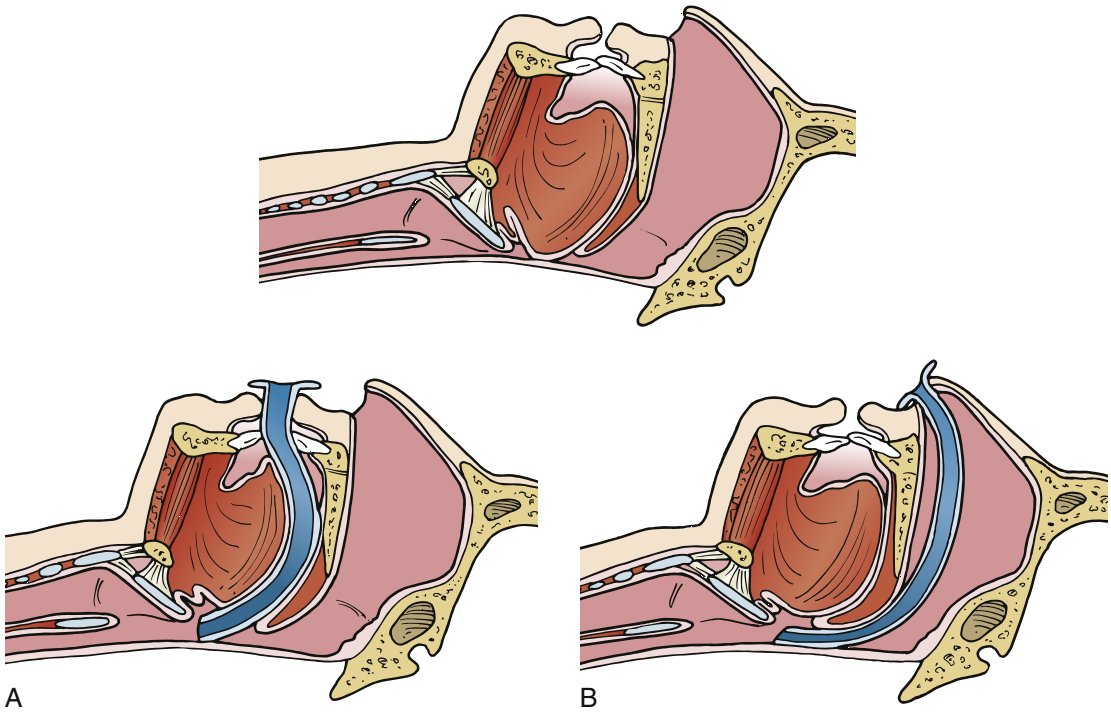
Protocol for Discharge

- All patients should be required to have a responsible individual accompany them home irrespective of whether they have received sedation or general anesthesia.
- Patients should be observed until they are no longer at increased risk for cardiorespiratory depression. There is no mandatory minimum stay.
- Selected patients should be screened for a full bladder.

Complications

Respiratory and Airway

- Hypoxia: General anesthesia and sedative drugs inhibit both the hypercapnic and hypoxic ventilatory drive. Even low doses of sedating medications can result in significant pharyngeal dysfunction and upper airway obstruction (Fig. 4.4). As noted previously, end-tidal CO_2 monitoring detects periods of apnea earlier than pulse oximetry. Other causes of hypoxia include atelectasis, pulmonary edema, aspiration of gastric contents, pneumothorax, and pulmonary embolism. A right-to-left shunt may also cause hypoxia.
- Hypoventilation with hypercarbic acidosis can be due to opioids and/or benzodiazepines. Flumazenil and/or naloxone may be required in select patients. Incomplete reversal of neuromuscular blocking drugs is another cause for postoperative respiratory failure.



• **Fig. 4.4** Loss of muscle tone in the upper airway results in airway obstruction. The airway can be restored by pulling the lower jaw forward or by placing an oral (A) or nasopharyngeal (B) airway. (From Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology,* ed 3. New York: McGraw-Hill; 2013.)

Hemodynamic Instability

- Hypotension may be due to hypovolemia (e.g., retroperitoneal hemorrhage), tamponade, cardiogenic shock, and tension pneumothorax.
- Arrhythmias such as ventricular fibrillation, ventricular tachycardia, bradycardia, and asystole may complicate interventional procedures. An external cardiac defibrillator with pacing capability should be readily available.
- Myocardial ischemia: Hypoxia, hypercarbia, hypotension, and pain may contribute to the development of myocardial ischemia.
- Hypertension may be a manifestation of preexisting diseases, but other causes such as pain, hypoxemia, hypercarbia, full bladder, fluid overload, and the administration of vasoactive medications should be considered.

Delayed Awakening

This is most frequently anesthetic drug related. Other causes include hypothermia, hypoglycemia, acid-base and electrolyte imbalances, hypothyroidism, hypoxia, and hypercarbia.

Postoperative Nausea and Vomiting

Management options are transdermal scopolamine 1.5 mg, dexamethasone 4 to 8 mg IV, serotonin antagonists (ondansetron 4–8 mg IV), and phenothiazines (promethazine 12.5–25 mg). Droperidol 0.5 to 1.0 mg is very effective but should only be used as a last resort owing to QT prolongation.

Pain

This is seldom a significant problem. However, patients with chronic back pain may require IV opioids when they are required to remain supine for several hours after the procedure.

Conclusion

The ultimate aim is to create the least stressful and the safest environment for the patient. Cooperation between the cardiologist and the nursing staff responsible for sedation and anesthesiology can decrease complications associated with the procedure and anesthesia. Institutional guidelines should be in place to standardize care of complex patients, and electrophysiology staff should be familiar with these documents. Nonanesthesia staff providing sedation care should be empowered to speak up if they are faced with a clinical scenario in which they do not have enough experience. Personnel and equipment should be rapidly available to deal with unexpected life-threatening events. An ongoing quality improvement system should be in place that can address deficiencies before an adverse event happens. Systemic problems should be identified and corrected when an adverse event does happen.

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5
Radiation Safety

MELISSA ROBINSON

X-ray fluoroscopy and cineangiography are required for lead positioning, cardiac electrical device implantation, and lead extraction using present technology. Substantial ionizing radiation exposure to the patient, operator, and staff can result. X-ray exposure is cumulative; it can have important adverse effects, including increasing risk of malignancies, cataracts, and skin necrosis.

Advances in radiographic imaging have helped optimize image quality, decrease patient and operator x-ray exposure, and improve usability, but exposure can remain unnecessarily high depending on operator and patient characteristics. The implanting operator and laboratory personnel are required to obtain and maintain a working knowledge of the radiographic systems.[1] A recent official statement on radiation use in cardiac procedures, the "ACCF/AHA/HRS/SCAI Clinical Competence Statement on Physician Knowledge to Optimize Patient Safety and Image Quality in Fluoroscopically Guided Invasive Cardiovascular Procedures," provides a detailed review of radiation concepts and their clinical applications.[2]

This chapter discusses radiation safety, provides guidance on minimizing use of unnecessary radiation, and offers practical recommendations for operators and staff.

Basic Radiation Terminology

Despite mastering complex and high technical concepts regarding cardiac rhythm device management, implanting physicians often lack a working knowledge of basic radiation terms of measurement. The nomenclature can be intimidatingly obtuse, but, nevertheless, it is essential because measurements of radiation exposure can help estimate biologic risks of ionizing radiation to patients and providers.

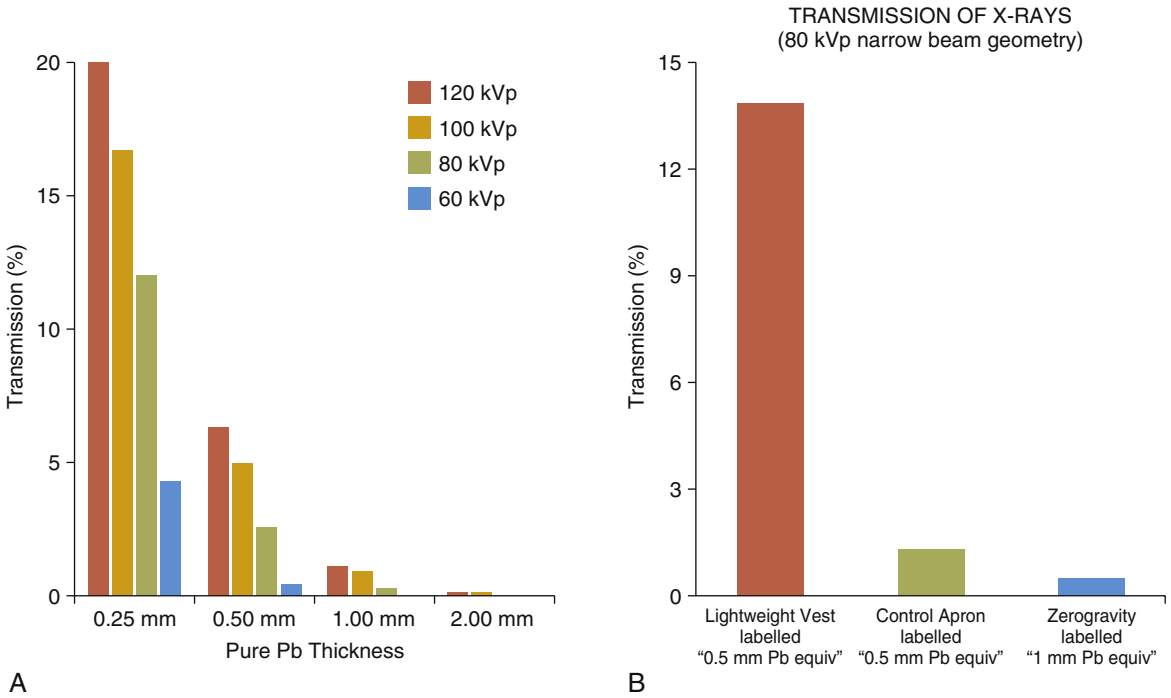
The energy from photons emitted from the anode of x-radiation equipment is 5000 to 75,000 times greater than visible light. X-ray photon energy, described in kilo electron volts (keV), is a spectrum of energy; some photons penetrate tissue only (higher energy), and others contact and stop at the skin, increasing dosage without an effect on imaging (lower energy). Compton scatter, the interaction of x-rays in tissue, describes stray (low-energy keV), nonpenetrating x-radiation that can be damaging but does not contribute to imaging. Higher-energy keV x-radiation penetrates for imaging and is less likely to cause nonpenetrating x-ray exposure. X-ray filtering in modern equipment can help eliminate low-energy nonpenetrating x-ray.

The most simple and least descriptive term regarding dose is *total fluoroscopy time.* This simply measures the total time the x-ray tube emits x-ray photons, without regard to the amount of radiation released. Although often reported clinically and for research purposes, time alone cannot be considered an adequate descriptive measure. Some laboratories, however, use a threshold exposure time, such as 30 or 60 minutes, to trigger termination of a procedure, patient follow-up for signs of radiation injury, or more formal case assessment by the medical physics department.

The standard measurement for the emitted x-ray dose is *air kerma*, generally given in milligrays (mGy), reported for a patient entrance reference point, generally 15 cm above the isocenter of the system. One gray represents 1 joule of energy deposited per kilogram of tissue. Though similar, the *absorbed dose* measures the concentration of energy actually deposited in tissue. This is also reported in milligrays and is used to estimate the biologic risk to the particular tissue. Many systems also report a *dose area product* (DAP), which is the absorbed dose multiplied by the x-ray beam cross-sectional area at that point (mGy/cm^2). The *effective dose* (sievert [Sv] or millisievert [mSv]) represents the estimated whole-body risk based on radiation exposure.[3]

This standard measurement is based on fluoroscopy dose and time. The dose is determined by the intensity of the radiation beam and the proximity to the source and to scatter. It is greater for cineangiography and less for fluoroscopy. Total x-ray dosing depends on equipment-related, patient-related, and procedural-related factors. Some equipment-related dosing is operator dependent, such as fluoroscopic pulse and dose rates and acquisition frame rate. The dose also depends on the tube voltage and current, filtration (to reduce scatter), and beam size. Acquisition dose rate, x-ray beam energy, and beam filtration are generally operator independent.

The principal patient-related factor, body (especially chest) size, affects radiation dosing in a direct and exponential manner; larger patients have more x-ray scatter, which affects contrast, signal quality, and noise. Dosing is modulated by lead



• **Fig. 5.1** (A) Percent radiation transmission through pure leads of varying thicknesses. (B) Percent radiation transmission through commercial operator protective aprons. Zero gravity refers to a counterbalanced, freestanding lead apron that the operator steps into for protection during procedures. (From Rees CR. *Beware the "lightweight lead apron."* Available at http://www.interventco.com/2016/03/01/beware-the-lightweight-lead-apron/.)

gowns, glasses, and thyroid shields, leaving only select parts of the body exposed.

Protective gowns are now made lighter with thinner lead lining or other protective metal barriers. The 0.5-mm lead equivalent aprons remain the standard. While weight reduction is possible in lead-free aprons, a 28% weight reduction of lead-free aprons can increase 70-kVp transmission by 73% and 100-kVp transmission by 31% (Fig. 5.1).[4]

Procedural-related factors such as positioning and proximity of the image intensifier to the patient have a major impact on total fluoroscopy dose. The intensity of exposure is related to the inverse of the distance squared from the radiation source. Oblique angulation, especially ipsilateral to the operator's position, increases scatter and x-ray dose. Beam collimation can reduce scattered radiation and improve image contrast but does not reduce, and may increase, radiation dose rate. Lower-input dose rates and lower frame rates are associated with lower x-ray exposure but may degrade the quality or detail of the image.

Patient Risk From Radiation Exposure

Although ubiquitous in diagnostic and therapeutic procedures throughout numerous medical fields, radiation can cause unintended harm to patients both acutely and remote from the actual procedure. The effects of radiation are generally divided into two types: *deterministic effects* and *stochastic effects*.

Deterministic effects occur when exposure exceeds a predictable threshold level. The most common of these is skin injury, including hair loss, erythema, and desquamation. Radiation-induced skin changes do not occur below an estimated skin dose of 0.9 Gy.

Stochastic effects are long-term effects that manifest years after exposure; these include cancer and genetic effects. As the dose increases, risks increase. There is no certain dose above which a stochastic effect will occur. As these effects are remote temporally from the incident radiation exposure, it is difficult to connect events causally.

Each implanting electrophysiology or cardiology laboratory should work with its institution's medical physicist to develop protocols for following patient exposure to radiation. In addition to reporting in the individual patient record, centralized recording of radiation exposure and dose is important. Possible adverse skin effects from fluoroscopy are summarized in Table 5.1.

Risk to Laboratory Personnel From Radiation Exposure

Laboratory personnel and the implanting staff do not receive any therapeutic benefit from the radiation exposure related to device implantation. Because laboratory personnel are subjected to repeat, albeit more remote, exposures, there are unique risks. These include a higher risk of cataracts, both benign and malignant thyroid neoplasms, brain tumors, and reproductive effects.[5]

Pregnancy in the implanting physician or staff presents a unique and important issue. There are very little data guiding occupational decisions around pregnancy for those working with ionizing radiation; however, pregnant women cannot be

TABLE 5.1 Skin Effects of Radiation

Adverse Effect	Radiation Dose (Gy), Single Exposure	Typical Onset	Peak Onset
Transient erythema	2	Within hours	24 hr post-procedure
Late erythema	6	10 days	2 wk
Temporary epilation	3	3 wk	NA
Permanent epilation	7	3 wk	NA
Dry desquamation	10	4 wk	5 wk
Moist desquamation	15	4 wk	5 wk
Secondary ulceration	20	>6 wk	
Late erythema	15	6–10 wk	
Dermal necrosis (first phase)	18	>10 wk	
Dermal atrophy (first phase)	10	>14 wk	
Dermal atrophy (second phase)	10	>1 yr	
Telangiectasia	12	>1 yr	
Dermal necrosis (late phase)	>15	>1 yr	
Dermal necrosis (late phase)	>15	>1 yr	
Skin cancer	None known	>5 yr	

Modified from Wagner LK, Eifel PJ, Geise RA. Potential biological effects following high x-ray dose interventional procedures. *J Vasc Interv Radiol*. 1994;5(1):71–84.

discriminated against by federal law. Currently, declaration of pregnancy is encouraged as soon as possible to the radiation safety committee to allow for early monitoring for any potential fetal exposure. Ideally, monitoring for potential exposure would begin before conception, although most radiation safety departments do not currently have mechanisms for this.

All personnel working in an environment with ionizing radiation must be given personal radiation dosimeters. Generally, a single badge is worn on the outside of a lead apron at chest or collar height.[6] Additional badges should be worn under the lead skirt for pregnant personnel to measure any fetal exposure. Some operators use a ring dosimeter to track hand exposure, though this is less common.

A monthly report is provided to the department and individual personnel outlining the prior month's exposure, quarterly accumulated exposure, and annual and lifetime totals. Any spike in exposure or passing of predefined limits should prompt investigation and action to decrease exposure immediately. Typical annual and lifetime exposure limits are shown in Table 5.2.

TABLE 5.2 Annual and Lifetime Radiation Exposure Limits

Anatomy	Area Maximum Dose/Year
Whole body	50 mSv
Eye lens	150 mSv
Skin or extremities	500 mSv
Fetus	0.5 mSv/month
Fetus	5 mSv/pregnancy

Data from National Council on Radiation Protection and Measurements. *Radiation Dose Management for Fluoroscopically Guided Interventional Medical Procedures, NCRP Report No. 168*. Bethesda: NRCP Publications; 2010.

Minimizing and Optimizing X-Ray Exposure to Patients and Staff

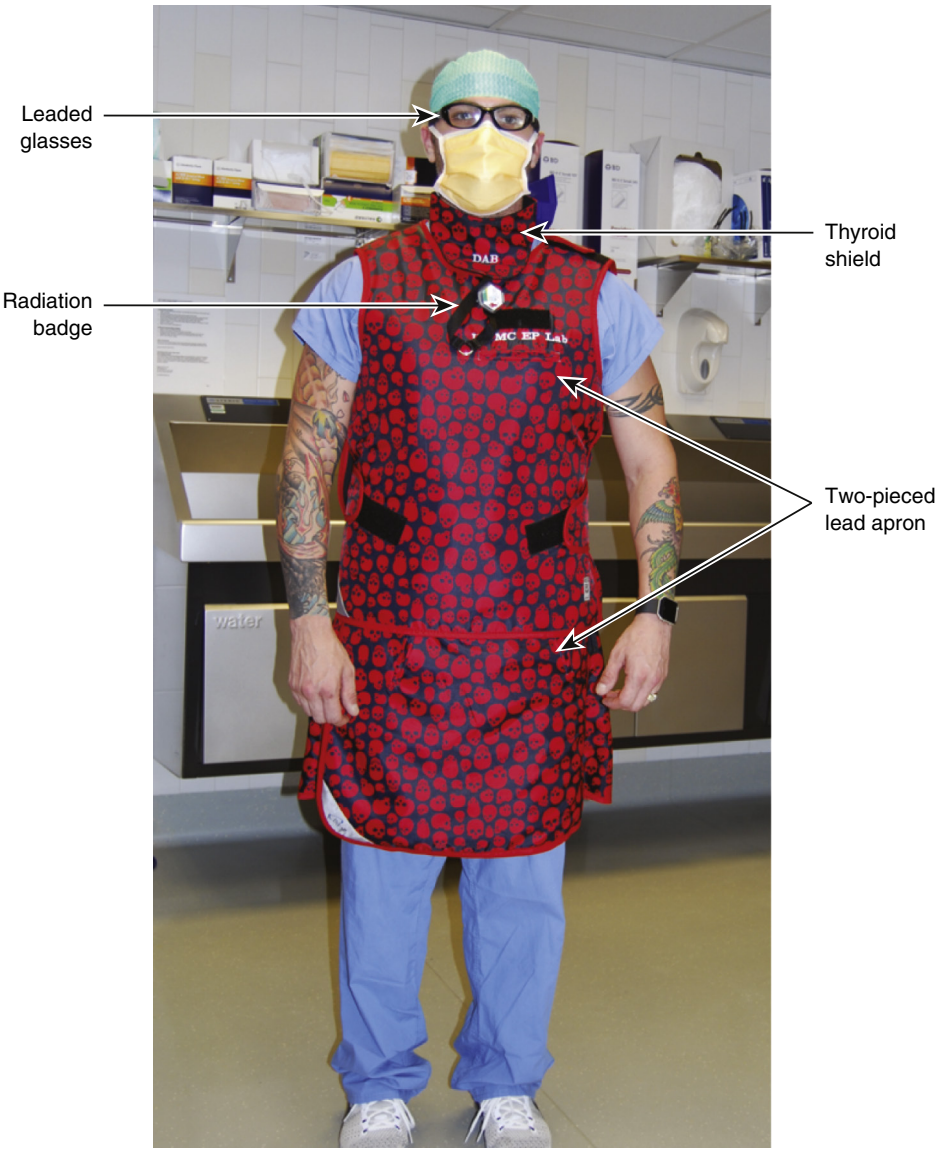
Equipment Operation

Operators and implanters are often unfamiliar with the radiation-minimizing features of an individual x-ray system. Implanting physicians may practice in multiple laboratories with various x-ray systems and find it overwhelming to become educated in the individual settings for each device. Ideally, however, each implanter and staff member running the equipment should receive training and refresher instruction on the optimal operation of the system to adhere to the ALARA (as low as reasonably achievable) principle.[7]

Cardiac rhythm devices should be implanted using x-ray equipment suitable for cardiac procedures. The image intensifier need not be overly large and should be able to achieve a short skin-to-image intensifier distance without interfering with the patient's head. Older equipment may emit higher amounts of x-radiation and have more leakage. Magnification modes should be minimized in systems in which it increases radiation exposure but should be used only when necessary. Beam-hardening filters should be used if available, and collimation should be used if possible. Using fluoroscopy without collimation exposes the patient and the team to excessive and unnecessary x-radiation.

Pulsed fluoroscopy (<8 to 10 fps), low-dose settings (if available), and last-image-hold modes should be used, as these can result in dramatic reductions in radiation. Use of cineangiography should be minimized. Short bursts of fluoroscopy should be used and only when necessary. For critical aspects of the implant procedure, continuous fluoroscopy may be necessary, but even so, it may be needed only for seconds at a time. Short bursts of fluoroscopy can be used, for example, when placing a wire in the superior vena cava or placing a sheath to follow the wire.

Similarly, when placing the lead, fluoroscopy can be used in short bursts and only as a lead is actually entering into the heart. Properly adjusting stylet curvature may reduce the need for manipulation and torqueing when it is in the vein or in the heart; besides minimizing the use of fluoroscopy, this could reduce the risk for lead manipulation inside the heart. One



• **Fig. 5.2** Electrophysiology laboratory staff wearing two-piece lead apron, thyroid collar, and leaded glasses in preparation for a device implantation. A radiation badge is seen at collar level.

should avoid standing on the fluoroscopy pedal unless it is absolutely necessary and anticipate positioning of the fluoroscopy unit before turning it on.

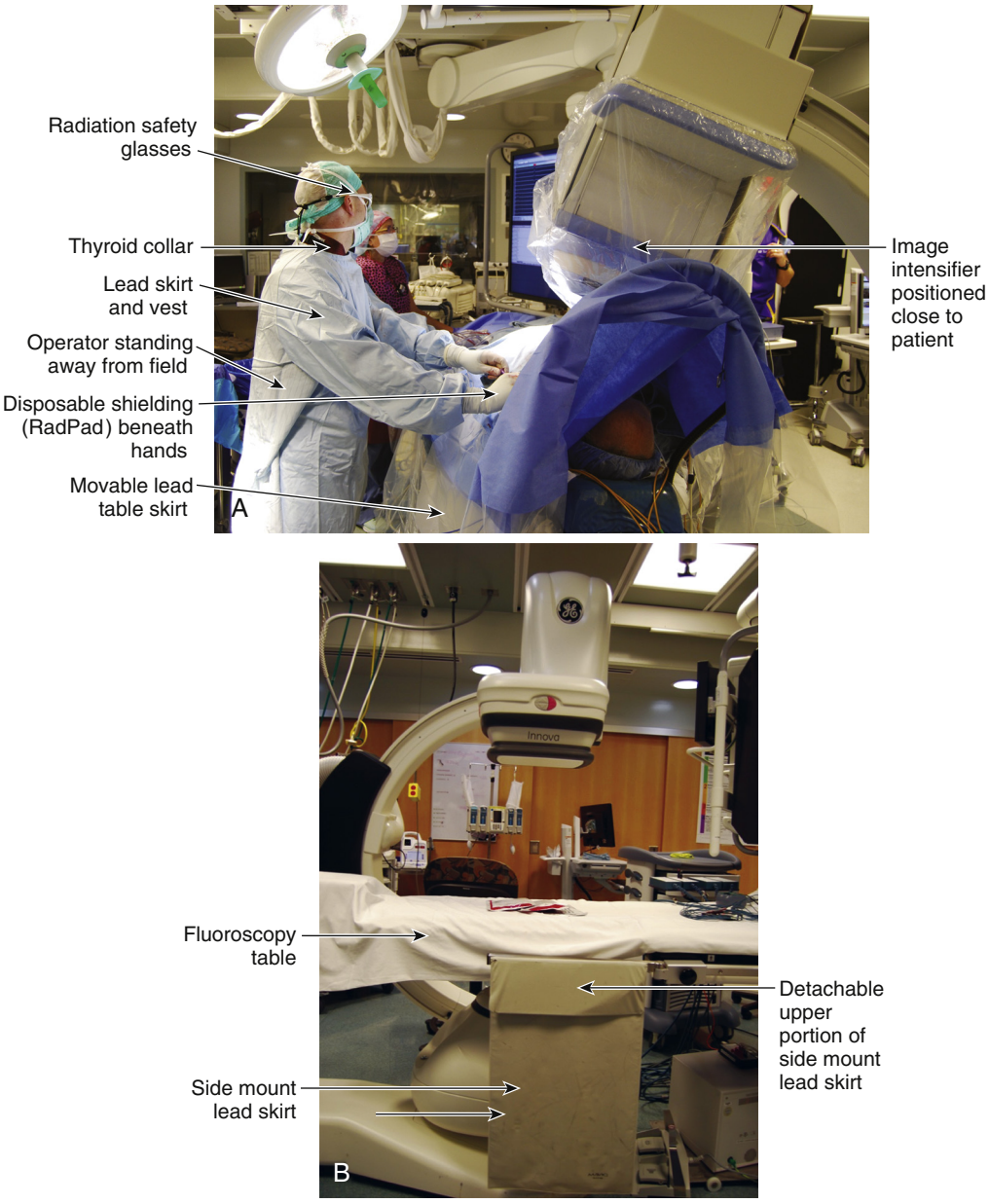
One should center the beam over the location that is needed before using fluoroscopy, rather than moving the table after the fluoroscopy is turned on. The beam should be kept near the equipment's isocenter. The operator must maintain the smallest distance between the patient and the x-radiation image detector that would maximize the distance between the x-ray tube in the patient, and never use fluoroscopy if his or her eyes are not on the monitor. This could save a substantial amount of x-radiation. Hands should be kept out of the fluoroscopy field, and the location and direction of the beam should be altered to avoid constant exposure in one position. Left anterior oblique positioning should be avoided if possible.

The operator should keep track of the use of fluoroscopy and make an effort to minimize its use. He or she should also record the dose and acquisition times for each patient and maintain the equipment and keep it calibrated. Older systems will likely require higher doses of x-radiation. Smoothing images will require higher doses of radiation.

Protective Shielding and Personal Equipment

Everyone working in the implant environment should wear a lead apron and a thyroid collar or be positioned behind a mobile lead shield during an implant procedure (Fig. 5.2). Many operators wear leaded glasses to minimize exposure to the eyes. A lead skirt shield should be positioned at the table and can be attached for convenience during the procedure (Fig. 5.3). Lead shields should be tested intermittently to make sure that there are no radiation leaks that develop through flexible x-radiation protection.

Occupational radiation exposure can also be reduced by the use of a bismuth-containing, sterile disposable shield. When



• **Fig. 5.3** Fluoroscopy table with side-mount lead skirt. Note the detachable upper portion, which significantly reduces scatter radiation exposure.

positioned just caudal and lateral to the intended device pocket region, these radiation-absorbing shields can decrease absorbed radiation to the operator by up to 80%.[8] Widespread use of this device may be hampered by cost considerations.

Recent data suggest that radiation (some from scatter) to the brain (particularly the left side of the brain for right-sided implant procedures and vice versa) may be detrimental. Head caps of flexible barium sulfate and bismuth oxide strips, constructed with lightweight cloth, can attenuate radiation to the brain equivalent to a lead that is 0.5 mm thick.[9] Full radiation protection cabins have been shown to reduce exposure to the operator by over 90%, without increasing procedure time or complication rates.[10] Despite this finding, they remain costly and somewhat cumbersome, limiting their overall use. Standing behind another individual in closer proximity to the radiation field may offer no protection. Instead, perhaps through scatter and more favorable positioning of protective devices toward the primary operator, radiation exposure may actually be higher for a second operator.[11] This has important implications for those in a position of instruction and emphasizes the need for optimal use of shielding protective for both the primary and secondary operators.

Bismuth radiation pads used to reduce x-radiation exposure to a patient's breasts and eyes for computed tomography scans have been proposed and are controversial but to date have not been used for cardiac implantable electronic device (CIED) implantation.

Careful use of fluoroscopy can reduce the amount needed and not affect the ability to perform the implantation and not

TABLE 5.3 Strategies for Reducing Overall Radiation Exposure During Device Implantation

Angulation of imaging	Avoid left anterior oblique; keep to <50 degrees
Minimize scatter	Lower the image intensifier
Collimation	Avoid nonessential structures in the image, especially lung fields
Distance	Maximize distance between operator and x-ray source
Minimize pedal-on time	Short bursts, remind assistants
Low frame rate	≤7.5 fps

adversely affect the safety to the patient. Experienced implanters can routinely implant single- and dual-chamber pacemakers and implantable defibrillators with less than 1 to 2 minutes of fluoroscopy, and in some cases only a few seconds. Table 5.3 provides recommendations for CIED implantation to reduce radiation exposure.

Fluoro-less Device Insertion

Initial enthusiasm for radiation-free catheter ablation procedures, utilizing three-dimensional mapping systems, has spilled over into device implantation. In most patients, the risk/benefit analysis favors using fluoroscopy but in the lowest amount possible. However, in certain circumstances, it may be desirable to not only minimize fluoroscopy but also eliminate it altogether, such as in the case of a pregnant patient needing a device implantation.

There have been numerous case reports and series of fluoro-less device implantations. Most have used a mapping catheter to create an anatomic shell of the chamber(s) of interest in one of the electroanatomic mapping systems. The lead is then connected to the system in a manner that allows for visualization of the lead tip as it enters the chamber. Nonfluoroscopic electromagnetic tracking systems have also been utilized.[12] These systems can be used to augment fluoroscopy and minimize overall exposure.[13] Intracardiac ultrasound has been used as an additional tool.

While these techniques have merit, it is our practice to reserve them for the most sensitive cases, such as that of the pregnant patient. Dual-chamber pacemaker or implantable cardioverter-defibrillator (ICD) implantation, for example, can often be accomplished with minimal fluoroscopy and an equivalently small dose to the patient.

Cardiac Resynchronization Therapy

One procedure deserves special consideration. Cardiac resynchronization therapy (CRT) devices involve implantation of not only a right atrial and right ventricular lead but also a left ventricular lead. Fluoroscopy exposure can increase significantly if there is difficulty in cannulating the coronary sinus, advancing the guiding sheath into the distal vein, using cineangiography runs to delineate venous anatomy, advancing the pacing lead into a suitable branch, or repositioning and stabilizing the left ventricular lead after initial placement and peeling the sheath.

Angulation of the imaging plane can increase overall radiation dose to the patient greatly. This is especially important in CRT implants, as left anterior and right anterior oblique views are used frequently for optimizing the anatomic evaluation of the coronary venous tree. Although there is a strong learning curve on overall radiation dose during CRT implantations, exposure remains relatively high even among experienced operators.[14,15] Use of low-dose protocols during these procedures is especially important for minimizing overall risks to personnel and patients.[16]

Summary

Radiation exposure, an inherent part of modern cardiac rhythm device implantation procedures, adds risk to the patient and the staff involved. Knowledge of common measurements of radiation exposure and careful use of fluoroscopy and cineangiography is necessary to minimize these risks. Implanting physicians must familiarize themselves with the features of the individual x-ray systems they are operating to optimize procedural success while minimizing harm.

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6

Patient Preparation

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Introduction

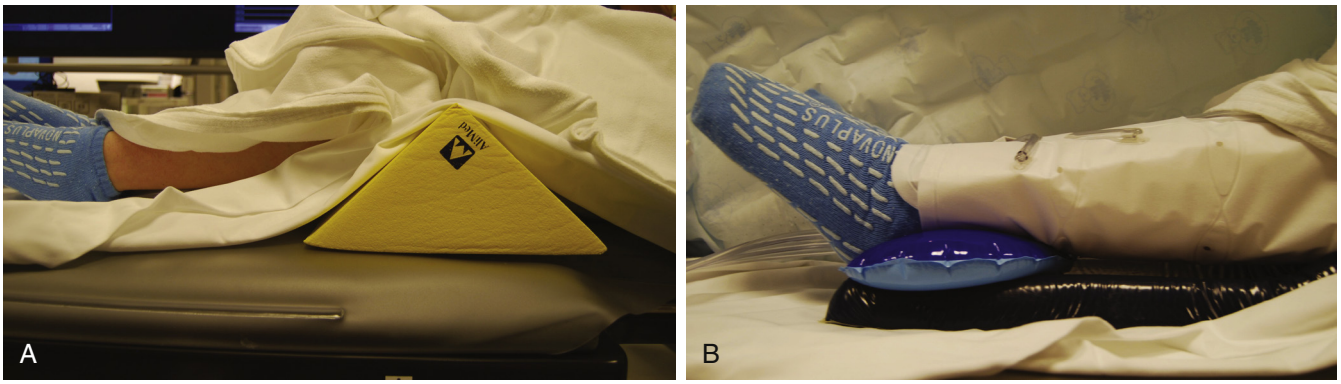
A safe and successful surgical procedure depends on careful patient preparation. Procedures may be delayed or prolonged owing to an inability to recognize or correct suboptimal patient positioning, improper placement of monitoring electrodes or lines, or absence of an electrocautery grounding pad once the surgical drapes have been placed on the patient. Moreover, erroneous or improper monitoring electrodes and lines may place the patient at risk should there be an acute change in the patient's rhythm, hemodynamic, or neurologic status. Regardless of the skill set of the operating physician, his or her efforts may be thwarted if the implanted rhythm device becomes infected from poor sterile technique. Finally, if the patient is uncomfortable because of inadequate pain control, sedation, or positioning, he or she may make abrupt or continuous movements during the course of the operation, which may increase the risk for serious complications such as pneumothorax, vessel laceration, and myocardial perforation. This chapter reviews appropriate patient preparation procedures, including positioning, monitoring, and using sterile technique such as gowning, gloving, and draping.

Patient Identification

Before the start of the procedure, a preprocedure review is performed. This may be known as a "time out," "huddle," or Surgical Clinical Outcomes Assessment Program (SCOAP). The purpose of this preprocedure review is to document the patient's identification, the procedure being performed, and the laterality of the procedure if appropriate, and to be certain that all necessary preparations have been completed. All music, conversation, and distractions should be suspended while this review is being conducted. Most procedural labs conduct the review at two specific time points. The first is before preparing and draping the patient and induction of conscious sedation or general anesthesia. The patient's identity (name, date of birth, medical record number) is confirmed by two personnel, one of whom should be a member of the surgical team. A current, signed consent form is verified, and allergies are reviewed. Anesthesia equipment is documented to be present and fully functional. All necessary personnel should be present, including the industry representatives, who should confirm that all necessary leads, devices, and specialized implantation hardware and equipment are present and in the room. If the patient is pacemaker dependent, programming to an asynchronous mode or using a transvenous temporary pacing wire should be discussed before the procedure starts. If the patient has an indwelling implantable defibrillator, tachyarrhythmia detection and therapy should be disabled and programming confirmed with the surgical team. Other important aspects of the preprocedure evaluation are airway and aspiration risk and whether a plan for deep vein thrombosis (DVT) and pulmonary embolus (PE) prevention will be required with the use of sequential compression devices. The second review is performed by the attending physician before the start of the procedure. The components of this review include reconfirming the patient's identification, the procedure to be performed, and that all procedure-specific equipment is present, antibiotics have been infused, a sharps management plan is in place, a diabetes plan is prepared if appropriate, and active warming is turned on if needed. Any clinical comorbid risk factors such as diabetes, recent dialysis, or recent cardiovascular events should be identified and a plan made for management of possible complications as appropriate.

Patient Positioning

Proper patient positioning provides optimal access for the implanting physician, his or her assistant(s), and the anesthesia team. It is also important to ensure that the patient is positioned properly relative to the imaging equipment and that patient comfort is maximized. Poor positioning can result in inadequate access to the surgical field, increased risk of contamination and infection, and pressure point damage and/or nerve injury. Pressure points include bony prominences such as the occiput, scapulae, olecranon, thoracic vertebrae, sacrum, coccyx, and calcaneus, as well as peripheral vessels and nerves such as the brachial plexus and ulnar nerves. Interventions for at-risk areas include pressure-reducing mattresses, additional padding to the extremities with gel pads, bean bags, various-shaped foam for specific areas, soft towels, pillows, and small wedges (Fig. 6.1A–B). Arm boards should be level with the mattress and the arms tucked at the patient's



• **Fig. 6.1** Protective padding of pressure points. (A) Foam padding. (B) Gel padding.

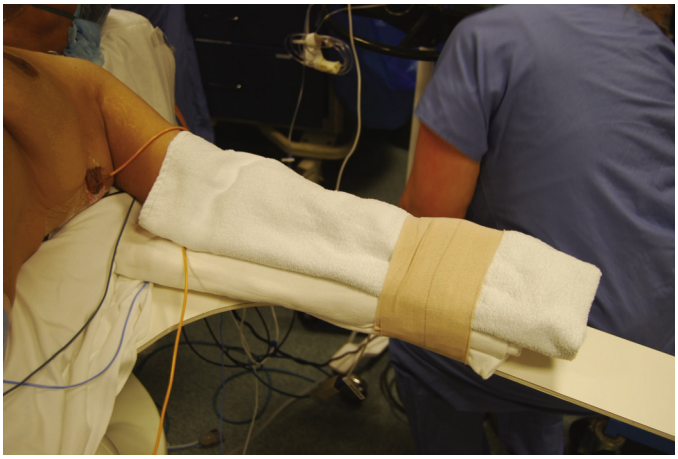
sides. A pillow or small wedge placed under the calves or knees may increase patient comfort in those with lower back issues and simultaneously decrease pressure on popliteal and heel areas.

When electrocautery is anticipated, care should be taken to ensure that no part of the patient's body is in contact with a metal surface, which could cause an electrical burn due to electrical current shunting. Similarly, all jewelry should be removed to prevent thermal injury.

Special positioning is required for the subcutaneous implantable cardioverter-defibrillator (ICD; see Chapter 10). The patient is placed in the supine position with the left arm abducted between 45 and 90 degrees on a padded arm board placed level to the mattress. Soft restraints or rolls of cast padding can be wrapped around the left wrist with the palm up to avoid ulnar nerve compression (Fig. 6.2). The right arm is typically tucked at the patient's side. It may be useful to tilt the patient toward the left side slightly for additional lateral chest wall visualization and accessibility to aid device pocket formation.

Placement of Monitoring Leads

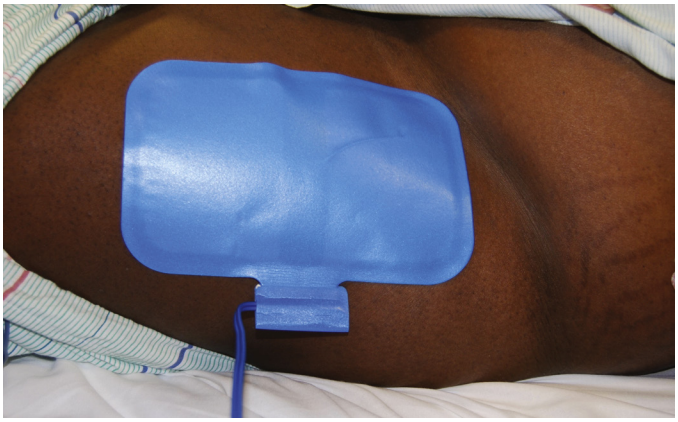
The 12-lead electrocardiogram (ECG) remains a cornerstone of cardiac diagnostics and is an important part of the cardiac implantable electronic device (CIED) implant procedure. Standard ECG electrode placement is used, or modified if needed for the surgical field. A baseline 12-lead ECG should be recorded upon arrival to the procedural room and examined by the nurses and technician staff. The supporting staff in the electrophysiology procedure lab are trained to be competent with basic electrocardiography, rhythm interpretation, and conduction abnormalities. The procedural support staff may observe a change in or abnormality of the patient's rhythm and can then bring these observations to the physician's attention before the procedure starts. Furthermore, observation of a preprocedure left bundle branch block will alert the team to be prepared for emergency pacing in the event of catheter-induced right bundle branch block. The nursing and technician support staff also are expected to monitor the patient's rhythm during the procedure and to alert the operating team to any changes as appropriate.



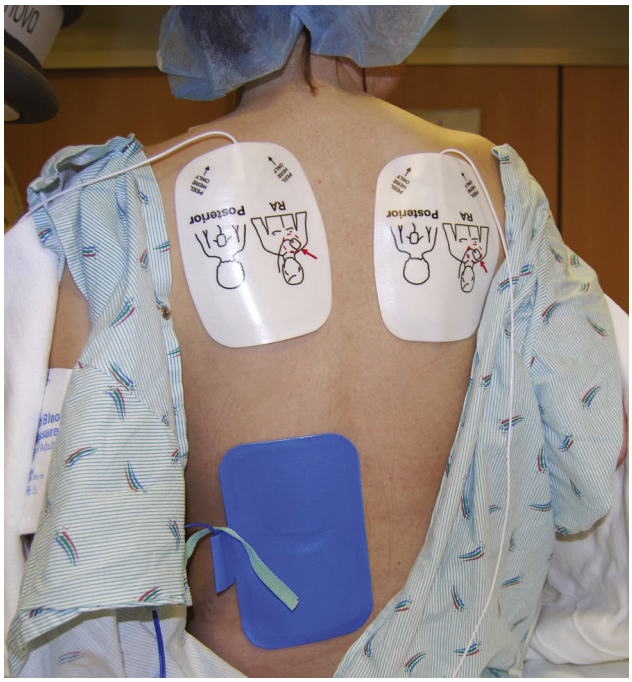
• **Fig. 6.2** Padding on arms used as self-restraints.

Placement of Electrocautery Grounding Pad

During monopolar electrocautery, current passes from the electrosurgical unit to an active electrode (handheld pen), through the patient to a grounding pad (dispersive electrode or return electrode monitor), and back to the electrosurgical unit to complete the circuit. Safe use of electrocautery depends on proper grounding pad placement. The grounding pad should be placed close to the operative site on the ipsilateral side of the surgical field. Whenever feasible, the grounding pad should be positioned on well-vascularized areas of the body, preferably with substantial muscle mass, to dissipate heat more readily (Fig. 6.3). Bony prominences and areas of significant adipose tissue should be avoided because they are poor electrical conductors. Areas of excessive hair, scars, and other poorly perfused sites also do not dissipate heat well and may contribute to thermal burns. Care should be taken to ensure that the grounding pad firmly adheres to the patient's skin without any folds or gaps, which could trap alcohol-based antiseptic preparations. The combination of evaporating alcohol, supplemental oxygen, and electrocautery sparks in the surgical field is a significant fire risk. Adhesive barriers applied over the grounding pad before application of the surgical scrub solution are recommended to decrease this risk. Electrocautery concepts and use are discussed in detail in Chapter 3.



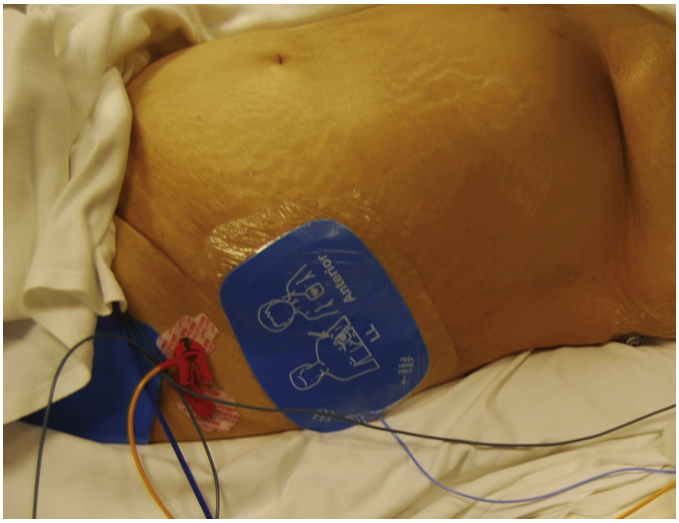
• **Fig. 6.3** Electrocautery grounding pad placement.



• **Fig. 6.4** Placement of two sets of defibrillator pads with grounding pad, posterior view.

Placement of Defibrillator Patches

Biphasic external defibrillators, present in all cardiac electrophysiology laboratories, are attached to the patient using adhesive electroconductive pads to provide emergent transcutaneous pacing, cardioversion, and/or defibrillation during the course of a cardiac rhythm device implantation. For patients at significant risk of ventricular arrhythmias and when induction of ventricular fibrillation for defibrillation testing is anticipated, two sets of defibrillator pads should be placed with opposing vectors (Fig. 6.4). In low-risk patients, a single set of defibrillator pads may be placed, and a second external defibrillation system immediately available. Electroconductive pads are standardly used with external defibrillation as they are safer, are less subject to user error, and have more consistent skin interfaces. The defibrillator pads are placed before preparing and draping the patient to keep them out of the surgical field. Anterior-posterior chest or left-lateral to right-posterior chest positions are preferred placement sites during device implantation procedures to incorporate as much myocardial tissue as possible for optimal defibrillation vectors. Morbidly obese patients and women with large breasts may require modification of defibrillator pad placement to optimize the defibrillation vector (Fig. 6.5).



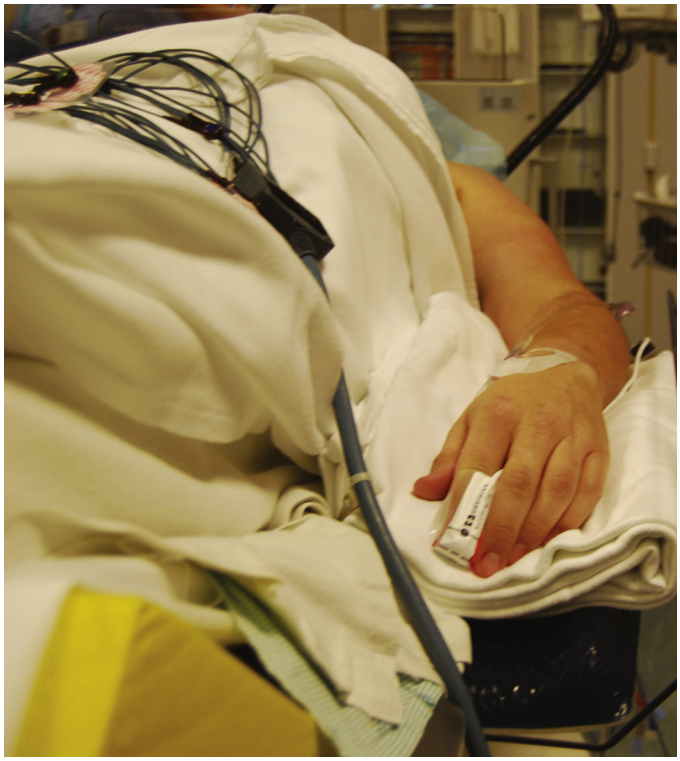
• **Fig. 6.5** Defibrillator pad placement for the left lateral subcutaneous implantable cardioverter-defibrillator.

Restraints

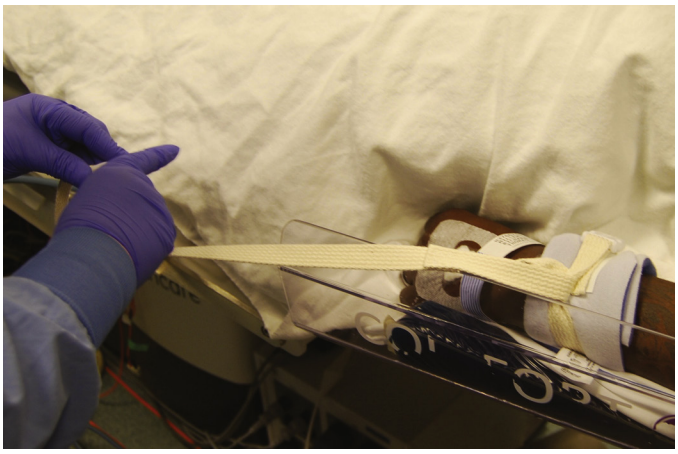
Patient safety is maintained with the use of soft restraints and should be considered in any case where the patient's cognition or ability to lie still is expected to be impaired (sedation, psychiatric illness, anxiety, back pain). Assessment of anxiety level, medical and psychological history, age, and prior response to narcotics and/or sedatives is useful to determine the need for restraints. Several options may be employed separately or together. A transfer sheet or blanket tucked under the patient's arms is commonly used and often all that is required (Fig. 6.6). If the patient's cognition is altered owing to sedation, the patient moves too much, or the patient becomes combative, soft wrist restraints with quick-release capabilities (Fig. 6.7) can be used. A security strap or wide Velcro-type fastener wrapped around the patient's legs or waist may further help a patient who is having difficulty remaining immobile during the procedure (Fig. 6.8).

Respiratory Monitoring

Pulse oximetry and end-tidal CO_2 are the hallmarks of respiratory monitoring. Pulse oximetry measures the degree of oxygen saturation (Spo_2) and is an estimate of the functional oxygen saturation (Sao_2) of hemoglobin in arterial blood. It is based on the fluctuation of the cardiac cycle, which creates a pulsatile wavelength. The wavelength is read as a change in amplitude of the absorbance of red to near-infrared light. A modulation ratio is calculated over a series of pulses and determines the Spo_2 based on a calibration curve. The resulting measurement is expressed as an O_2 saturation percentage.[1]



• **Fig. 6.6** Arms tucked at sides to prevent movement and allow room for operator to stand near surgical field.

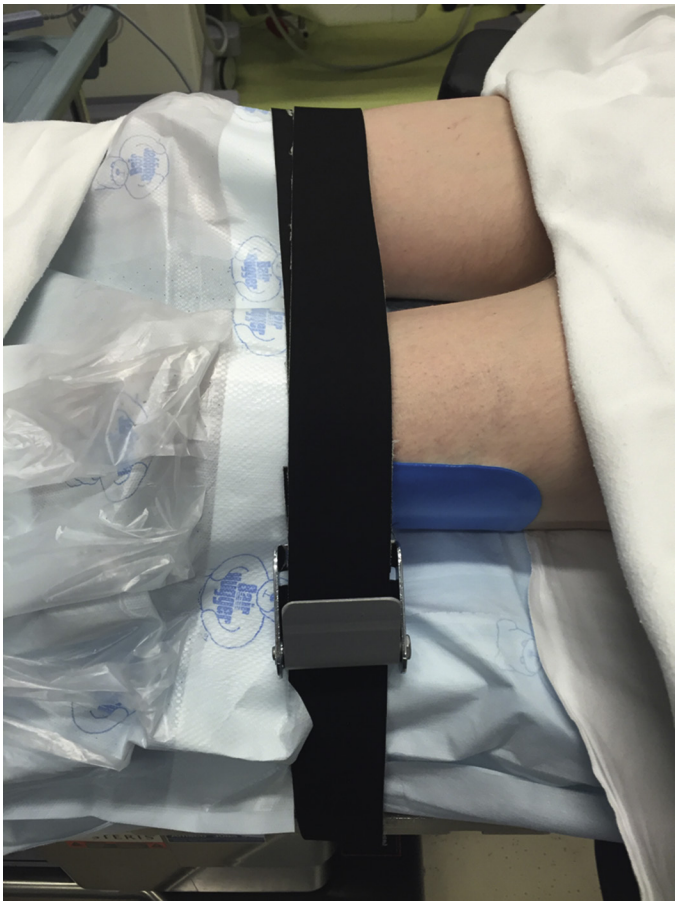


• **Fig. 6.7** Soft restraints applied on wrists.

End-tidal CO_2 ($ETco_2$) reflects the level of carbon dioxide gas released at end expiration. Because it provides a more rapid detection of respiratory depression or apnea leading to hypoxia than pulse oximetry, the American Society of Anesthesiologists (ASA) now requires capnography for monitoring moderate to heavy sedation unless precluded or invalidated by the nature of the patient, procedure, or equipment.[2] Practice Guidelines for Sedation and Analgesia by the Non-Anesthesiologist are available at http://www.anesthesiology.org.

Moderate Sedation

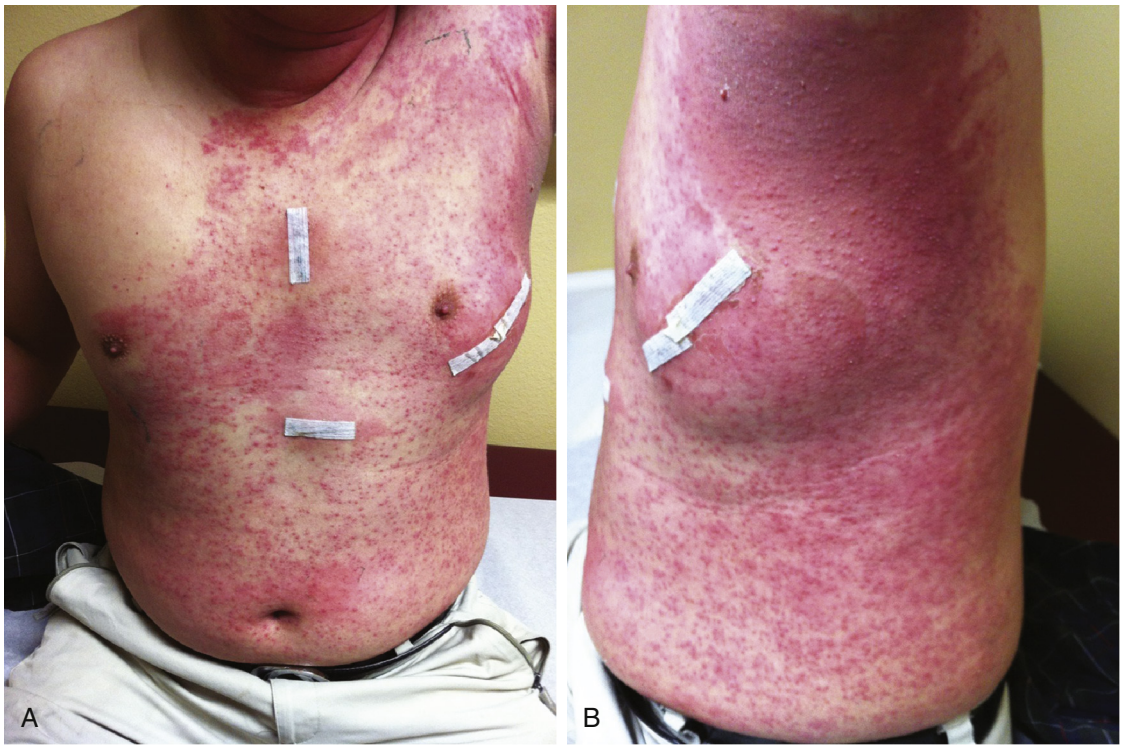
Moderate sedation within the electrophysiology laboratory has become an important responsibility of registered nurses



• **Fig. 6.8** Leg restraints.

(RNs) and certified registered nurse anesthetists (CRNAs). The 2014 Heart Rhythm Society Expert Consensus of EP Laboratory Standards recommends, "An RN should be present for every implant in the EP Laboratory. The nurse (either RN or CRNA) is the primary individual responsible for the direct observation, sedation and nursing care of the patient during the EP procedure and must be prepared to respond to any emergency."[3] The extent to which each nursing specialty may administer conscious sedation is governed by state licensure and the specific institution's policies and bylaws. To ensure a uniform standard of care, RNs function in accordance with the ASA Practice Guidelines for Sedation and Analgesia by Non-Anesthesiologists and the Center for Medicare and Medicaid Services (CMS) Interpretive Guidelines for Hospitals. For any procedure that will include moderate conscious sedation, the ASA classification and Mallampati score for the patient must be assessed preoperatively (see Chapter 4). If this assessment identifies a higher-risk patient, alternate plans for deeper sedation to optimize patient safety, made with the assistance of anesthesia personnel, may be required.[3]

Once the patient is positioned, monitoring leads are in place, an electrocautery grounding pad and defibrillation/external pacing patches are applied, and restraints (if needed) are attached, a final check of the patient's cardiopulmonary status is performed. At this point, appropriate sedation may begin.



• **Fig. 6.9** (A–B) Allergic reaction to chlorhexidine surgical prep.

Scrub Antiseptics

Bactericidal skin preparatory solutions are used for surgical hand scrub and on the patient's skin before draping. Available agents have a long antibacterial effect (bacteriostatic) and leave a barrier on the skin that keeps resident bacteria to a minimum.[4,5] Additionally, they should be nonirritating to tissue and not elicit a topical or systemic allergic response (Fig. 6.9). Three common skin preparatory solutions are 7.5% povidone-iodine, 4% chlorhexidine, and 3.3% chloroxylenol.

Povidone-iodine (Betadine) is a stable complex containing povidone (polyvinylpyrrolidone), a synthetic polymer vehicle widely used in the pharmaceutical industry, and elemental iodine. Povidone-iodine is water soluble and effective against vegetative bacteria, *Mycobacterium tuberculosis*, most viruses, and fungi. It is classified as an antiseptic with germicidal efficacy with no sporicidal capability.[6] It can be used on open wounds, granulation tissue, and mucosa, and has been used for pleurodesis. Its use is contraindicated in patients with allergies to iodine, following treatment with radioiodine, and in diseases of the thyroid (hyperthyroid) as absorption of elemental iodine may occur.

Chlorhexidine is a cationic polybiguanide alcohol-based antiseptic with a 70% or 90% isopropyl alcohol and chlorhexidine combination. Its salt dissociates at physiologic pH, releasing positively charged ions that bind to bacterial cell walls, providing a prolonged bacteriostatic effect.[9] Both chlorhexidine and alcohol are water soluble and provide germicidal, bactericidal, tuburculocidal, fungicidal, and virucidal efficacy. When applying an alcohol-based antiseptic disinfectant, it is important to let the skin dry completely before draping due to a potential fire risk with the combination of oxygen, cautery, and alcohol vapors. The Centers for Disease Control and Prevention (CDC) recommends in its Guidelines for the Prevention of Surgical Site Infection (SSIs) that for any surgical procedures below the chin area, the patient should perform two preoperative showers with 4% chlorhexidine gluconate (CHG) within 12 hours of elective surgery.[10] Although one large study showed no difference between povidone-iodine and chlorhexidine in preventing implantable device infections, in several other studies, povidone-iodine was associated with more infections than chlorhexidine.[7,8,11]

If the patient is allergic to both povidone-iodine and chlorhexidine, chloroxylenol (cocamidopropyl dimonium-chloridephosphate) may be used. Like chlorhexidine, its ionic properties allow binding to cell membranes, providing bacteriostatic protection for more than 6 hours. It does not contain alcohol but is formulated as an emulsion, requiring longer drying times, and provides less adhesion for the surgical occlusive drapes. Properties of the surgical skin preparations are compared in Table 6.1.

Surgical Field Preparation

All principles described next are applicable to the nurse or technician preparing the surgical table, as well as the physician and any assistants performing the procedure.

While the surgical team scrubs, the circulating nurse/technician can prepare the surgical site with the appropriate surgical scrub while another nurse/technician prepares the surgical table.

Scrubbing and Gowning

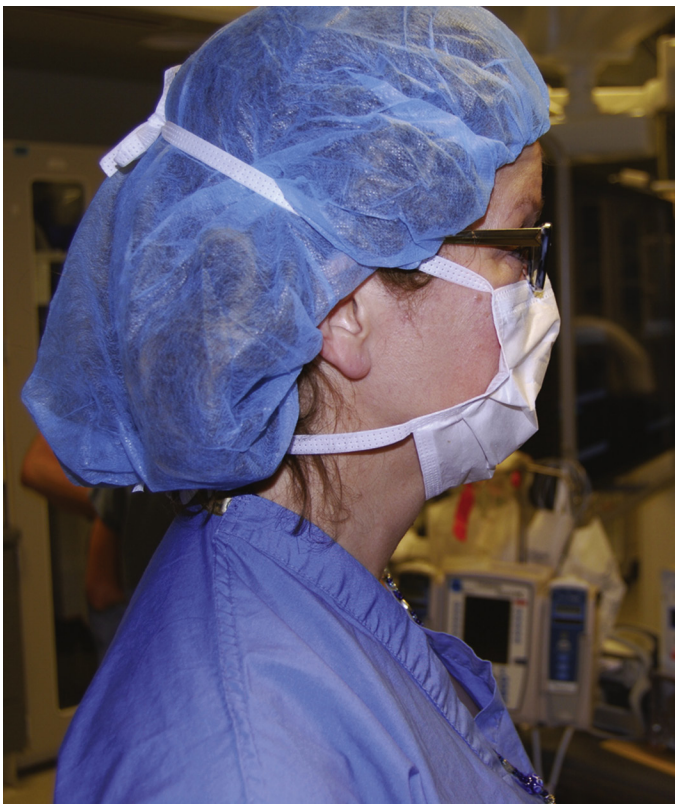
Each person who is going to scrub should inspect his or her hands to ensure the nails are short, the cuticles are in good

TABLE 6.1 Comparison of Surgical Prep Solutions

Product	Time to Optimal Effect	Persistence	Required Contact Time	Required Drying Time
Povidone-iodine	3 min	Washes off if wet (blood, sweat, irrigation)	5 min	Dependent on multiple factors[a]
Chlorhexidine	3 min	>6 hr	4 min	Dependent on alcohol content
Chloroxylenol	30 s	>6 hr	2 min	Emulsion, does not completely dry

Modified from One-stop guide to surgical skin preps. *Outpatient Surgery Magazine.* Available at: https://www.outpatientsurgery.net/resources/forms/2005/pdf/OutpatientSurgeryMagazine_0502_SkinPreps02.pdf

[a]Ambient temperature, humidity, surgical site, patient's skin.



• **Fig. 6.10** Surgical team member with jewelry removed, wearing hat, mask, and eye protection.

condition, and no cuts or skin problems exist. All jewelry on the hands and forearms needs to be removed, and all head and facial hair should be covered before donning a surgical mask and protective eyewear (Fig. 6.10).

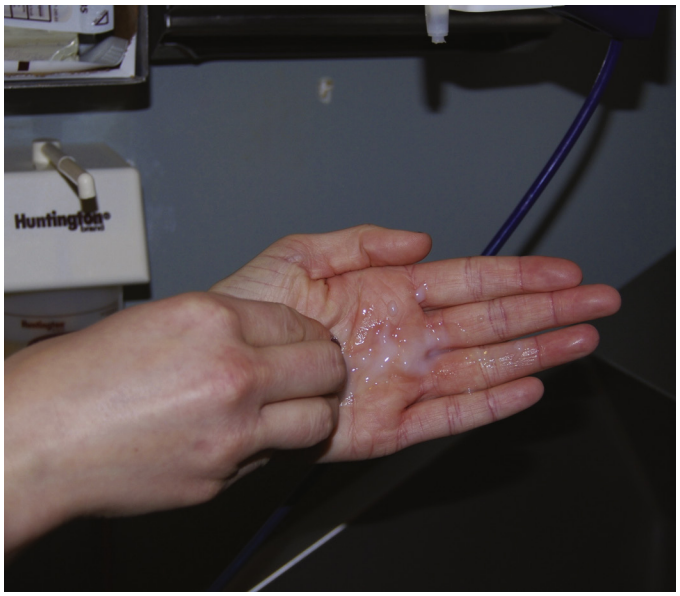
Before running the hands and arms under warm water at the scrub sink, the clinician opens the scrub brush/sponge package and removes the antimicrobial soap–impregnated brush and nail cleaner. If the brush is not impregnated with antimicrobial soap, soap is applied to the brush using the foot control pump from the wall-mounted dispenser. The clinician rinses his or her hands and arms under the running water, cleans beneath each nail with the nail cleaner, and then washes his or her hands and forearms with the antimicrobial soap.

Two methods are acceptable for surgical scrubbing, either the timed or the number-of-strokes method.[12] The timed

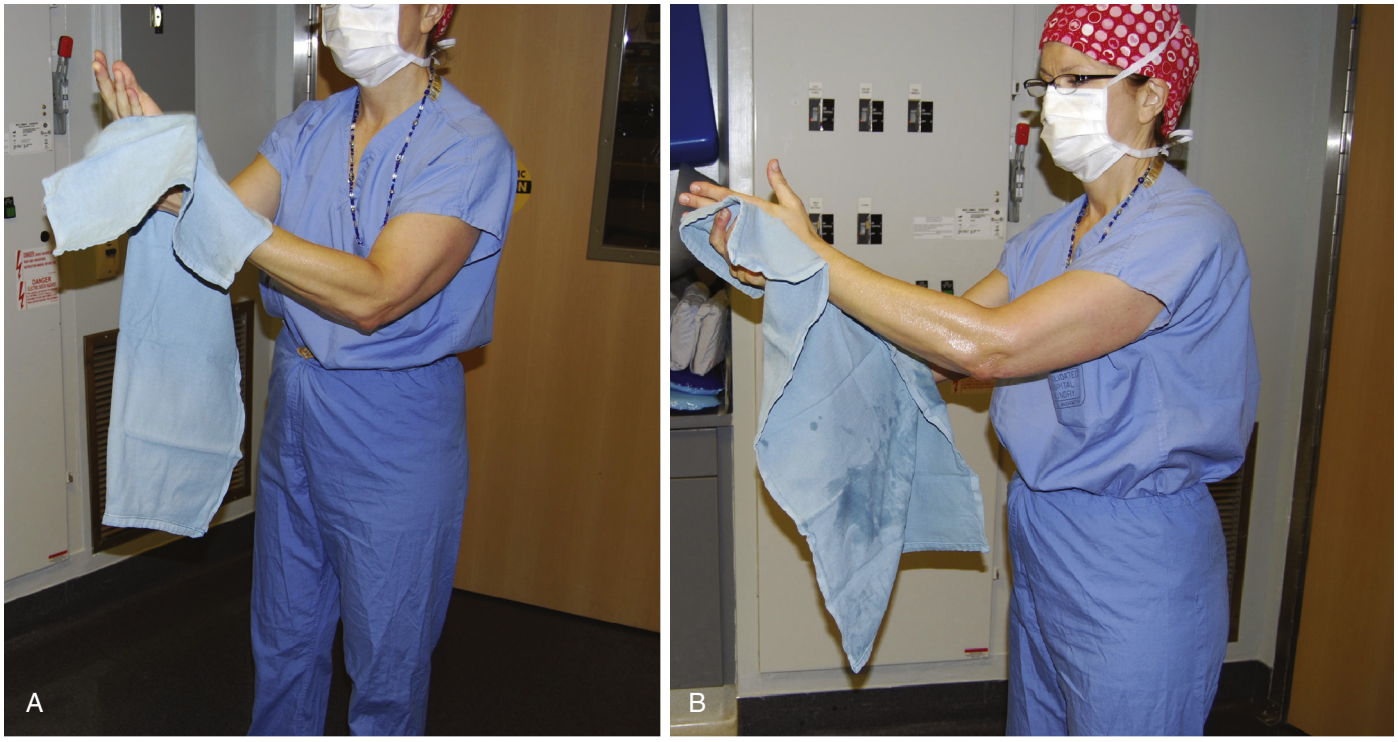
• **Fig. 6.11** Completing surgical scrub at scrub sink.

method includes 5 minutes for the initial scrub of the day and 3 minutes for each successive scrub. If using the number-of-strokes instead of the timed method, 30 brush strokes to the nails and 20 to the other areas of the hands, forearms, and elbows are required. It is important to avoid splashing the surgical attire while scrubbing as the scrub soap and water could splash back on the previously cleaned areas. After the antimicrobial-impregnated brush has been moistened, the scrubbing begins at the fingertips, the nails, and all sides of each digit including webbed spaces. Next, the palm and back of the hand are scrubbed. When the hand scrub is completed, the person progresses up each side of the forearm with a circular motion up to the elbow. The same procedure is repeated on the other hand and arm. While scrubbing, it is important to hold the hands and arms away from the body with the hands above the level of the elbows. This allows the water to flow from the first-scrubbed and cleanest area to avoid contamination. When the prescribed time or number of strokes has been reached, the person rinses his or her hands and arms thoroughly, ensuring that they are kept up and in front of the body (Fig. 6.11).

Many institutions have instituted a waterless hand scrub using chlorhexidine gel as an option or replacement to the



• **Fig. 6.12** Hand scrub with waterless gel prep.



• **Fig. 6.13** (A–B) Sequence of drying hands with towel.

traditional hand scrub. Following the manufacturer's written directions, the recommended amount of hand rub gel (3–5 mL total) is worked into the fingertips and fingernails (Fig. 6.12). The hands are used to rub the gel into the skin and cover all surfaces to the level of the elbows, including the backs of hands, fingertips, inner webs, and palms, until the hands are dry. One study showed that the waterless hand scrub technique is as effective as the traditional hand scrub in cleansing the hands of microorganisms and more efficient in terms of scrub time.[13]

A sterile gown and set of sterile gloves should be opened on a flat surface within the procedure room by a circulator. The folded towel that accompanies the gown is grasped near the open corner and lifted straight up and away from the sterile gown to prevent contaminated water from dripping on the sterile field containing the gown and gloves. This is achieved by stepping away once the towel is grasped and bending forward from the waist while holding the hands and elbows above the waist and away from the body. The top half of the towel is held with one hand and the opposite hand is dried to the forearm. The dried hand then grabs the lower half of the towel and repeats this procedure on the other hand and forearm. The towel is disposed of without the hands dropping below the level of the waist (Fig. 6.13).

• **Fig. 6.14** Circulating personnel tying surgical gown.

The gown is donned by the individual, with care taken not to touch any unsterile surface. A circulating staff member ties the two ties at the neck and one at the waist before gloving, again with care taken not to touch any sterile surface (Fig. 6.14). Gloves may be placed by a sterile assistant or using the closed glove technique (Fig. 6.15). Sterile gowns are considered sterile from the front shoulders to the level of the sterile field and at the sleeves from 2 inches above the elbow to the cuff. The scrub person should not allow his or her hands to fall below the level of the sterile field or the waist level to maintain sterility. The sleeve cuff, neckline, shoulders, axillary area. and back are considered unsterile (Fig. 6.16).

Preparation of the Patient

The skin preparation begins at the proposed site of the incision and continues beyond the periphery of the surgical area (Fig. 6.17). When prepping, staff wear sterile gloves while applying the antimicrobial agent, taking care not to touch any area of the prepped surgical site. If alcohol-based preparations are being applied, the skin surface should be allowed to dry completely before a second application. The second application should dry for a minimum of 3 minutes before placement of the surgical drape to prevent inadvertent pooling of solution under the drape, which could present a risk of fire (Fig. 6.18).

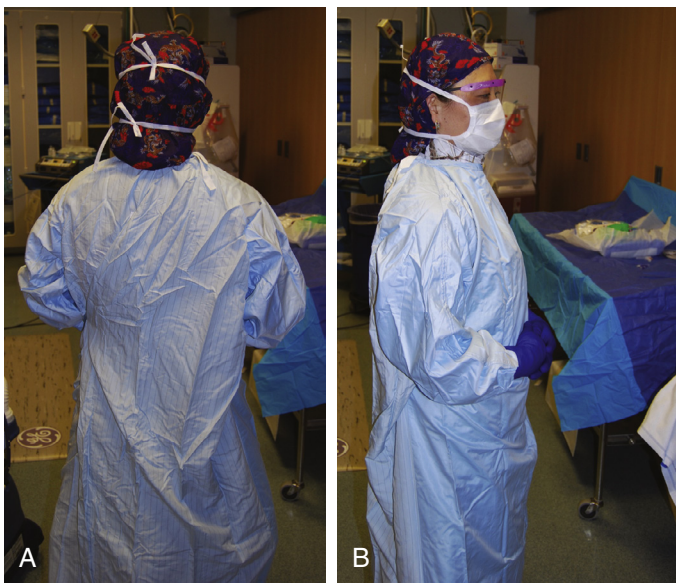
The surgical table containing the appropriate equipment and instruments necessary to perform a device implantation is prepared by a scrubbed individual while the circulating nurse or technician applies the first of two surgical prep solutions on the surgical site. This should be performed in a standard manner such that each individual of the surgical team is familiar with the presence and location of each instrument, suture, and so forth, present on the table. Each institution will have a specific preference on table setup and instruments used (Fig. 6.19).

Individuals handing off sterile items to the sterile field must check for package integrity and expiration dates before placing them on the sterile field. Many institutions have the individual who is handing the items announce the expiration dates on packages before they are opened to be certain that expired product is not inadvertently implanted. Supplies of doubtful sterility must be assumed unsterile. For instance, if an item is found with an expired sterility date or without an internal indicator to verify sterility, the supply item should not be used.

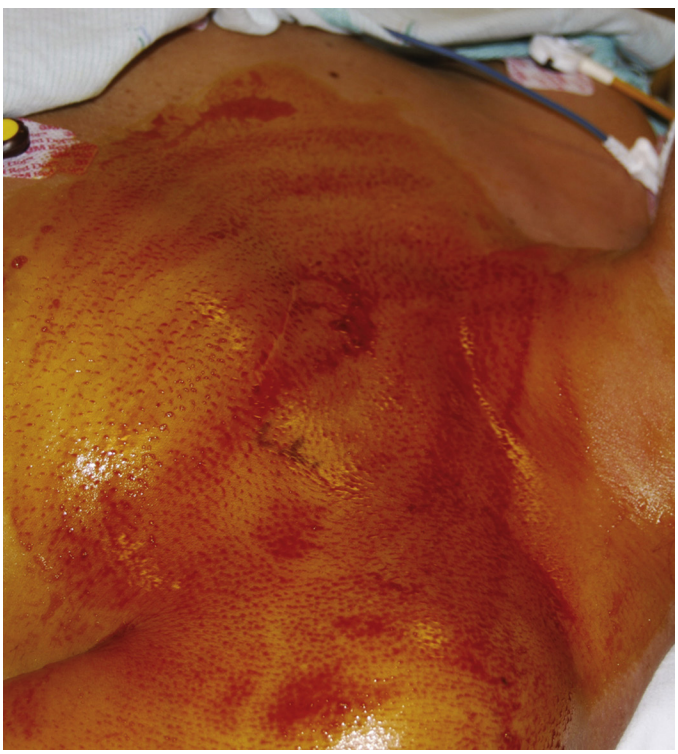
After the surgical table is prepared and a second surgical prep solution has been applied and given time to completely dry, the periphery of the surgical field is isolated with cloth towels or disposable paper surgical towels (Fig. 6.20). A fenestrated drape is applied with care to position the fenestration over the operative site (Fig. 6.21). The drape should be large enough to cover the patient, the operating table, and the foot of the bed and extend over the anesthesia screen at the head of the bed. The drape covering the patient's head must provide adequate ventilation for the patient and allow those managing the head of the bed to have direct visualization of the patient's face. This allows for patient comfort and reassurance, as well as monitoring and airway management, without disturbing the surgical field or operator's movements (Fig. 6.22). Once the drape is in place, it should not be repositioned as this may compromise the sterile field (Fig. 6.23).

After the patient draping is complete, the fluoroscopic imaging tower should be covered with a sterile cover intended specifically for that use. The surgical lights should be positioned to provide optimal lighting without equipment or

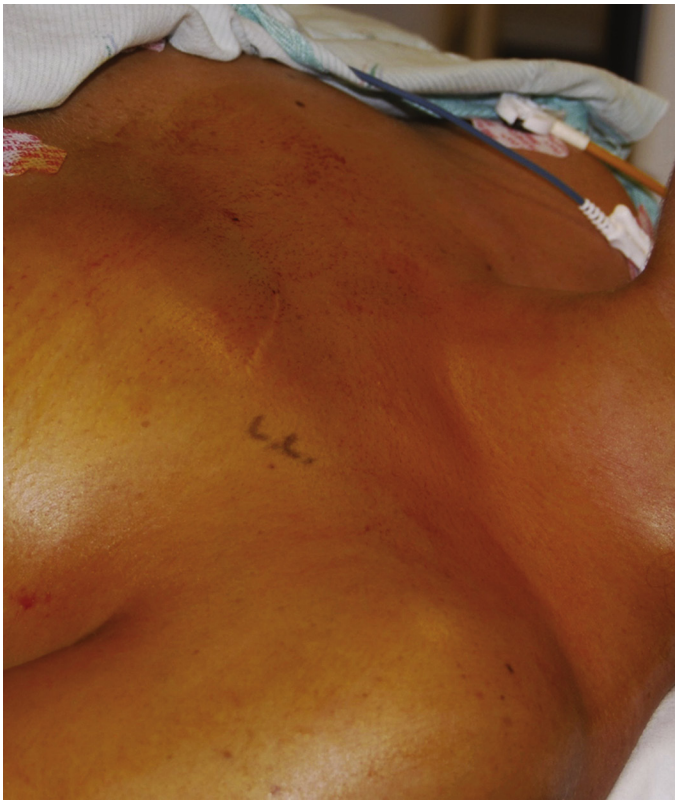
• **Fig. 6.15** Sterile gloving. (A) With assistant. (B–C) Closed glove technique.



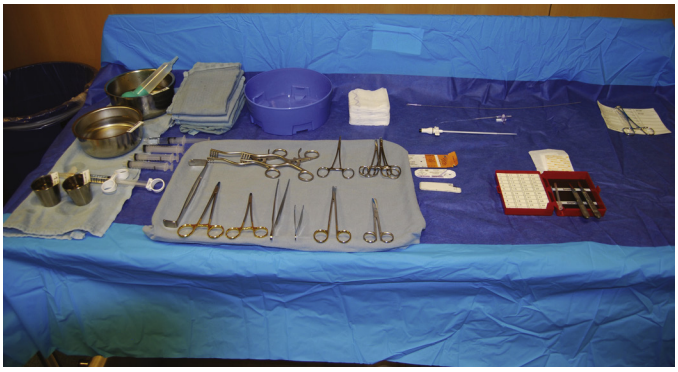
• **Fig. 6.16** (A–B) Parts of surgical gown not considered sterile.



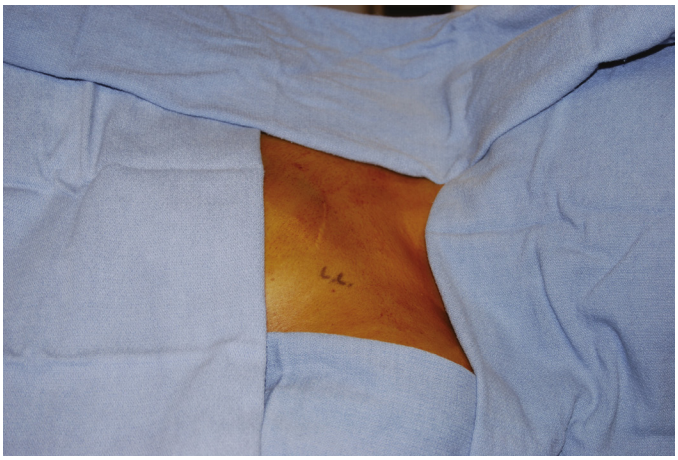
• **Fig. 6.17** Patient skin preparation (wet).



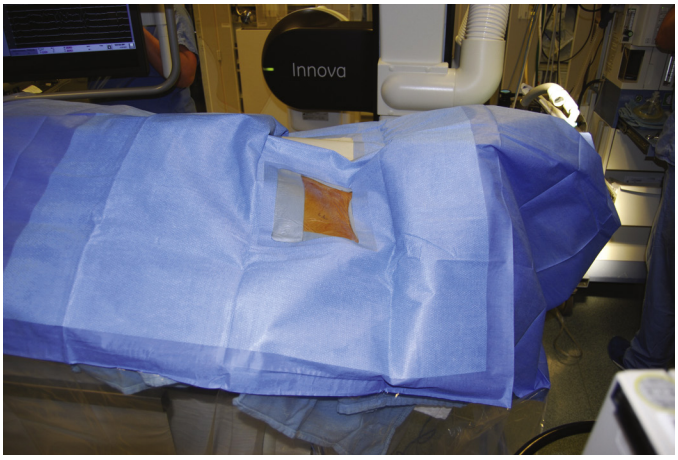
• **Fig. 6.18** Patient skin preparation (solution dry, ready for draping).



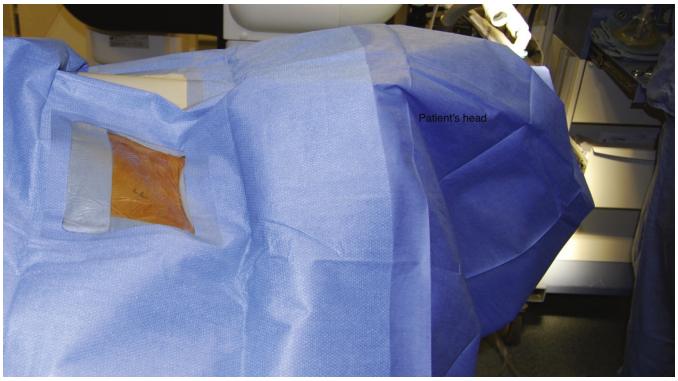
• **Fig. 6.19** Surgical table with instruments arranged.



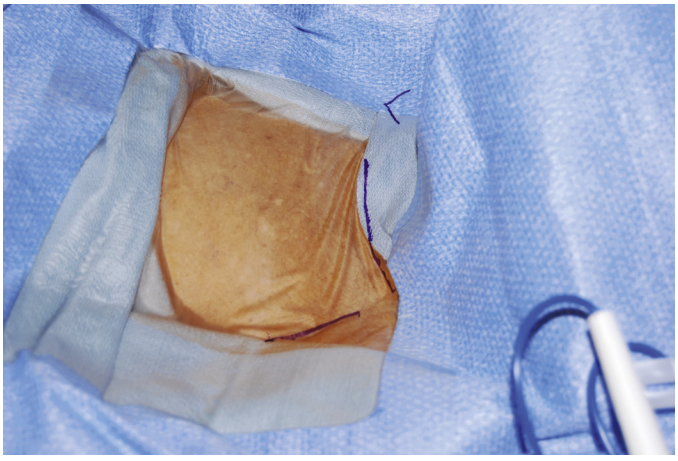
• **Fig. 6.20** Sterile towels applied following surgical prep.



• **Fig. 6.21** Fenestrated drape placement following sterile towels.

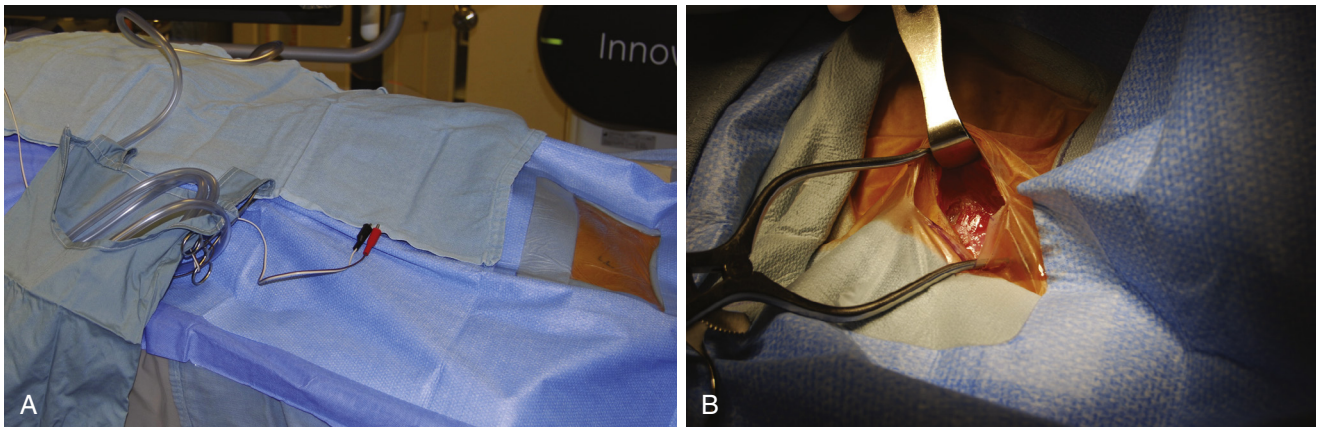


• **Fig. 6.22** Patient's head positioned under sterile drape.



• **Fig. 6.23** Example of drape repositioning with compromise of surgical field.

personnel casting shadows onto the surgical field or being at risk of contamination by the head of the implanting physician. The remaining equipment (suction, pacing cables, electrocautery pen) is attached to the drape using nonpenetrating towel clamps (Fig. 6.24A–B). At this point, the operating physician should perform a final check of all draping, monitoring lines, and equipment placement before commencing with the procedure.



• **Fig. 6.24** (A) Surgical draping completed. (B) Example of drape that has been moved enough to question sterility of field.

Summary

Optimal patient preparation is the responsibility of the entire electrophysiology procedural team. The procedural staff must be trained in the fundamentals of proper patient preparation to ensure a safe and comfortable procedure. The entire team must ascribe to a culture of safety, which includes patient identification upon entering the procedure room and again prior to the start of the procedure. Correct placement of monitoring lines, ECG electrodes, and defibrillation patches and checking for all necessary airway management equipment permit rapid assessment of acute changes during the procedure. Proper patient preparation, positioning, and draping minimize patient anxiety and maximize procedural efficiency and safety. Understanding of and adherence to the principles discussed in this chapter are vital components of a successful surgical procedure.

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7 Cardiac Implantable Electronic Device Surgical Implant

JEANNE E. POOLE, LYLE W. LARSON, BRIAN OLSHANSKY

Introduction

Cardiac implantable electronic devices (CIEDs) were initially placed by surgeons via thoracotomy. The implant was a major surgical endeavor. Advancement in lead and device technology, simplification of the implant technique, and refinement of and emerging complexity in device capabilities have led to transvenous lead and subcutaneous chest wall device implants by electrophysiologists and other implanting cardiologists. CIED implantation is now an integral part of cardiac electrophysiology practice and is a major component of electrophysiology fellowship training programs worldwide, necessitating a systematic approach to the surgical procedure to achieve successful and safe results.

The implanting cardiologist must understand the basics of the chest anatomy and realize it is a dynamic structure. During CIED implantation, the patient is lying in one position, but the device and the leads must function even with position changes and must accommodate considerable motion owing to expected normal physical activity, including deep breathing, arm extension, and arm lifting (Fig. 7.1). A generator placed too high will impinge upon the clavicle, causing discomfort. Similarly, normal adductor and abductor motion of the arm can be limited if the generator is placed too laterally (Fig. 7.2).

A lead placed too medially via a subclavian venipuncture may pass through the subclavius muscle, entrapping it between the clavicle and the first rib, which when exposed to the normal action of the clavicle can lead to lead fracture. On the other hand, a lead placed too laterally by a cephalic or axillary venous approach may be at greater risk for retraction out of the heart with arm extension or standing. An experienced implanter understands the proper placement of the device and leads, providing enough lead redundancy and positioning the device to avert a disastrous long-term outcome.

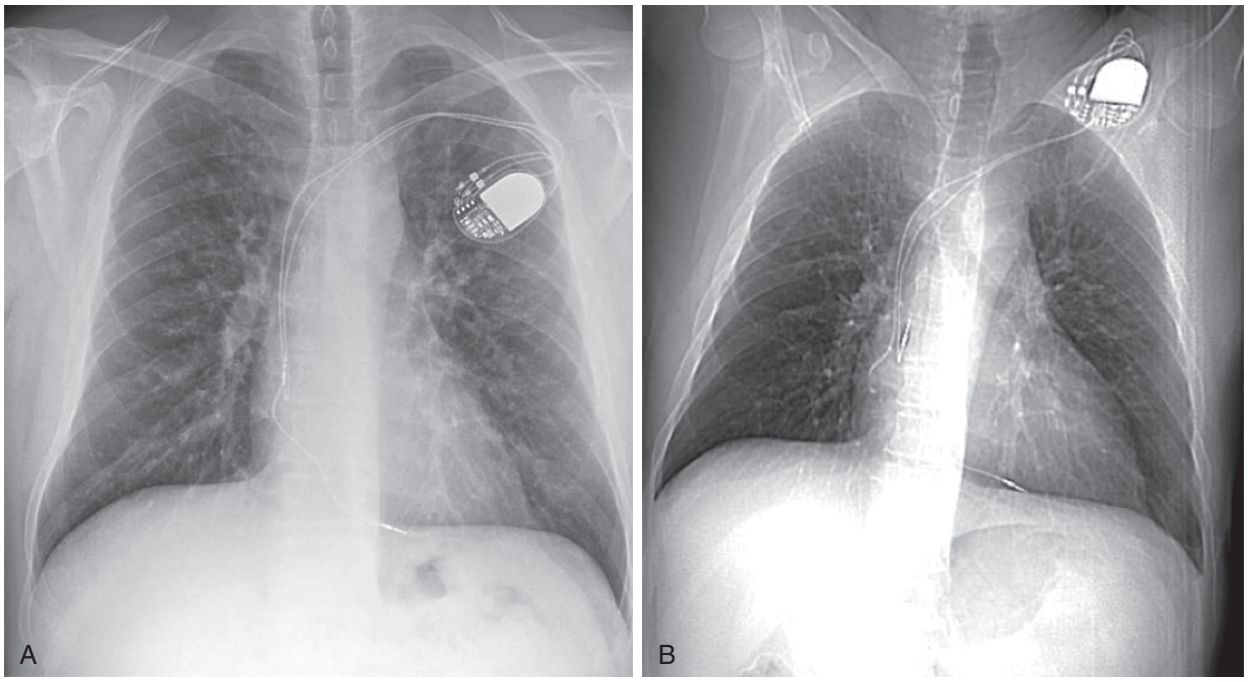
Ironically, however, and perhaps more important to many patients, is the look of the "finished product." The patient and family may not appreciate the complexity of the implant procedure itself. The patient sees the wound and will observe how it heals; he or she sees the visual appearance of the pocket and incision. Good surgical technique therefore includes careful attention to the implant and the wound closure to avoid complications and provide the best cosmetic appearance possible. Thus, no single step in the implant can be disregarded.

Careful attention to the steps detailed in this chapter can lead to a successful CIED implant. Following these steps may appear easy, but an experienced implanter knows that every step must be performed with care since even the smallest mistake can result in a suboptimal procedure. The implanter and trainee who adhere to good surgical principles and learn to anticipate the consequences of unexpected obstacles and potential complications are much more likely to have a good outcome. With practice, the implanter can gain experience and perform successful implants in a safe, effective, and efficient manner without excessive fluoroscopy. To achieve this, the implanting physician must adopt a systematic approach to every implant and perform enough procedures regularly to maintain competency. As such, requirements are set forth for the numbers of implants for trainees in clinical cardiac electrophysiology program fellowship training programs to achieve competency.[1]

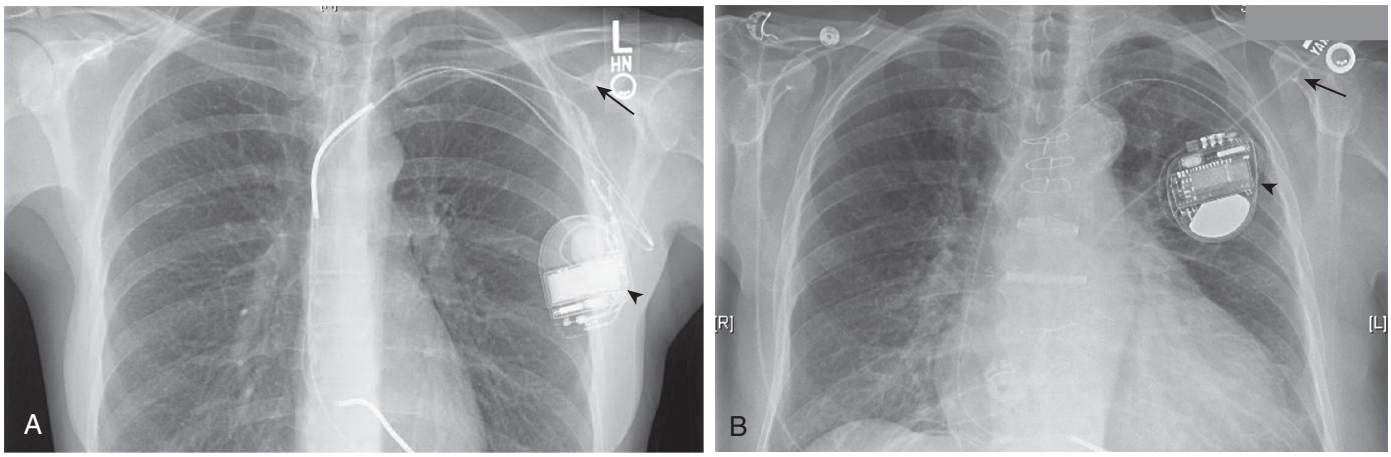
Preprocedure Planning for a New CIED Implant

Once it is determined that a patient is a CIED candidate, patient-specific and device-specific factors need to be considered. Patient-specific factors include the general health of the patient, comorbidities, and proper assessment of preoperative laboratory values and anatomic issues. Important points of the chest exam include the amount of subcutaneous tissue in the upper chest, identification of any skin rashes or lesions in the area of the surgical procedure, bony abnormalities such as prior clavicular fractures, prior thoracic surgery, a barrel chest, forward fixation of the shoulder (owing to either prior deformity or injury), or abnormalities of the cervical-thoracic curvature (Fig. 7.3).

The morbidly obese patient may present difficulty with access and challenges in device fixation, whereas the petite elderly patient may be at greater risk of pneumothorax, lead perforation, or pocket dehiscence. If there are prior leads placed, there may be obstruction of the subclavian vein. It is important to know whether there are suspected anatomic issues



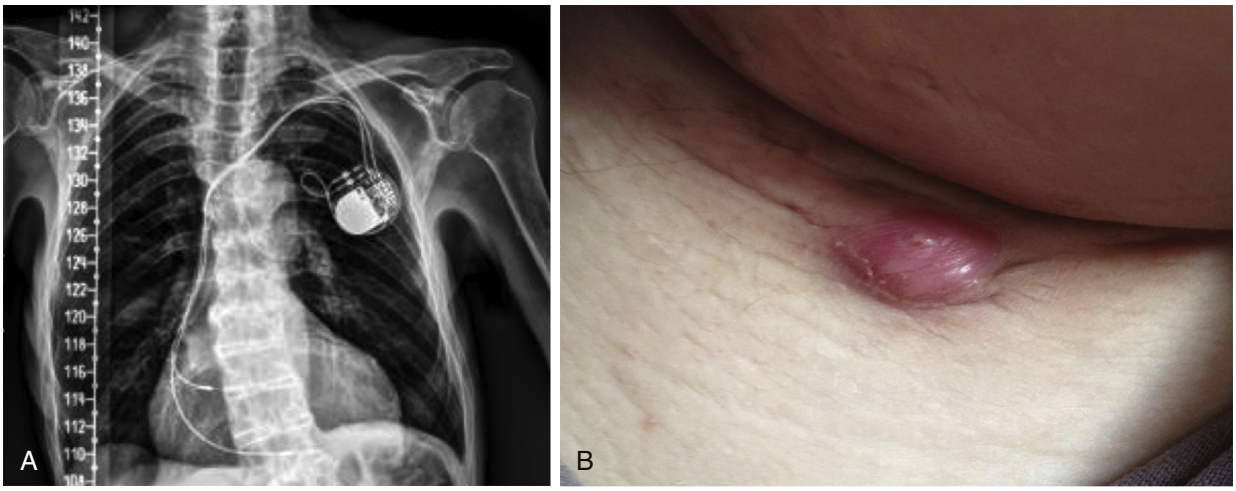
• **Fig. 7.1** (A) Chest radiograph of a patient with a dual-chamber pacemaker showing the position of the pacemaker with the arms hanging down along the patient's side. (B) The position of the pacemaker with arms raised above the head. Note how the pacemaker generator has moved superiorly with associated movement of subcutaneous tissue.



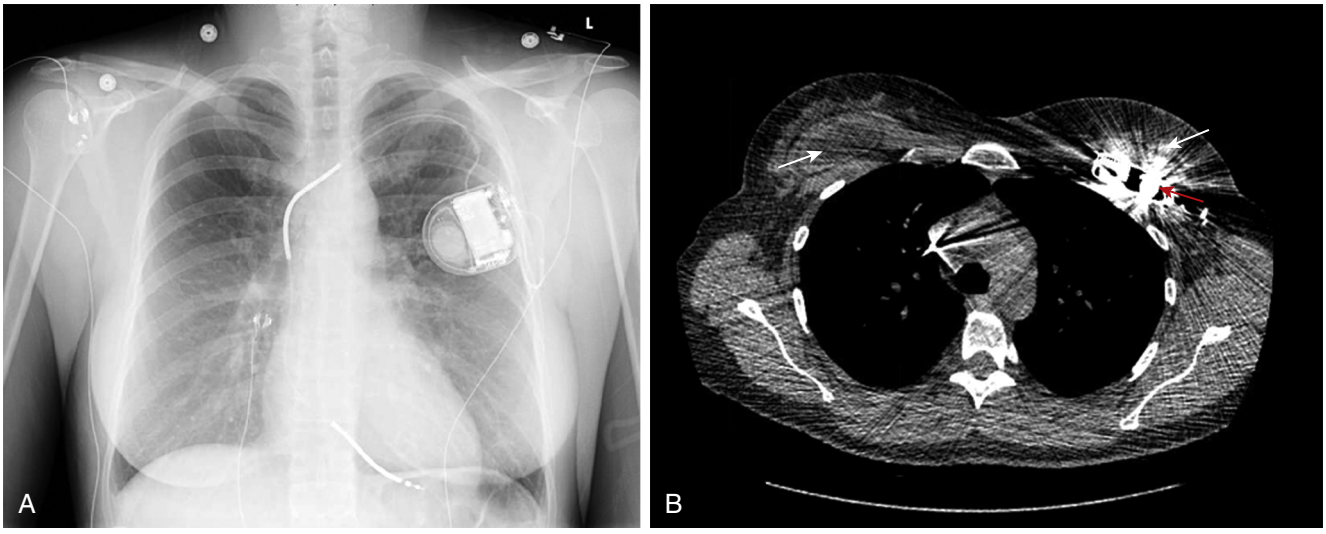
• **Fig. 7.2** (A) A chest radiograph demonstrating a dual-chamber implantable cardioverter-defibrillator (ICD) generator that was implanted laterally with resultant impingement on the deltoid muscle causing pain with upper extremity motion. Noted are the coracoid process (*arrow*) and the lateral margin of the ICD generator (*arrowhead*), which extends lateral to the coracoid process. (B) An example of ideal placement of the generator. Noted again are the coracoid process (*arrow*) and the lateral margin of the generator (*arrowhead*), which is placed medial to the coracoid process.

including specifics related to the heart (left-sided superior vena cava [SVC] or congenital heart abnormalities). Preparation for a successful implant includes knowledge of specific patient characteristics and conditions (severe tricuspid regurgitation or significant pulmonary hypertension) or abnormalities of the right atrium or right ventricle that may impair lead placement (Chiari network, previous cardiac surgery, atrial baffles, Ebstein anomaly). For those receiving a cardiac resynchronization therapy (CRT) implant, the coronary sinus may also be difficult to access for those who have had prior cardiac surgery. Similarly, if there is a persistent left SVC, a CRT implant may pose a major challenge.

In women, it is important to identify whether augmentation mammoplasty (breast implants) are present and, if so, to identify the margins and the implant tissue space (submuscular or submammary) (Fig. 7.4). If a woman has had breast cancer, the physician should document whether radiation therapy was administered, as well as prior surgical interventions including the extent of any surgical reconstruction. Radiation therapy may increase the risk of bleeding during vascular access owing



• **Fig. 7.3** Examples of preoperative findings that can affect implantation. (A) An overpenetrated chest radiograph of a patient with significant scoliosis. The positioning of the patient on the table could be altered, and the anatomic landmarks distorted, potentially increasing the risk of vascular access. (B) It is important to examine the skin for any evidence of breakdown or possible infection. In this patient, an infected axillary sebaceous cyst required treatment before the planned procedure.

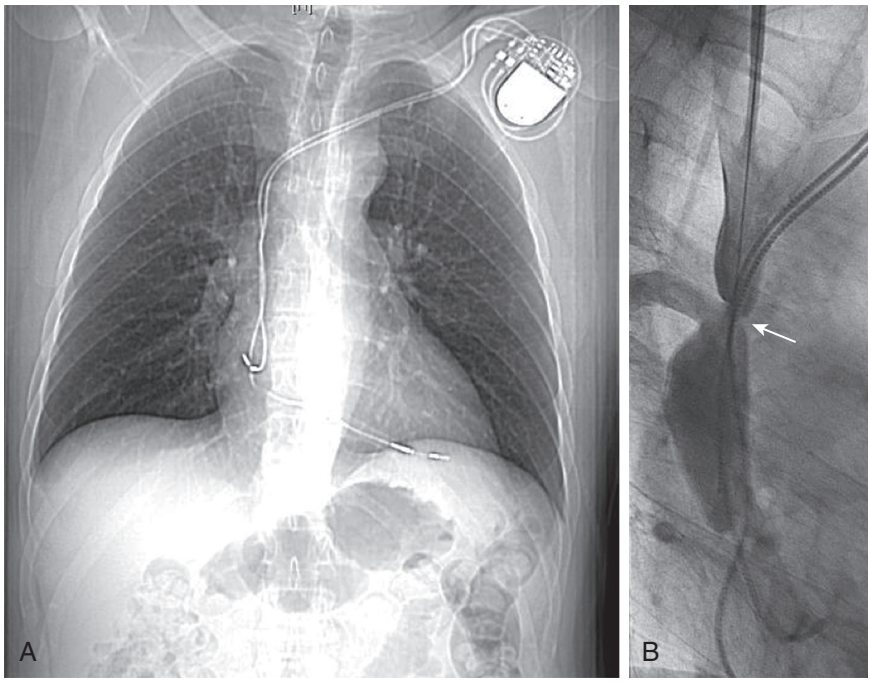


• **Fig. 7.4** Patient with breast implants. (A) It is often difficult to identify the presence and borders of breast implants on a routine chest radiograph. (B) Chest computed tomography scan illustrating the breast implants (*white arrows*) and an implantable cardioverter-defibrillator (ICD), which in this case has also been implanted in the left submuscular space (*red arrow*).

to changes in the integrity of the vessel wall. Additionally, the vascular supply to the tissue may also be compromised in such patients with or without the presence of skin grafting. Alternative implant sites may need to be considered in such situations as attempting to form a pocket in areas of prior radiation therapy, or skin grafting may disrupt the tenuous vascular supply, resulting in poor healing, infection, or tissue necrosis.

Other considerations include the patient's handedness (best to implant on the contralateral side), preexisting upper extremity limitation of motion (prior cerebrovascular accident), dialysis fistulas, or orthopedic issues (e.g., fractures, rotator cuff injuries). Further, unique activities such as hunting, golfing, or playing the violin should be identified to choose on which side the device will be implanted. Importantly, it is best to avoid reimplantation on the ipsilateral side of a prior device infection under all circumstances except the rare occasion of having no other option and after appropriate antibiotics have been administered, the site has healed, and preferably a new tissue plane is used (submuscular if the prior device was subcutaneous).

Comorbid health conditions, such as obstructive sleep apnea, use of a continuous positive airway pressure mask, general cardiovascular health, and the need for a diabetes management plan, must be considered. The patient's ability to lie supine must be assessed; it may be affected by shortness of breath due to heart failure or disabling back pain, among other causes. Determining the type and route of anesthesia is part of the preoperative assessment (see Chapter 4). It is best to avoid an implant when there is any recent preexisting and incompletely treated infection or suggestion of possible infection. Part of the routine preoperative assessment is a white blood cell



• **Fig. 7.5** (A) Chest radiograph of a patient with a dual-chamber pacemaker who developed symptomatic superior vena cava stenosis. (B) Contrast venography demonstrating stenosis of the superior vena cava (*arrow*). A catheter can be seen (*arrow*) advanced from the right internal jugular vein to be used in balloon angioplasty of the superior vena cava stenosis.

count and a urinalysis (the latter is generally needed only for inpatients, those living in assisted living facilities, or those with chronic urinary tract infections). Identification of all allergies is critical to avoid significant and even life-threatening complications. Allergies to betadine or chlorhexidine can dictate which skin preparation solution to use.

Dialysis patients pose a particular challenge. Venous access and the available site of implant may be restricted by the presence of an arteriovenous (AV) fistula or a hemodialysis port. Implantation on the ipsilateral side of an AV fistula may result in substantial bleeding during vascular access and following sheath removal owing to high venous pressures and subjects the leads to bacterial contamination during dialysis. Similarly, if a dialysis port is present, inadvertent damage to the port may occur when forming the pocket for the CIED. A general principle is to use the contralateral side to an actively used dialysis port.

To aid in vascular access, the patient should receive preprocedure hydration. This can be given once the patient has been checked into the preprocedure area. For patients without heart failure, an intravenous bolus can be given followed by maintenance intravenous (IV) fluids. Repeat fluid boluses can be given to the patient once they are in the procedure room if needed and if access is difficult. For patients with heart failure, an assessment of volume status is required with IV fluids adjusted accordingly. Lifting the legs on a wedge during attempted venous access can be helpful.

Device implant planning includes deciding the side of implantation, location of incision, and choice of vascular access. While venography is performed routinely by some implanters, others will use venography only for specific situations such as difficult venous access or in the setting of prior lead implant procedures.

Patients with existing leads are at higher risk for venous occlusion, and an assessment of vessel patency prior to the procedure is recommended. Upper extremity ultrasound can be performed before the date of the planned procedure, although in patients with well-formed collaterals, the presence of vascular flow by ultrasound cannot exclude a vessel occlusion or stenosis. Alternatively, venography can be performed either before the planned date of the procedure or just prior to prepping and draping the patient in the event that an alternative approach to the procedure must be considered, such as venoplasty or use of the contralateral side. While the risk of venous occlusion may increase with the number of leads, patients can form venous occlusions even with only one or two leads, possibly owing to a procoagulant state (Fig. 7.5). Potential clues to venous occlusion are the presence of chest wall collateral vessels.

Patients at increased risk of bleeding include those on continued anticoagulation or hemodialysis and those with poor tissue turgor (elderly, chronic steroid use, prior radiation). Many implanters routinely implant CIEDs in patients taking warfarin with international normalized ratio (INR) values in the therapeutic range.[3,4] Little data exist to support a consistent approach to patients taking the factor Xa inhibitors or direct thrombin inhibitors.[5,6] Currently, many implanting physicians ask the patients to hold their dose the day before and/or the day of the procedure. It may not be possible to discontinue antiplatelet drugs early enough to mitigate their effects, particularly in patients in whom antiplatelet drugs are necessary, such as

following coronary stent placement. As such, it should be anticipated that these patients are at substantial risk for bleeding and subsequent pocket hematoma. Others at risk for bleeding include patients with renal failure and those with thrombocytopenia. In addition to careful intraprocedural attention to good hemostasis, some physicians use local thrombotic substances such as D-stat, Gelfoam, or Surgiseal (see Chapter 3). However, good hemostasis obtained through meticulous attention to the surgical technique is always preferable.

Temporary transvenous pacemakers may be present in patients undergoing pacemaker implantation in the setting of new complete or high-grade atrioventricular block. If present, the site of placement and position of the temporary pacing lead should be carefully assessed beforehand for risk of dislodgment during the implant procedure.

Occasionally, however, a patient with complete heart block may appear to have a stable escape rhythm and a temporary pacemaker is not deemed necessary. This can be deceptive because such a patient may quickly lose their escape rhythm owing to suppressive effects of the anesthesia or manipulation of the lead within the heart. The implanting physician should therefore consider the risk versus the benefit of placing a temporary pacemaker solely for the implant procedure.

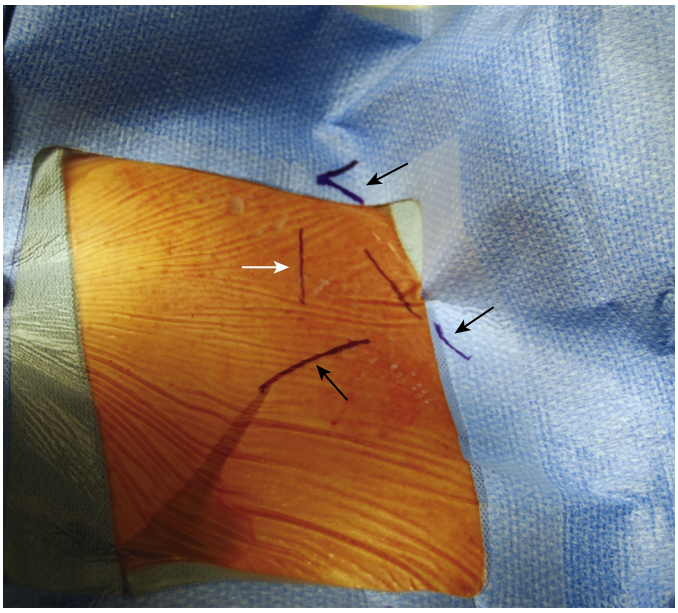
A temporary pacemaker could also be considered during a generator replacement in a patient who is apparently pacemaker dependent. The patient should be assessed prior to the procedure for documentation of an underlying escape rhythm. Slowly lowering the pacemaker rate to 30 beats/min may allow for emergence of a hemodynamically stable escape rhythm. If significant dissection of the pocket tissue is anticipated and particularly if the leads are old, there is an increased risk of damage to the leads during the new device implant that could result in lead malfunction. Many clinicians do not place a temporary pacemaker but are ready to exchange the lead to the new device carefully but quickly. A short period of asystole is not likely to be dangerous to most patients.

Patient Position, Prepping, and Draping

The procedure does not begin with the incision, but rather when the patient is brought into the procedure room. The implanting physician should review all significant aspects of the planned procedure and the patient positioning and preparation. These are covered in detail in Chapter 6.

It is important to verify that the patient is supine on the procedure table and the head is not elevated. Rotation and/or elevation of the patient on a wedge may result in misleading fluoroscopic images. The physician should note if the patient has scoliosis or kyphosis. For a transvenous lead implant, the arms should be tucked securely in adduction. For a subcutaneous (nontransvenous lead) device, the left arm is abducted to an angle less than 90 degrees and should be secured on an arm board (see Chapter 10). A modification of arm placement may be required to have better access to the lateral chest wall to correct a prior significant lateral migration of the pulse generator.

Appropriate application and location of electrocardiogram (ECG) monitoring electrodes and defibrillation patches should be confirmed by the physician. For patients undergoing implantable cardioverter-defibrillator (ICD) implantation in whom induction of ventricular fibrillation for defibrillation threshold testing is planned, some physicians have used two sets of defibrillation patches with opposite vectors. Although failure of external defibrillation is rare, it is better to have a backup defibrillation system than to be without immediate resuscitative equipment when needed.



• **Fig. 7.6** Pertinent landmarks on an iodine-impregnated occlusive dressing are demonstrated. The boundaries of the surgical field including the suprasternal notch, clavicle, and deltopectoral groove are shown (*black arrows*), as is the proposed transverse incision (*white arrow*).

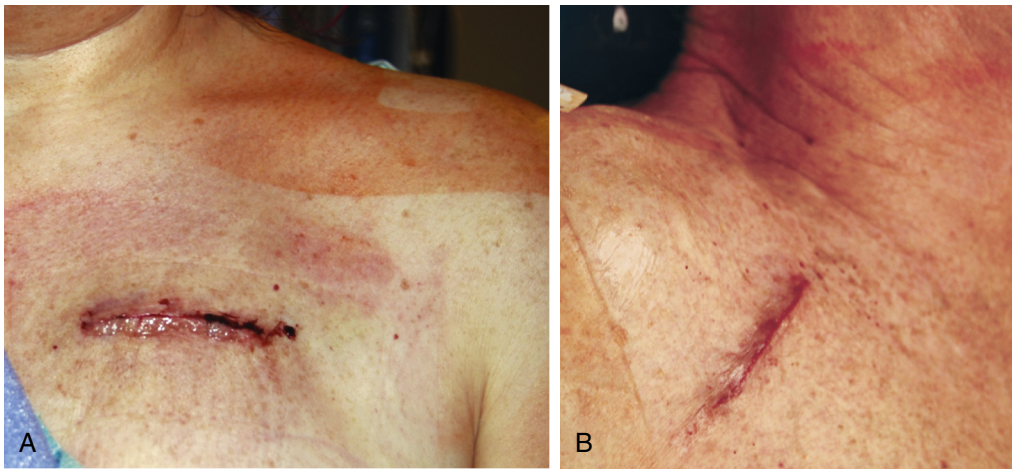
Intravenous antibiotics (cefazolin preferably or if necessary vancomycin) should be given within 30 minutes of making the incision (and documented). The sterile skin preparation and the draping of the patient should be carried out in a way that provides a generous surgical field and meticulous skin sterility (see Chapter 6). Once the patient has been prepped and draped, the anatomic landmarks (suprasternal notch, clavicle, deltopectoral groove) should be marked directly on the iodine-impregnated occlusive drape with an indelible sterile ink pen. This is helpful in planning the location of the incision and formation of the pocket (Fig. 7.6).

Performing the Procedure

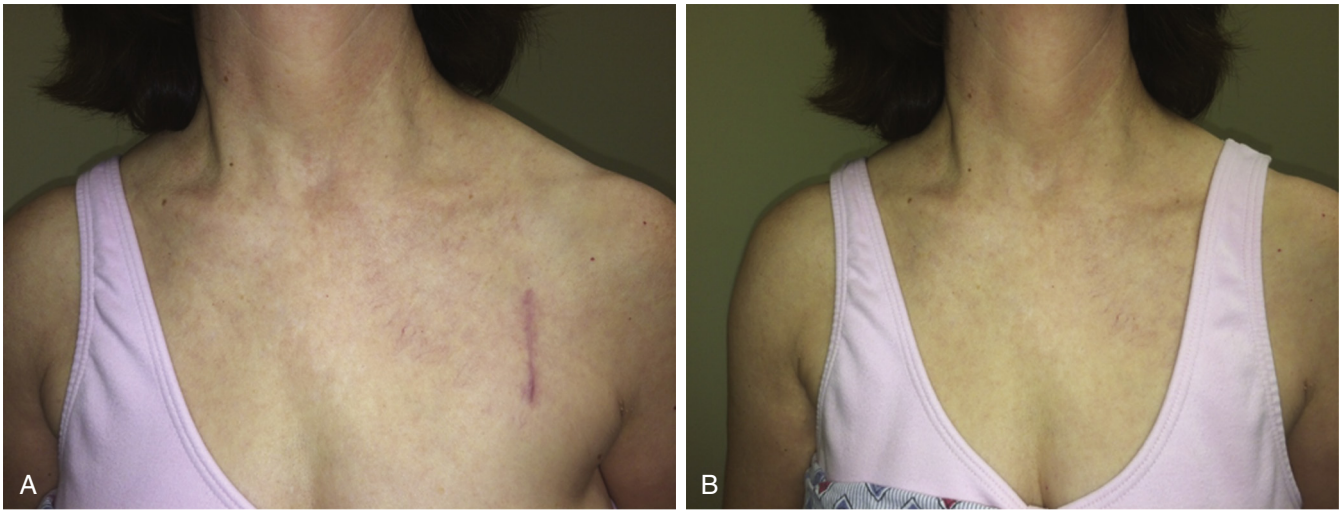
Incision and Pocket Choices

Prior to making the incision, it is important to ensure that the patient is comfortably positioned and adequately sedated with a properly maintained airway sufficient to prevent unwanted movement during the procedure. Adequate local analgesia (lidocaine, bupivacaine) should be given subcutaneously in the prospective location of the incision and the pocket.

For the typical pocket made in the right or left upper chest, either a transverse or deltopectoral groove incision is generally



• **Fig. 7.7** (A) A patient following a recent left-sided implantable cardioverter-defibrillator (ICD) implant via a transverse incision. (B) A patient following a right-sided implant via a deltopectoral groove incision.



• **Fig. 7.8** (A) A vertical incision was made to implant an implantable cardioverter-defibrillator to provide better concealment behind clothing. (B) The cosmetic appeal of this incision is demonstrated.

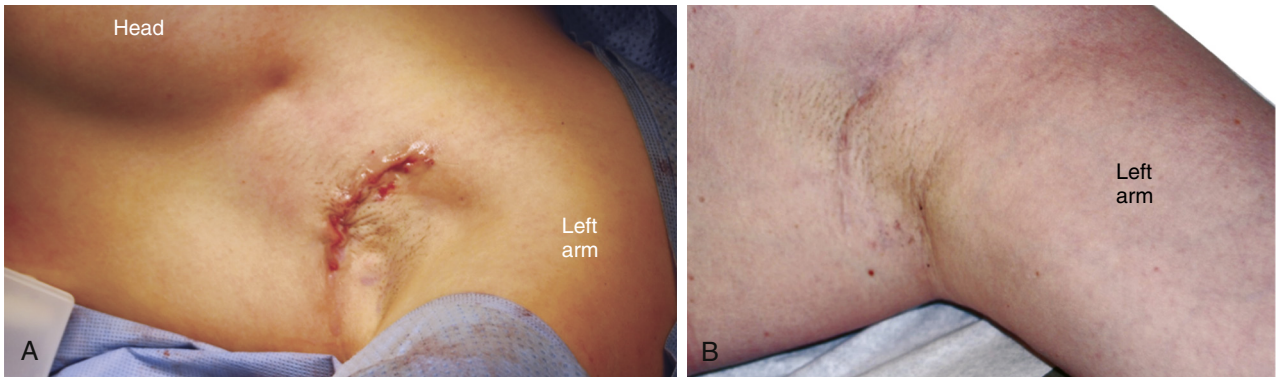
chosen. Attempts to approximate Langer lines have sometimes led to angled incisions; this approach, however, likely does not improve wound healing.[2] Generally, a transverse incision or deltopectoral groove incision provides the best aesthetic appearance for the patient while providing adequate exposure to vessel choice (Fig. 7.7).

Some implanting physicians have used a vertical incision, termed a "bra strap" incision, for cosmetic reasons. While the intent of this approach is for better concealment behind clothing, this incision can present challenges in pocket formation, particularly if placed in a subpectoral position (Fig. 7.8).

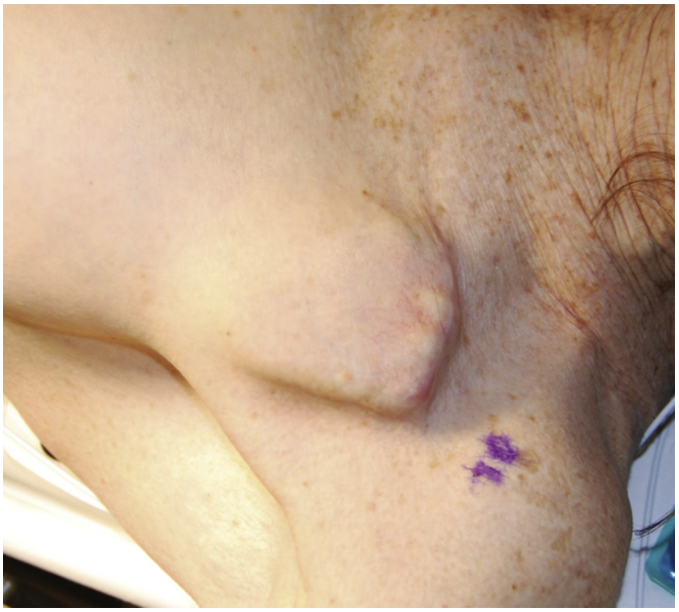
A submammary pocket may be performed through an incision under the breast in the inframammary crease, or a subpectoral pocket may be performed through an incision high in the midaxillary line (Fig. 7.9). These approaches are often chosen for improved cosmetic appearance, to avoid any incisions on the upper chest wall, or in patients requiring a greater degree of protection from erosion of the pulse generator when little subcutaneous tissue is present.

The subcutaneous ICD, as described in Chapter 10, is placed in the left midaxillary line. Two incision choices for the generator pocket have been used by implanting physicians: either curvilinear beneath the left breast toward the lateral chest, or a transverse incision a few centimeters anterior to the level of the planned pulse generator pocket.

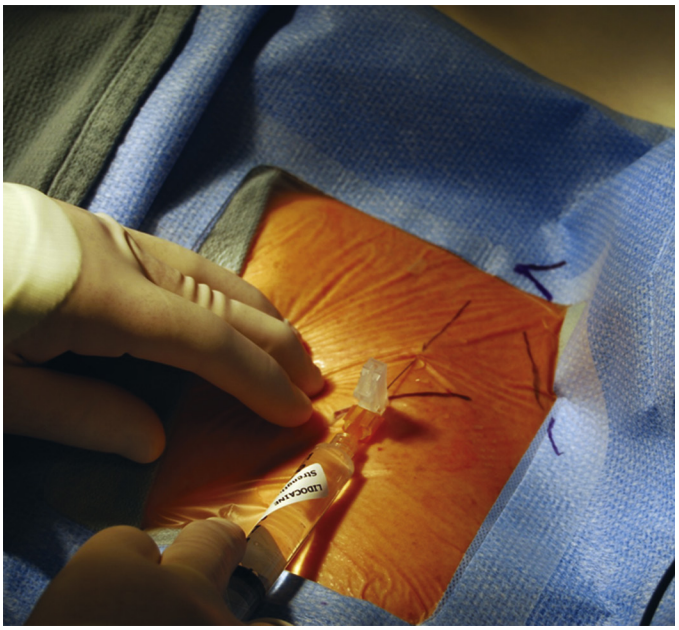
Most patients undergoing transvenous CIED placement require only a subcutaneous pocket. Patients with poor subcutaneous tissue may benefit by having the device placed beneath the pectoralis muscle to provide better protection against erosion or from pain caused by the device abutting against the deltoid muscle or the clavicle (Fig. 7.10). A subpectoral pocket can also improve the cosmetic appearance of the implanted CIED (a priority for some patients, as mentioned earlier); however, the implant is typically more uncomfortable in the immediate postoperative period. This type of implant is also more technically challenging to perform and can be even more challenging at the time of generator replacement.



• **Fig. 7.9** (A) The position of an incision made through the axilla for an implantable cardioverter-defibrillator implant that was placed submuscularly. (B) The appearance of a healed axillary incision in another patient. This approach is used both for cosmetic advantage and for patients who have little subcutaneous tissue.



• **Fig. 7.10** An implantable cardioverter-defibrillator generator with the superior-lateral border overlying the anterior deltoid muscle, restricting upper extremity arm movement. This generator was moved to a subpectoral position.



• **Fig. 7.11** Injection of lidocaine into the subcuticular tissue for a cardiac implantable electronic device implant.

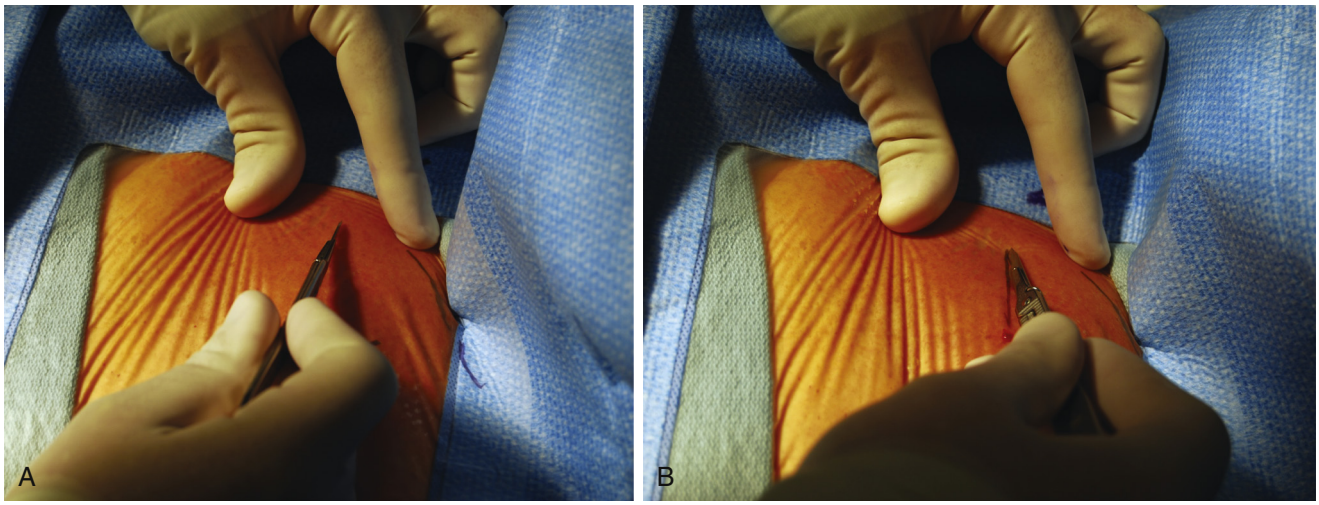
A subpectoral pocket can be made with relative ease if an understanding of the anatomy is appreciated. The risk of a subpectoral approach includes the potential for damage to the acromiothoracic neurovascular bundle, resulting in compromised arterial blood supply to the pectoralis major or damage to neural innervation.

Forming the Incision and Pocket

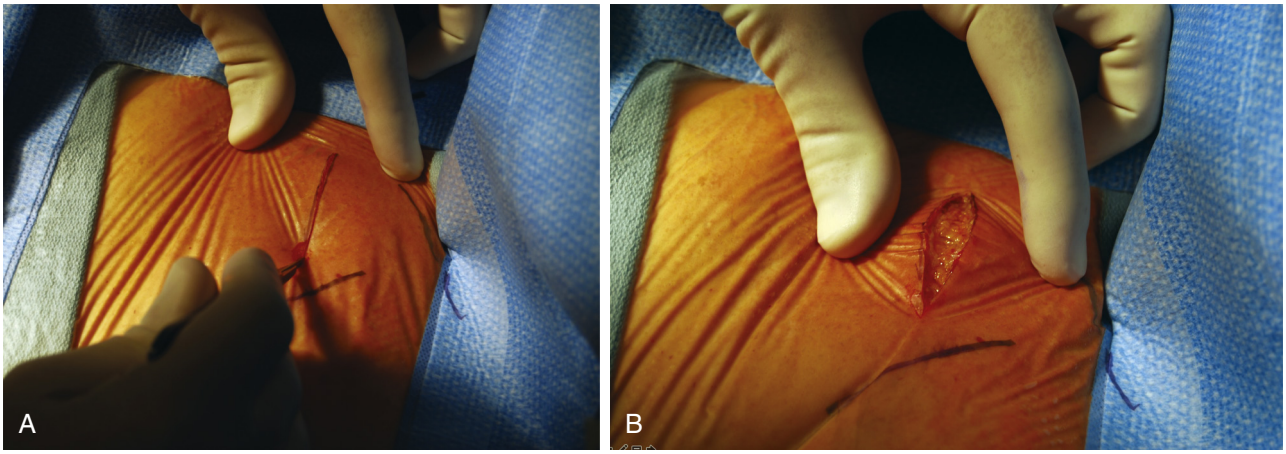
Once the patient is prepped/draped, the preprocedure timeout has been performed, and systemic anesthetic provided, local anesthetic is administered. Achieving adequate local anesthesia is very important. Using a small-gauge needle, 2% lidocaine should be infiltrated subcutaneously along the line of the intended incision and surrounding tissue. A few minutes should be allowed for the lidocaine to have an effect before the incision is made. Patients under conscious sedation should not have to endure pain from lack of adequate anesthesia. Local anesthetic will be supplemented by the use of intravenous anxiolytics and pain medication (see Chapter 4) (Fig. 7.11).

The incision is made using a No. 15 or No. 10 blade. The blade should be held such that the incision is made perpendicular to the patient's chest, not perpendicular to the floor (Fig. 7.12). Careful attention to this detail helps to avoid beveling of the incision, which promotes better wound closure and healing. The downward pressure used while making the incision should be enough to cause a full-thickness incision through the epidermis and dermis. Using three points of tension (thumb, index finger, and knife blade), the incision is made with a single steady stroke (Fig. 7.13).

Multiple strokes across the incision to achieve the correct depth should be avoided; this creates uneven vertical planes and makes closure more difficult (Fig. 7.14) (see Chapter 3). The skin incision length should be adequate to form a pocket that



• **Fig. 7.12** (A) Proper position of the knife blade held perpendicular to the curvature of the patient's chest. (B) Improper position of the knife blade held perpendicular to the floor and not to the patient's chest. If performed improperly, a beveled incision will result, potentially impairing wound healing.



• **Fig. 7.13** (A) Three points of tension to prevent distortion of the skin during the incision. (B) Result of a single, fluid stoke of the knife blade through the epidermis and dermis.

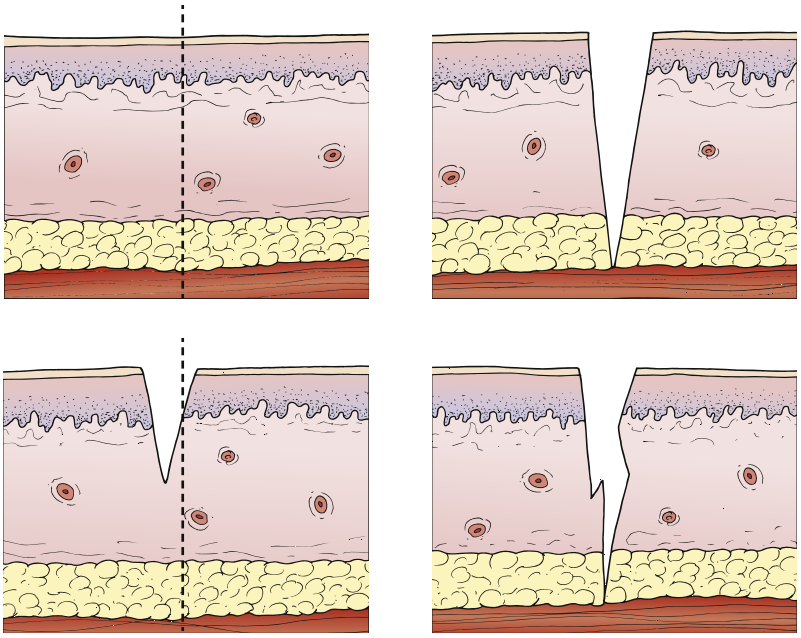
will accommodate the specific device chosen but not significantly larger. In younger patients, the tissue is more elastic and offers the opportunity for an incision that is shorter than the width of the pulse generator, improving cosmetic appearance.

Once the incision is complete, one or two self-retaining retractors (Weitlaner) may be inserted to improve exposure (Fig. 7.15). Dissection is carried downward with electrocautery and sharp and/or blunt dissection to the pectoralis major fascia. Patients with greater subcutaneous adiposity may have more small vessels, which can bleed during this portion of the procedure, and can generally be directly cauterized. Care must be taken to avoid contact between the electrocautery tip and the patient's skin or the Weitlaner retractor; this could result in thermal damage to the skin, which may burn the skin and inhibit healing (Fig. 7.16). Occasionally, larger vessels may be encountered. These may be addressed using a forceps or hemostat to grasp the bleeding vessel. The electrocautery tip is held against the instrument to transfer energy to the vessel, producing hemostasis (Fig. 7.17). Rarely, a vessel is large enough that it should be ligated rather than cauterized (Fig. 7.18).

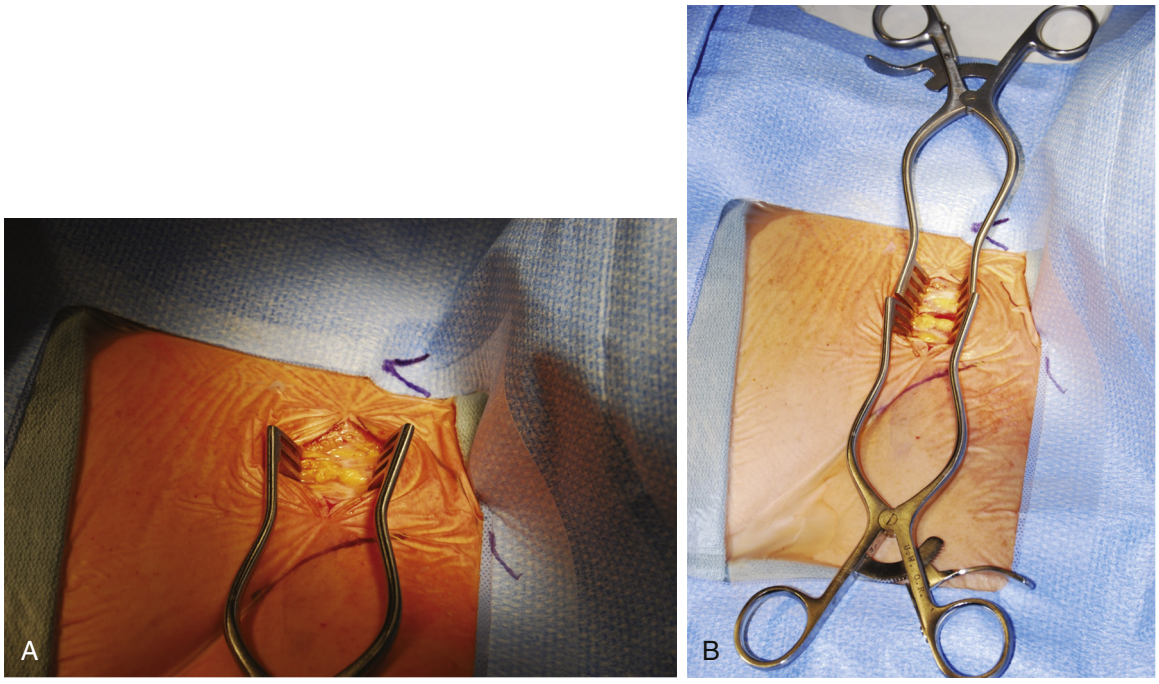
Superficial neural fibers may be encountered during the dissection, resulting in significant pain when using electrocautery. Further infiltration of lidocaine should be administered whenever the patient is uncomfortable, up to a maximum dose of 4.5 mg/kg. Lidocaine is a vasodilator and can increase the risk of bleeding. Generally, in patients receiving CIEDs, lidocaine without epinephrine (a local vasoconstrictor) has been used to avoid any risk or proarrhythmia, although this risk is likely quite small with judicious use of the local anesthetic.

Many clinicians use a blunt dissection technique without electrocautery; however, the use of electrocautery or Plasma-Blade is preferred to decrease the bleeding risks and thus is a more efficient tool. It cannot be emphasized enough that having sufficient hemostasis within the pocket at the end of the procedure is critical to reduce hematoma formation and infection risk.

The subcutaneous pocket is most often formed anterior to the pectoralis fascia. The fascia is easily identified as a white fibrous covering on top of the muscle (Fig. 7.19). At this point, additional lidocaine should be infiltrated into the tissues, taking note to infiltrate beneath the prepectoralis fascia to provide



• **Fig. 7.14** Incision comparing a single stroke versus improper multiple strokes. (From Romfh RF, Cramer RS. *Technique and Use of Surgical Tools,* ed 2. New York: Appleton & Lange; 1992.)

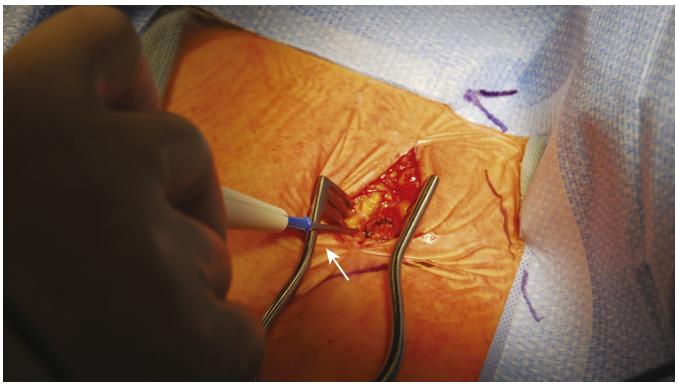


• **Fig. 7.15** (A) Single Weitlaner retractor applied in the center of the incision. (B) Two Weitlaner retractors applied to the incision, which may be beneficial to improve exposure, such as in patients with significant adiposity.

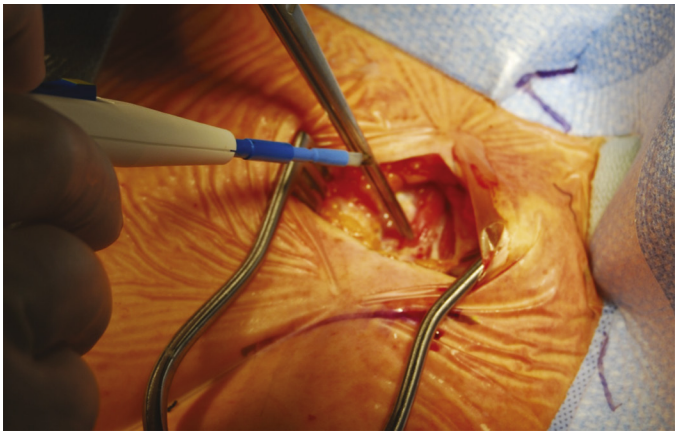
adequate anesthesia to the pectoralis muscle surface (Fig. 7.20). Occasionally it is advantageous to form the pocket between the fascia and the pectoralis muscle to provide an extra layer of protection for the generator in patients with little subcutaneous tissue (Fig. 7.21).

Using electrocautery, the implanter dissects the pocket by carefully separating the subcutaneous tissue from the fascia (Fig. 7.22). Care should be taken to make the pocket large enough to accommodate the device such that there is no tension on the tissue once the pocket is closed.

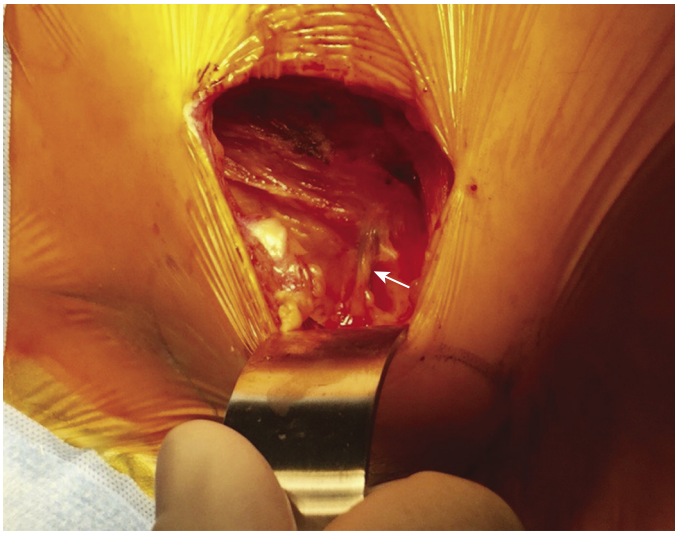
The operator should take care not to extend the pocket beyond the lateral border of the pectoralis major. Once the fascial plane is breached laterally, there is a risk that the generator could migrate into the axilla, causing discomfort for the patient and potentially limiting arm motion. A helpful fluoroscopic landmark is the relationship of the coracoid process to the lateral edge of the



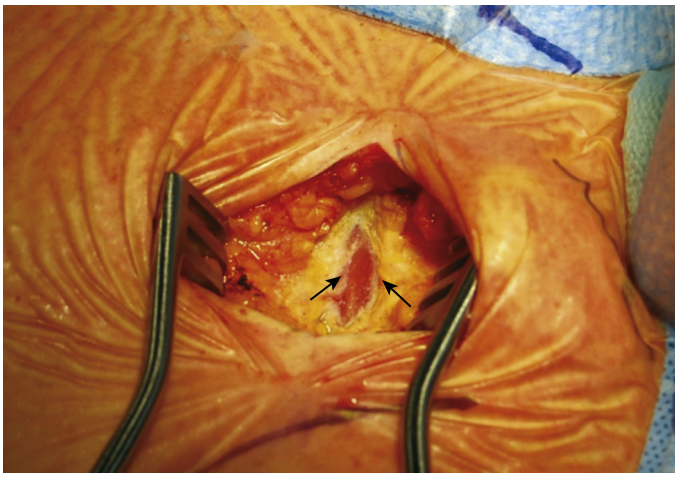
• **Fig. 7.16** Inadvertent contact of the electrocautery tool and the Weitlaner retractor (*arrow*). This may result in thermal damage to the skin.



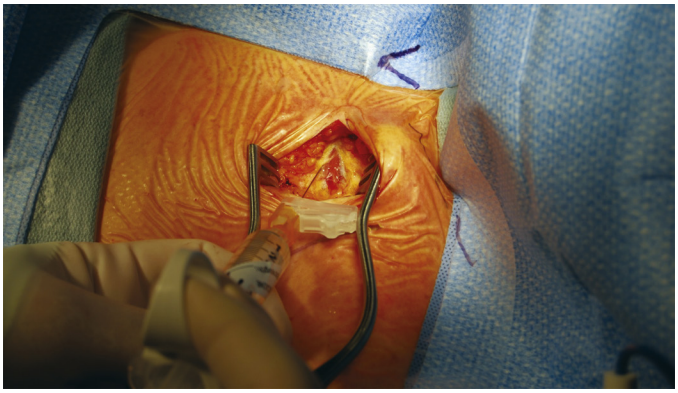
• **Fig. 7.17** Forceps grasping a bleeding blood vessel and the appropriate application of an electrocautery pen to the forceps to transmit thermal energy, resulting in hemostasis.



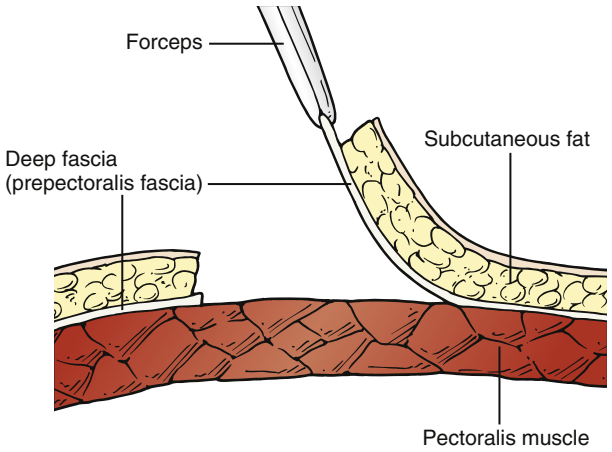
• **Fig. 7.18** Large superficial venous vessel (*arrow*) encountered during a subcutaneous pocket dissection. The size of this vessel will necessitate that it be ligated to achieve hemostasis.



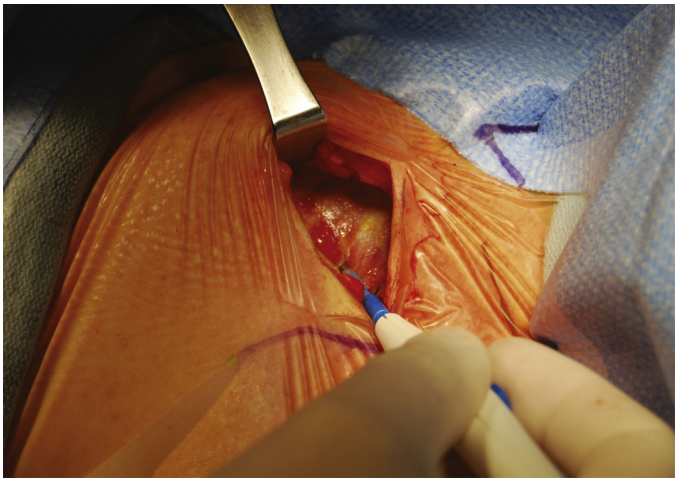
• **Fig. 7.19** A subcutaneous pocket dissection is being performed. The dissection has been carried down to the prepectoralis fascial layer. The borders of the fascia are shown (*arrows*) with the underlying pectoral muscle fibers exposed below.



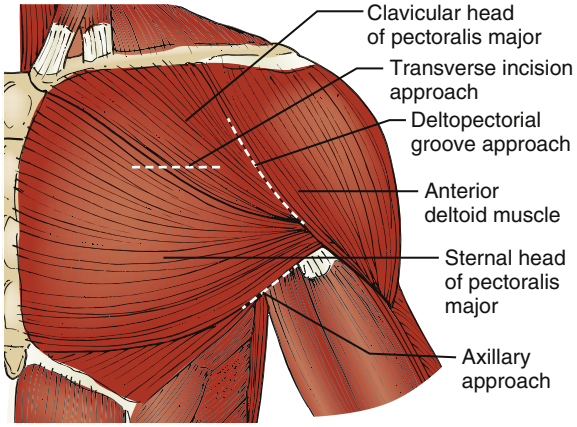
• **Fig. 7.20** Additional injection of lidocaine is administered below the fascial layer to provide further analgesia.



• **Fig. 7.21** Surgical plane for placing a cardiac implantable electronic device generator beneath the deep muscular fascia (prepectoralis fascia), providing an extra layer of protection.



• **Fig. 7.22** Electrocautery is applied to separate the subcutaneous tissue from the prepectoralis fascia.



• **Fig. 7.23** Three surgical approaches to the subpectoral space. The major muscles are identified and the anatomic location of the surgical approach is illustrated (*dashed lines*).

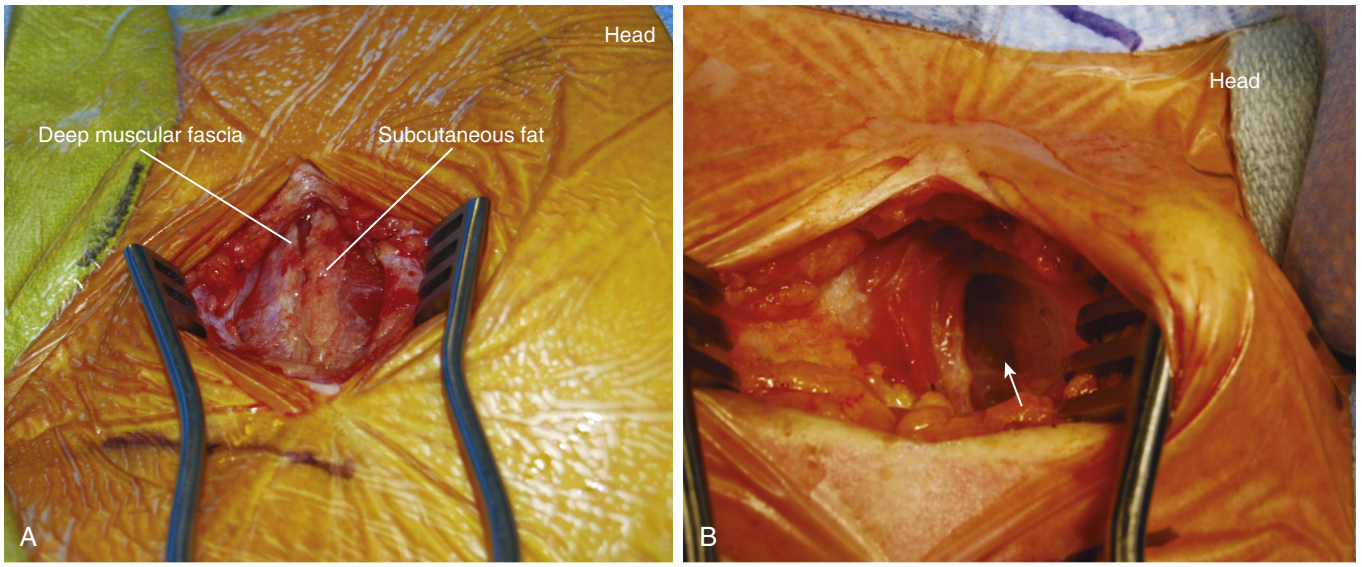
generator. The generator should always remain medial to the coracoid process (see Fig. 7.2). This is more likely to be a challenge using a deltopectoral groove incision as the pocket begins near the lateral margin of the pectoralis major muscle.

Subpectoral Pocket

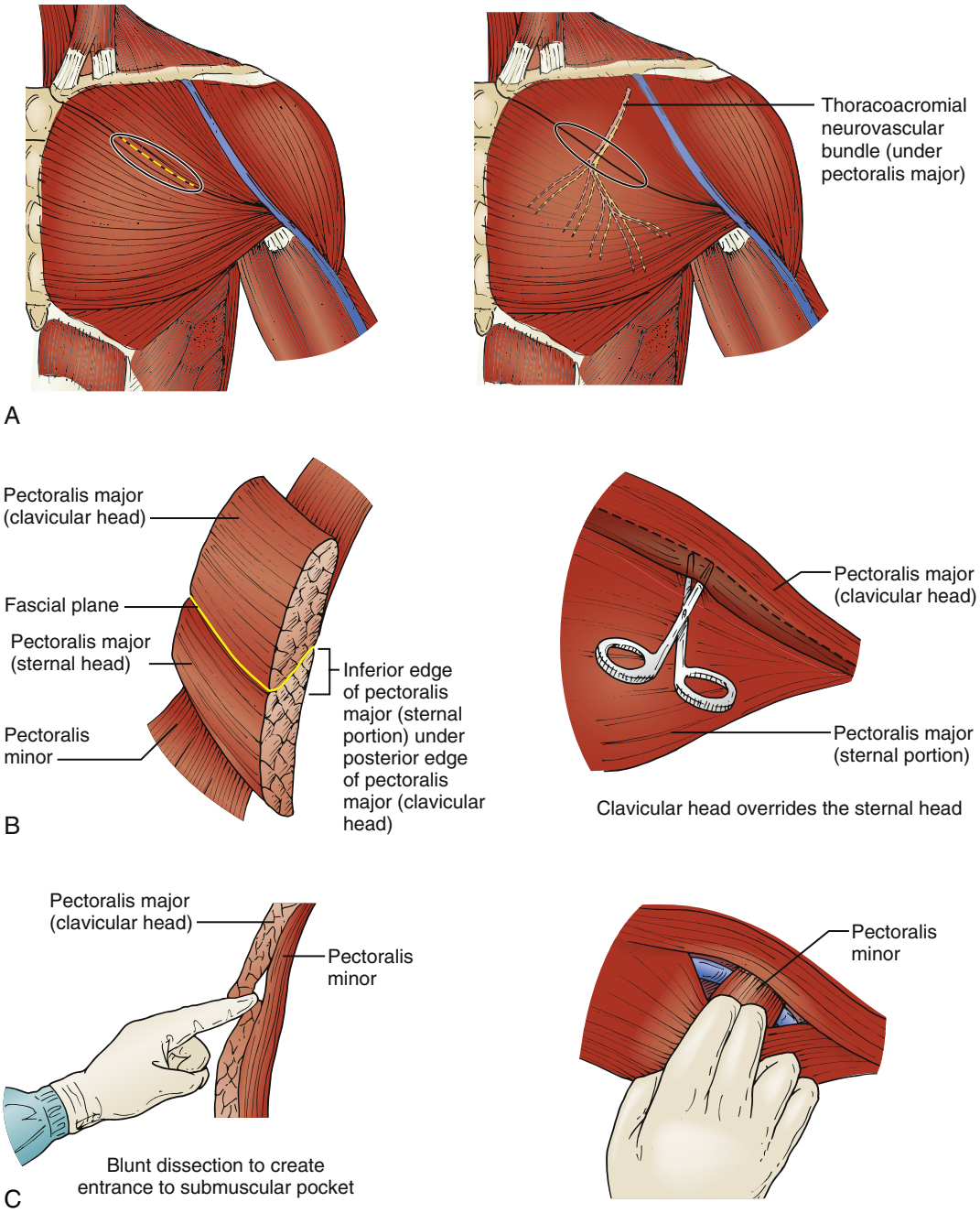
The subpectoral pocket is an excellent option for patients with little subcutaneous tissue, or for cosmetic indications. The two primary approaches to the subpectoral pocket are from a transverse or a deltopectoral groove incision. Occasionally an approach from an axillary incision is used, primarily for cosmetic purposes. These three surgical approaches relative to the pectoralis major muscle are illustrated in Fig. 7.23.

The transverse incision allows convenient identification of the plane between the sternal and clavicular heads of the pectoralis major muscle. This plane can be identified as a thin stripe of fascia and can be separated with Metzenbaum scissors along the fascial plane (Fig. 7.24). Alternatively, it can be identified by brief application of electrocautery to the muscles and observing the delineation between contracting and noncontracting muscle. If the clavicular head is touched with electrocautery, it will contract, whereas the sternal head will not, and vice versa. By using this technique, one can see where the separation of these two muscle bellies are, if it is not immediately apparent.

Once the separation between the pectoral muscle heads is identified, Metzenbaum scissors can be used to separate the fascia gently. The clavicular head overlays the sternal head and thus the dissection is first carried cranially for approximately 1 cm to access the subpectoral space. The implanter can then use his or her fingers to form the pocket with blunt dissection. There should be minimal resistance encountered if within the correct surgical plane between the pectoralis major and minor



• **Fig. 7.24** (A) Thin stripe of fascia denotes the separation between the clavicular and sternal heads of the pectoralis major muscle. In this example, the fascial stripe and a small amount of subcutaneous fat that is overlying the clavicular head of the pectoralis muscle are shown. (B) Separation of the two heads (*arrow*) of the pectoralis major muscle following gentle blunt dissection.

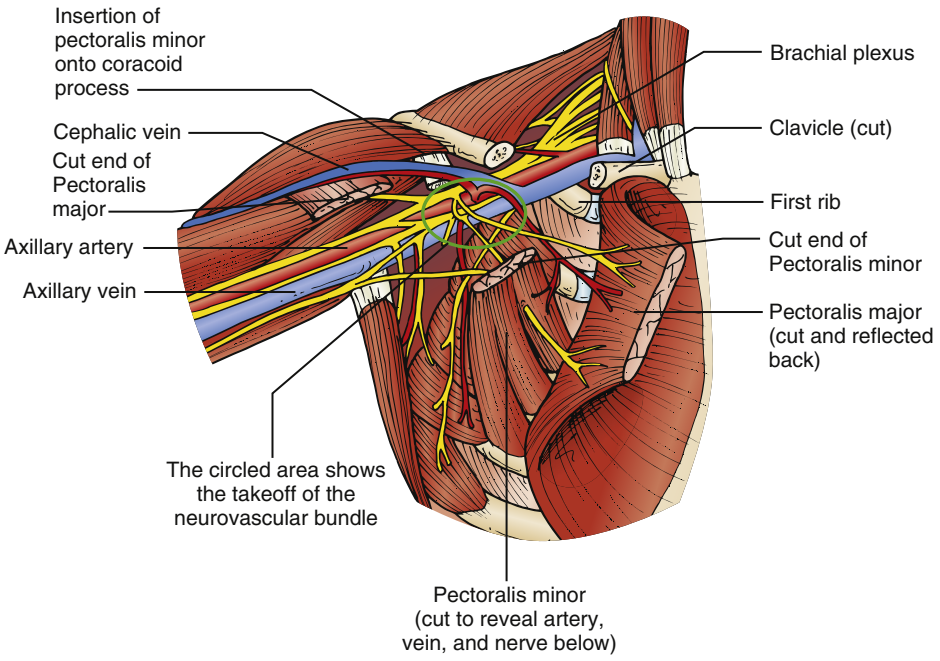


• **Fig. 7.25** Subpectoral pocket formation. (A) *Left,* The fibrous band of fascia (circled) demarcates the separation between the clavicular and sternal heads of the pectoralis major muscle. *Right,* Location of the neurovascular bundle beneath the muscle. (B) *Left,* Clavicular head overlying the sternal head of the pectoralis major muscle. *Right,* Use of Metzenbaum scissors to bluntly dissect between the two sternal heads into the subpectoral plane. (C) *Left,* A finger is used to further the dissection between the two heads of the pectoralis major muscle. *Right,* A finger is used to bluntly dissect beneath the pectoralis major muscle to form the submuscular pocket.

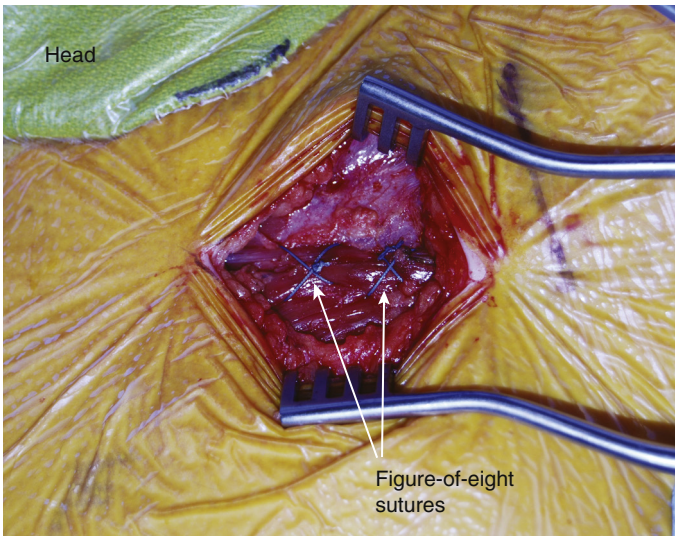
muscles. The pocket should be made only large enough to sufficiently accommodate the device, and care should be taken to not breach the lateral axillary fascial border to avoid device migration. This technique is demonstrated in Fig. 7.25.

When forming the subpectoral pocket, the acromioclavicular neurovascular bundle must be identified and the pocket made medial enough to accommodate the generator in a position that will not place undue pressure on this structure. The arterial and venous branches originate from the axillary artery and vein, coursing on the *anterior* surface of the pectoralis minor, on the underside of the pectoralis major, and laterally to the deltoid (Fig. 7.26). Damage to the neurovascular bundle can result in atrophy of the pectoralis major muscle.

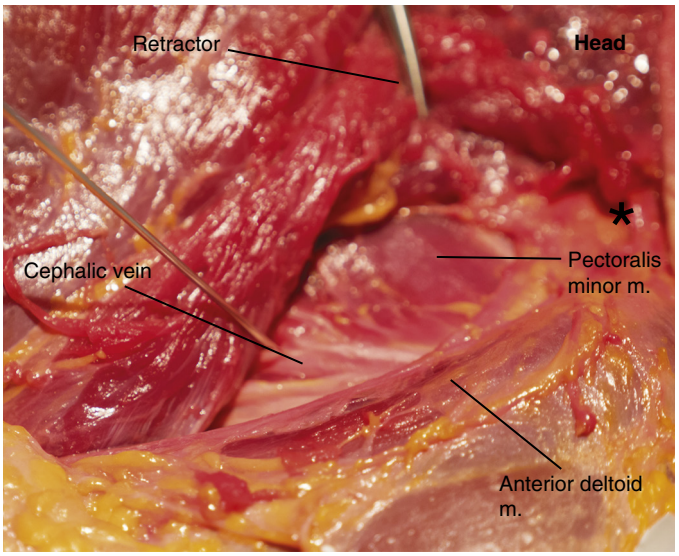
Closure of the subpectoral pocket by approximating the sternal and clavicular heads with nonabsorbable suture using a figure-of-eight closure is recommended (Fig. 7.27) to prevent the lead and generator from herniating through the separation of the two muscle bellies and help identify the plane between



• **Fig. 7.26** Acromioclavicular neurovascular bundle.



• **Fig. 7.27** Approximation of sternal and clavicular heads with a nonabsorbable suture using a figure-of-eight closure to prevent herniation of the generator and assist with identification of the separation between the pectoralis sternal and clavicular heads at the time of generator replacement.

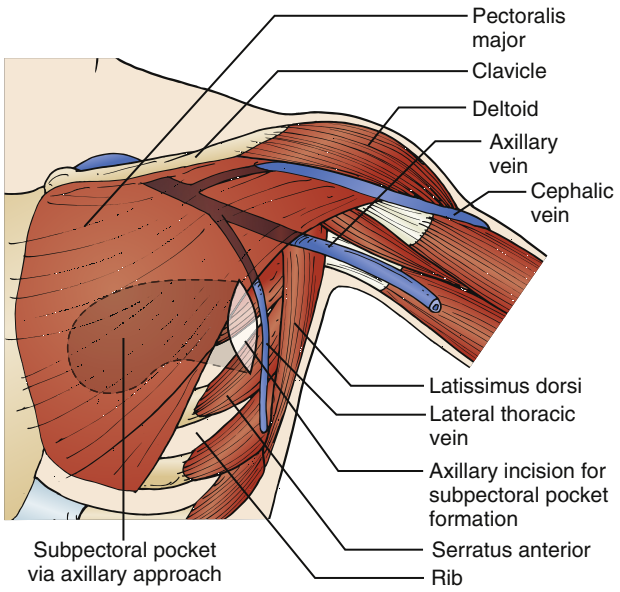


• **Fig. 7.28** Cadaveric dissection of subpectoral approach via a deltopectoral groove incision. The lateral border of the sternal head of the pectoralis major muscle being reflected by the retractor, the cephalic vein, the pectoralis minor muscle, and the medial border of the anterior deltoid muscle are identified. Also depicted is the location of the coracoid process (*asterisk*), the insertion site of the pectoralis minor muscle.

the two muscles at the time of generator replacement. Additionally, a suture placed through the header block and tied to the figure-of-eight suture aids in the removal of the generator from the subpectoral pocket at the time of generator replacement.

When the subpectoral pocket is formed from the deltopectoral groove incision, dissection is carried beneath the sternal head of the pectoralis major muscle. Care must be exercised to identify the pectoralis minor muscle such that the dissection stays anterior to the pectoralis minor muscle and inferior to the pectoralis major muscle and not within the bellies of either of these muscles (Fig. 7.28).

The correct position for the generator is flat against the pectoralis minor muscle. The generator may need to be placed more medial than anticipated using the deltopectoral groove approach. A helpful observation is that the fibers of the pectoralis major muscle run horizontal to the plane of the body, and the fibers of the pectoralis minor run more vertically. Securing the generator with an approach via the deltopectoral groove can be challenging. A long-reaching retractor should be used to elevate the pectoralis major muscle to apply a nonabsorbable suture as medially as possible, usually placed on the undersurface of the pectoralis major muscle.



• **Fig. 7.29** The approach to the subpectoral space using an axillary incision.

A third approach to the subpectoral space is through an axillary incision. In this case, the pocket is formed beneath the sternal head of the pectoralis major muscle. Implanting physicians unfamiliar with this technique should request assistance from their surgical colleagues as this approach might encounter the axillary artery, lymphatic tissue, and the long thoracic nerve. Encountering lymphatic vessels and/or nodes may result in a lymphocele within the pocket, while damage to the long thoracic nerve may result in a winged scapula. For an initial implant, this approach can be performed entirely through the axillary incision, as this provides direct access to the axillary vein for lead placement. The longest length of lead should be used, as several centimeters of lead length will be needed to transverse the distance from the pocket to entry into the heart (Fig. 7.29).

Vessel Choices

The details of venous access and lead placement are discussed in Chapter 8. Here, an abbreviated summary will highlight the salient points.

There are three primary choices for venous access: subclavian, axillary, or cephalic. Rarely, other approaches are required such as the internal jugular vein or even the iliac vein. Gaining proficiency in several techniques is useful if venous access cannot be achieved with the first approach during the procedure. There are benefits and limitations to consider for each choice. The techniques for vascular access are discussed in detail in Chapter 8 and their anatomy described in Chapter 2.

One variation in practice is worth noting: some implanters achieve venous access before making a pocket, then tunnel the leads into the pocket, while others achieve venous access from the pocket after it is created. Most implanters prefer the latter approach as the former may expose the guidewire to the epidermis, allowing for a track into the pocket. Despite good surgical skin preparation, no skin surface is truly sterile. No data support one approach over the other.

Subclavian vein cannulation has been associated with the highest pneumothorax risk owing to its intrathoracic location but has the advantage that the lead courses through the subclavicular tissues flush to the tissue plane. This allows the lead to exit the vessel and lie flat within the pocket with the least angling of the lead, reducing the risk of lead failure at sites in the pocket. Keeping the access to the subclavian vein as lateral as possible reduces risk of damage to the lead (subclavius crush).

If difficulty is encountered attempting to access the subclavian vein, the implanting physician can have an assistant lift the shoulder to open the space between the clavicle and first rib. Other helpful maneuvers include use of the Trendelenburg position or a wedge under the lower extremities to increase venous return to the thorax and further dilate the target vein. Many experienced implanters using this technique have mastered the location of the vein by palpating the suprasternal notch and the bend of the clavicle and then drawing an imaginary line where the vessel courses along the lower one-third of the clavicle. Vascular access is achieved by cautiously advancing the needle toward the suprasternal notch along this line, with care to keep the needle horizontal and immediately beneath the posterior aspect of the clavicle while pointing the needle in the direction of the suprasternal notch.

The axillary vein is a popular alternative to the subclavian vein. The vessel can be accessed extrathoracically by using anatomic landmarks or venography. The vessel, a continuation of the brachiocephalic vein, courses high in the axilla, crossing onto the chest over the first rib where it becomes the subclavian vein. Thus, the vessel can be accessed safely at several points: over the anterior shadow of the first rib or more laterally using the posterior shadow of the second rib as fluoroscopic landmarks to direct the venipuncture. The risk of pneumothorax is reduced using this technique, as long as the operator uses correct technique that includes holding the venipuncture needle at the appropriate angle to the chest (see Chapter 8).

The cephalic vein approach is popular as it is associated with the least risk of pneumothorax; but for those less experienced with this technique, it can take longer to master and to perform. The vessel is isolated by a direct cutdown within the deltopectoral groove. Potential drawbacks are the time it takes to isolate the vessel, transecting the vessel with excessive bleeding, and variations in the vessel potentially affecting lead acceptance. Another potential disadvantage is that the lead/generator system is at risk for being placed too laterally, with impingement on the anterior deltoid resulting in discomfort or interference with upper extremity mobility. An important anatomic difference between the cephalic vein and the subclavian or axillary veins is that there is no accompanying artery to the cephalic vein.

Once the introducer guidewires are placed correctly, a sheath needs to be placed without damaging or transecting the vessel. Most implanters use sheaths with a hemostatic valve to prevent back-bleeding. Placing the sheath can sometimes be problematic, especially through the cephalic vein, which often joins the subclavian vein at an acute angle. Using brief fluoroscopy during this portion of the procedure is important to maintain the guidewire beyond the tip of the sheath and to document that the guidewire is advancing where expected.

Regardless of the venous access approach used, guidewires should always be advanced into the inferior vena cava (or, if this proves difficult, clearly into the pulmonary artery) to avoid inadvertent advancement of sheaths and leads into unintended anatomic locations such as the azygos vein, the coronary sinus, or the arterial system. Overly aggressive movements of the sheath can lead to potentially catastrophic vascular complications including perforation of the brachiocephalic (innominate) vein or superior vena cava. Moving the guidewire back and forth slightly while advancing the sheath (milking technique) can ensure that the wire is truly intravascular and the sheath is advancing smoothly over the wire. Excess force while advancing the sheath should *never* be used. In elderly patients or those with prior leads where the integrity of the SVC wall is altered, the risk of performing this part of the procedure without due caution can result in vascular perforation. Perforation above the pericardial reflection will result in a hemothorax or pneumothorax; if below the pericardial reflection, a pericardial effusion with possible cardiac tamponade may ensue.

Finishing the Procedure

Securing the Leads

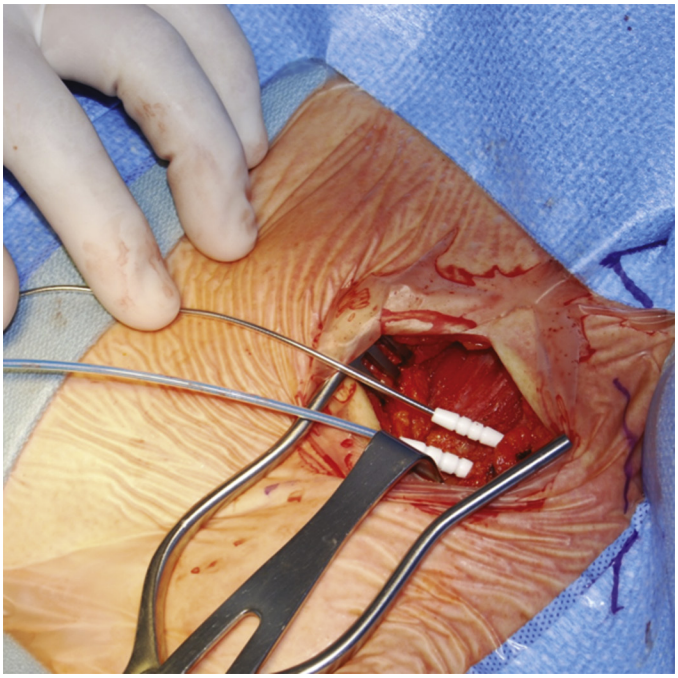
After the leads have been placed in their desired positions (see Chapter 8), the lead-anchoring sleeves are sutured to the underlying muscle using two or three nonabsorbable sutures (Fig. 7.30). This is a critical part of the procedure as the leads can move if not anchored properly. If tied too tight, sutures can compromise the insulation, resulting in lead failure. If tied too loose, the leads may dislodge. The suture is placed around the anchoring sleeve and then sutured to the underlying fascia. Sutures should never be tied directly around the lead body (Fig. 7.31).

After suturing the anchoring sleeves, appropriate lead redundancy should then be confirmed with fluoroscopy. Regardless of the vascular approach used, caution must be exercised to avoid acute angles on the leads when anchoring them. Acute angles can lead to lead insulation disruption or conductor fractures. The secured lead sleeve can potentially act as a fulcrum for excessive lead motion. This can be avoided by ensuring that the lead and lead-anchoring sleeve are flush to the underlying tissue and using gentle curves as the lead is placed within the pocket. When a cephalic vein is used for the venous access, care must be taken to secure the lead sleeve to the pectoralis muscle and not to the deltoid. Any lateral approach to device implant could result in unacceptable angles of the lead sleeve, causing patient discomfort (Fig. 7.32).

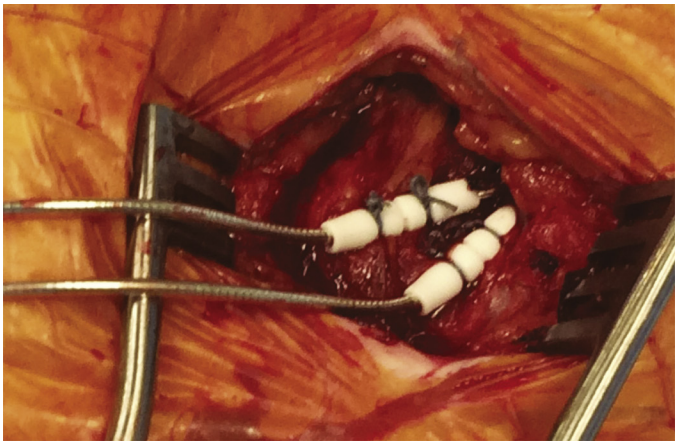
After the leads are secured, the pocket is irrigated with antibiotic solution or plain saline and hemostasis is confirmed (Fig. 7.33). If bleeding is seen following irrigation, a careful search for the source of bleeding must be performed. Sources of bleeding include muscle surfaces deep within the pocket, tissue oozing, or back-bleeding from around the vessel entry sites.

Generator and Lead Attachment

The leads must be placed into the device header block with care. It is never a waste of time to be overly cautious when performing this step. If multiple leads have been placed, the serial numbers of each should be verbalized to confirm that the correct portal is used for each lead. Placement of the wrong lead into the wrong header port could result in loss of pacing in the correct chamber and is an avoidable complication. The wrench should be placed into the header before advancing the lead into the header; some manufacturers' devices need to be "burped" before the lead is placed since an airlock under pressure could cause the lead to push out of the header over time (Fig. 7.34). The pins of the leads should be visualized passing the grommets before the wrench is engaged. A clockwise motion of the wrench is used, with audible clicks confirming secure attachment (Fig. 7.35).



• **Fig. 7.30** Anchoring sleeves advanced along the lead until contact is made with the entry point through the muscle and in a manner to avoid acute angles of the leads.



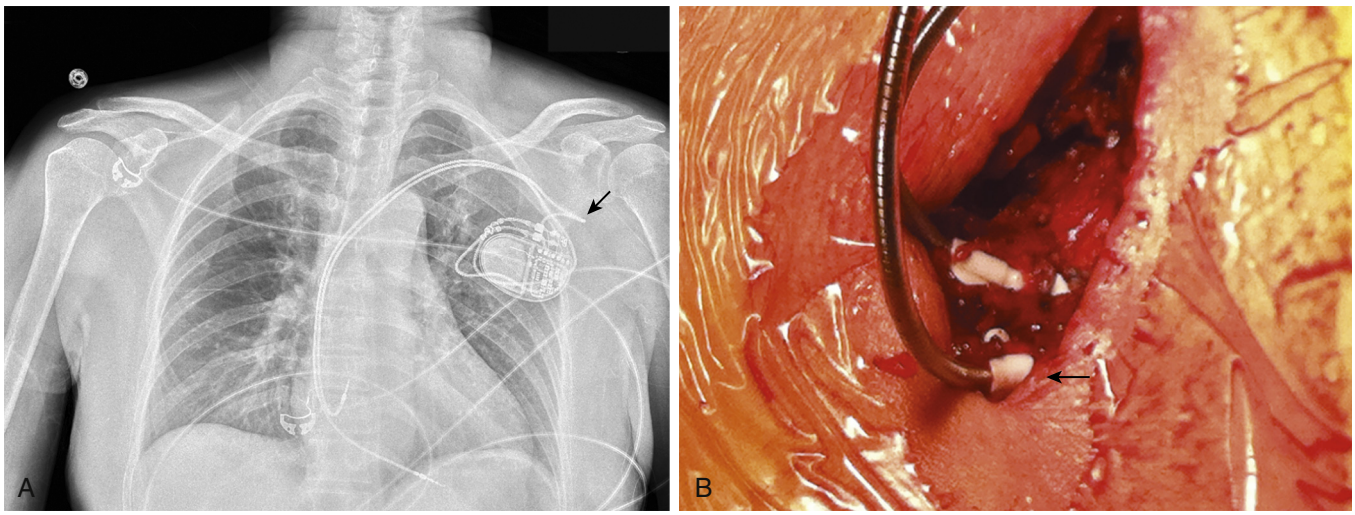
• **Fig. 7.31** Leads should be anchored with a minimum of two sutures per anchoring sleeve. The most laterally placed lead should be sutured first to provide sufficient room for the medial lead to be anchored thereafter.

The implanting physician should then perform a "tug test," pulling gently back on the lead to be certain it remains secure within the header block.

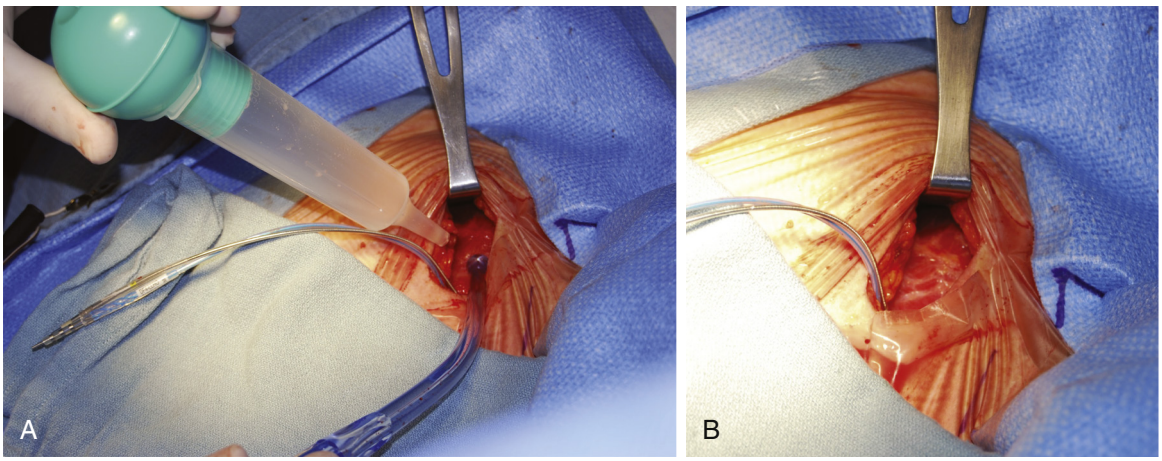
Prior to placing the generator and leads into the pocket, a long-acting anesthetic (0.25% bupivacaine) infiltrated into the pocket and subcutaneous tissue can provide comfort for up to 6 hours. The generator and leads are then positioned carefully within the pocket, with any redundant loops of lead curled behind the generator to protect the leads from blunt trauma and to protect them at the time of reentry into the pocket, where inadvertent nicking of the leads with the scalpel, electrocautery, or other instruments could occur. It should be noted, however, that despite careful attention to this step, vigorous or even normal upper extremity motion often causes the loops of lead to assume positions other than how the implanting physician placed them.

The generator should be anchored to muscle to prevent movement or migration of the device. If the generator pocket is anterior to the pectoralis major, a nonabsorbable suture placed into the clavicular or sternal head of the pectoralis major muscle and suturing hole of the header block is recommended. Care should be taken to ensure a sufficient amount of muscle and fascia are incorporated to prevent the suture from avulsing through or strangulating the muscle and to prevent the suture being placed too cephalad to prevent the generator from abutting the clavicle (Fig. 7.36).

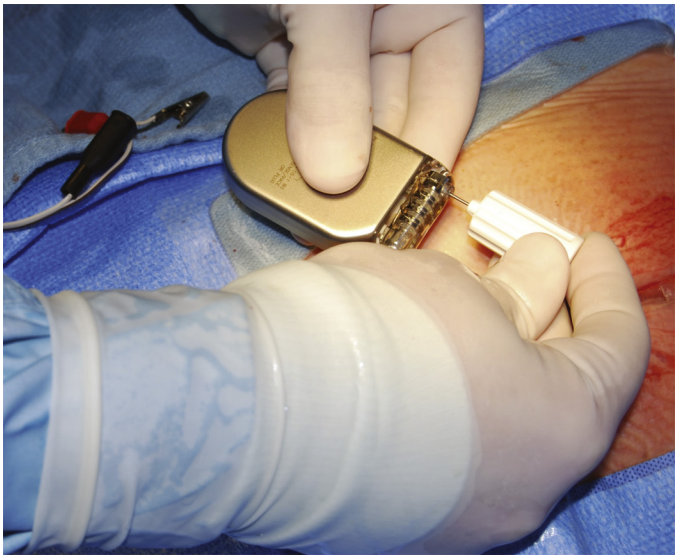
Two options exist if the device is placed in the subpectoral plane. If the device is placed through the separation of the clavicular and sternal heads of the pectoralis major muscle, a nonabsorbable suture placed through the header block and tied to the previously placed figure-of-eight sutures used to approximate the two muscle heads will secure the generator and aid in replacement (easy identification of the correct plane once a capsule has formed). If the



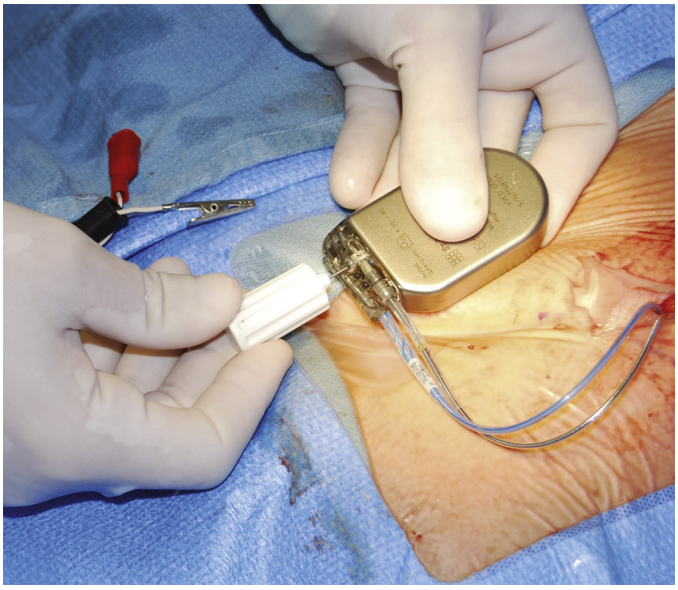
• **Fig. 7.32** (A) Chest radiograph from a patient 3 months after device implant. She complained of pain in her axilla. The radiograph clearly shows an acute angle of a lead in the area of the deltopectoral groove. The area of sharp-angled lead is indicated (*arrow*). (B) Illustration from the same patient at the time of pocket exploration revealed the sharp angles of the lead (angles now relieved after opening pocket and examining the leads) and showed that one of the lead sleeves was angled upward, clearly contributing to her pain (*arrow*).



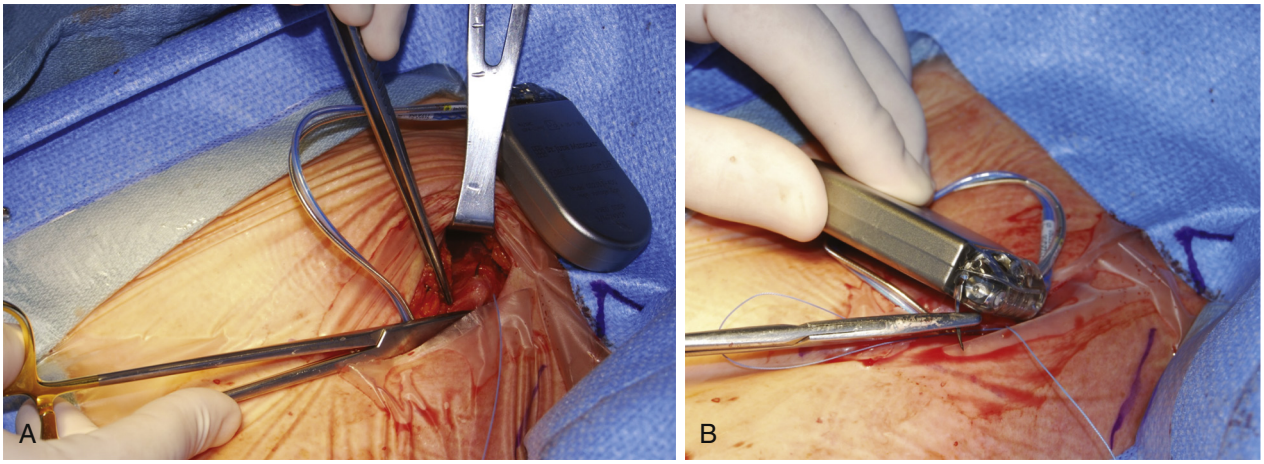
• **Fig. 7.33** (A) Irrigation of the cardiac implantable electronic device pocket using a bulb syringe. The key to this procedure is vigorous irrigation to remove all coagulated blood and demonstrate hemostasis. The physician should then examine the pocket to confirm hemostasis (B).



• **Fig. 7.34** The ratchet wrench is placed into the header block grommet to release the airlock prior to inserting the lead.



• **Fig. 7.35** The ratchet wrench is used to secure the leads in their appropriate header block ports.



• **Fig. 7.36** (A) Nonabsorbable suture placed perpendicular through the clavicular head of the pectoralis major muscle to use for securing the generator within the pocket. (B) Placing the suture through the header block suture hole. In this instance the needle is back-loaded, easing this process by not advancing with the sharp end of the needle, which can catch on the header block plastic. Once the suture is drawn through the header block portal, the needle is removed from the suture and the suture ends are tied.

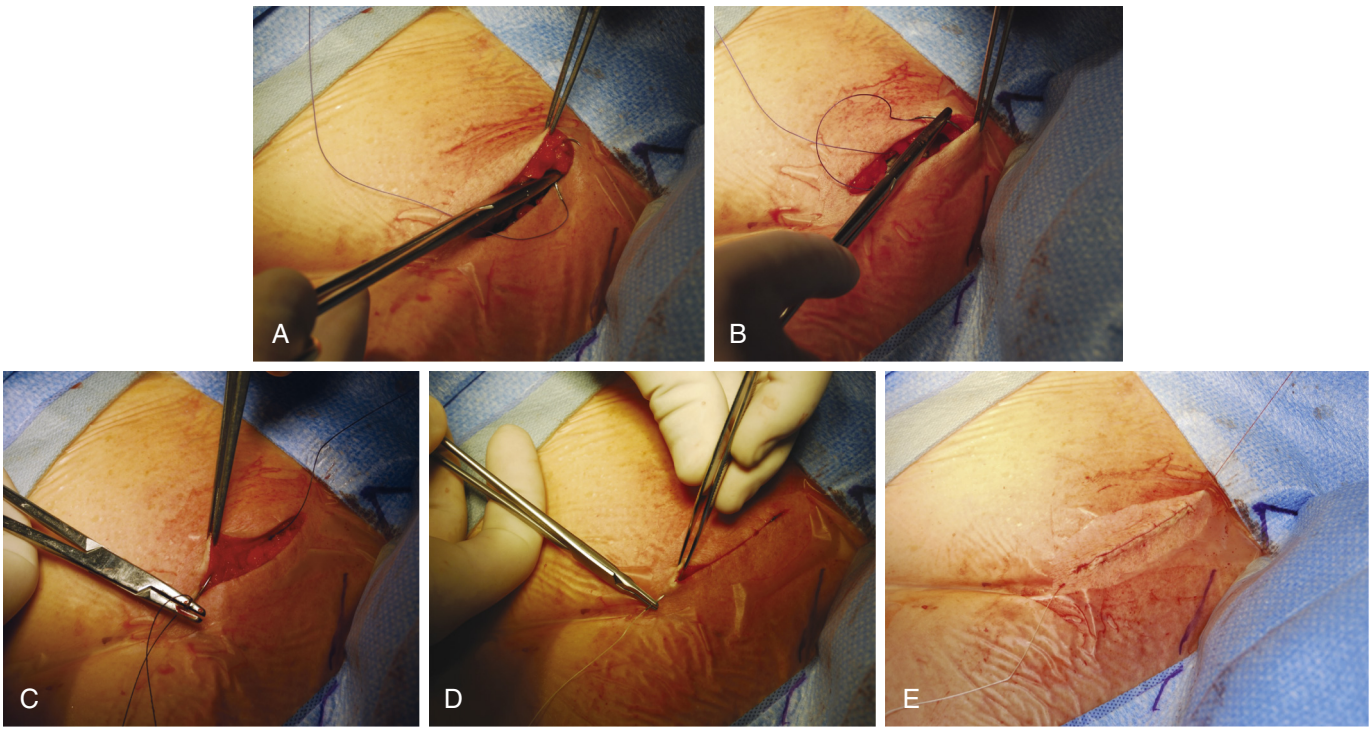
device is placed in the subpectoral space through the deltopectoral groove, the generator should be secured to the undersurface of the pectoralis major, taking care not to encounter the acromioclavicular neurovascular bundle in the process (see Fig. 7.27).

After the leads and generator have been placed in their desired positions, a final check of the lead electrical parameters should be performed. A final fluoroscopic survey of the entire system should be completed prior to pocket closure to ensure adequate lead redundancy, absence of acute angles at the site of the anchoring sleeves, and an appropriately medially positioned generator. Pacing and sensing thresholds should be repeated at this point. These final steps can also identify an early lead dislodgment—it is much better to identify this while still in the procedure lab than once the patient is in recovery.

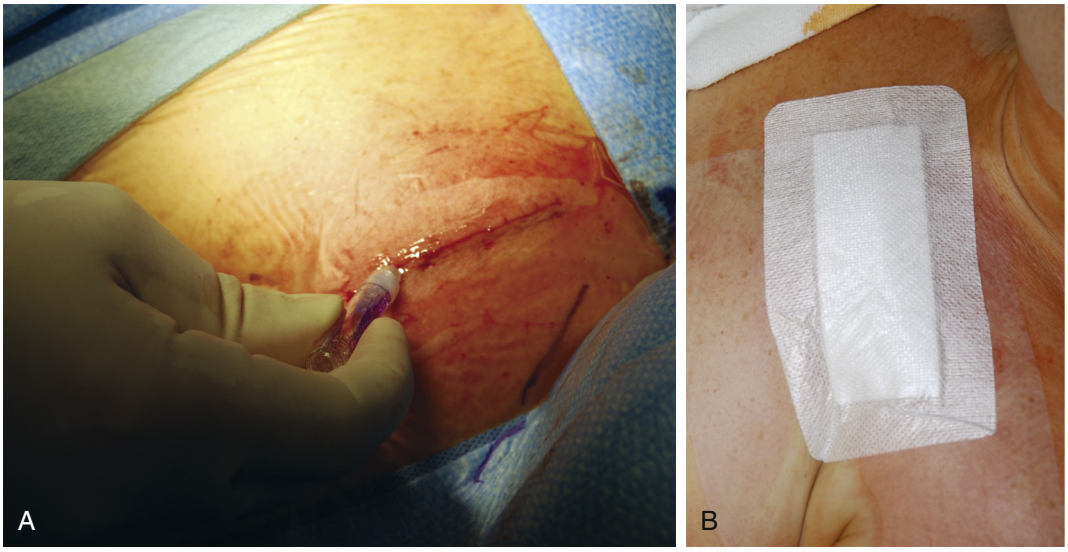
Closing the Pocket

The pocket and subcutaneous layers are closed with two to three layers of interrupted and/or running absorbable suture (Fig. 7.37). Care should be taken to ensure that all dead space is eliminated. The skin can then be closed with a running subcuticular suture or staples.

The wound is then dressed with sterile skin adhesive ("glue"), Steri-Strips, or a combination of both and covered with a nonwoven (Primapore) dressing (Fig. 7.38). Some implanting physicians use sterile skin adhesive rather than the subcuticular suture layer. However, this may not provide adequate skin approximation if the deep layers are not carefully apposed. Using skin adhesive over the final layers allows the patient to shower within 48 hours. If only Steri-Strips are used, the wound must be kept



• **Fig. 7.37** (A) Pocket closure beginning at the deep layer with absorbable suture. (B) Continuation of deep layer closure. (C) Beginning closure of the dermal layer. Careful attention to this layer will affect final wound appearance and alleviates tension from the subcuticular layer. (D) Beginning closure of the subcuticular layer. Note that there is no tension on the skin edges because of careful closure of the dermal layer. (E) Completion of the subcuticular layer.



• **Fig. 7.38** (A) Sterile glue being applied to the wound following closure. (B) Primapore dressing placed over the wound.

clean and dry for 5 to 7 days. A postprocedure chest radiograph is taken to check lead placement and rule out pneumothorax.

Pulse Generator Replacement Procedures

CIED generator replacements are typically performed for battery depletion. They may also be performed during the addition or replacement of one or more leads, or when an upgrade of the system is warranted. Depending on the type of CIED (pacemaker, ICD, CRT), the programming outputs, and the frequency of pacing, replacements may occur anywhere from 3 to 14 years from the initial or previous implant.

Upgrade procedures have become more common as patients with heart failure live longer and their needs change over the course of their illness. These situations may include upgrade from single-chamber pacemakers or ICDs to dual-chamber

devices or to a CRT. Additionally, a patient with a previous pacemaker may develop indications for an ICD.

If the leads are found to have normal electrical parameters during interrogation, a generator replacement procedure should generally be straightforward. However, the implanting physician should always be prepared for the presence of a lead malfunction sufficient to require lead replacement.

A lead malfunction may be evident before the procedure or may be unmasked, or may occur from iatrogenic damage caused during the course of the generator replacement. This possibility should be discussed with the patient (or legal representative) when obtaining informed consent, and lead replacement should be included on the consent form. A "simple" generator replacement can quickly transition into a more complicated procedure.

Patient Evaluation Specific to Pulse Generator Replacements

Patients should undergo a thorough preprocedure evaluation including assessment of their functional status and clinical stability similar to an initial implantation. If the patient has not had a left ventricular ejection fraction (LVEF) documented within the past year, it is reasonable to obtain an assessment prior to the planned procedure. A patient with a pacemaker may require consideration for upgrade to an ICD owing to a significant decrease in their LVEF. Similarly, an ECG may reveal a new widened QRS, suggesting the need for an upgrade to a CRT if there is concomitant ventricular dysfunction.

Lead addition and replacement procedures are not without risk. As discussed in Chapters 13 and 14, major complication rates associated with a generator replacement without lead addition are approximately 4% to 5% over the first year of follow-up. The risk of lead addition or revision carries an even higher risk, largely driven by the comorbidities of the patient and the complexities of the surgical procedure in patients with prior vascular access and indwelling leads.[4,7,8]

The physician should carefully examine the patient's chest where the incision and pulse generator pocket are located. A generator pocket revision may be required if tissue tension is present, the patient complains of chronic pain, or an upgrade to a larger generator size is planned.

As with any CIED procedure, the patient's chest radiograph should be examined prior to the planned procedure. Documentation of the number and type of indwelling leads including any abandoned leads should be confirmed. Often patients are referred or have not been followed continuously by the implanting physician, and assumptions should not be made. The chest radiograph and physical examination can help identify the presence of loops of lead positioned on top of the generator, the location of the lead sleeves, and the presence of acute lead angles, which the physician may want to modify. If the patient had his or her previous CIED implantation performed elsewhere, attempts to obtain the implant notes should be made. It is necessary to determine whether the generator and leads are positioned above or below the pectoralis major muscle, which could affect preprocedural planning.

A careful CIED interrogation should be performed within 6 months of the planned generator replacement for a pacemaker and within 3 months for an ICD. An interrogation should be repeated the day of the procedure. Documentation of the patient's underlying rhythm and all electrical parameters, including programming, lead-related data, and battery status, should be made. High rate episodes should be examined to distinguish possible noise from true arrhythmia. It should be determined whether the patient has had any inappropriate or appropriate ICD therapies or any arrhythmias detected within the treatment or monitor zones. It is critical to determine whether the patient has a lead under advisory or recall status. If such a lead is present, a decision should be made as to the benefit of retaining the advisory lead versus replacement or extraction/replacement of this lead.

In CRT patients, appropriate capture of both left ventricular and right ventricular leads should be assessed prior to the procedure. Situations that may decrease the percentage of true biventricular pacing below ideal levels (≥~95%), such as atrial fibrillation with rapid rate response or frequent premature atrial or ventricular beats, should be recognized. The generator replacement procedure provides an opportunity for the electrophysiologist to review appropriate CIED function and device programming.

In pacemaker-dependent patients, the device should be programmed to an asynchronous mode. If the battery depletion is such that reprogramming to an asynchronous mode cannot be achieved and the patient is pacemaker dependent without an adequate escape rhythm, a temporary pacemaker wire should be strongly considered, or dissection should be performed with a scalpel and Metzenbaum scissors (avoiding the use of electrocautery).

Brief episodes of bradycardia or asystole may be tolerated by the patient during assessment of the leads and transfer to the new generator. However, it may be beneficial to use a temporary transvenous pacemaker in patients with long-dwell-time leads (>10 years) and when pocket dissection is anticipated to be difficult (multiple leads with significant twisting of the leads around each other, obesity, prior generator replacement procedures that identified heavy fibrosis, and fragile clinical status, such as severe heart failure). If a temporary transvenous pacemaker is not used, preparing the femoral area for easy access if an urgent temporary pacemaker is required may be a reasonable alternative.

If lead replacement or addition is anticipated as part of the generator replacement procedure, the patient's upper extremity vascular status must be assessed. As discussed earlier in this chapter, even patients with a single indwelling lead may develop venous thrombosis of the ipsilateral upper extremity. Preprocedure evaluation should include an examination of the anterior chest for evidence of collateral vessels suggestive of upper extremity thrombosis. If present, a preprocedure upper extremity venous ultrasound and/or venography may be warranted. It is reasonable to request examination of both upper extremities to plan appropriately for possible venoplasty of the affected side or use of a contralateral upper extremity vein.

Anticoagulation

Most implanting physicians continue anticoagulation for generator replacement procedures. For patients taking warfarin, an INR should be measured the day of the procedure. If the INR is greater than 3.5, many physicians consider postponing the procedure until the INR is 3.0 or less to avoid excess bleeding, particularly if placement of a new lead is anticipated; however, practice may vary among implanting physicians. Less is known about the target-specific anticoagulant medications and the risk of bleeding. Generally, aspirin does not need to be discontinued, whereas safely stopping the oral thienopyridine class of antiplatelet drugs when possible may be helpful to avoid excess bleeding.

Performing the Generator Replacement Procedure

Incision and Dissection

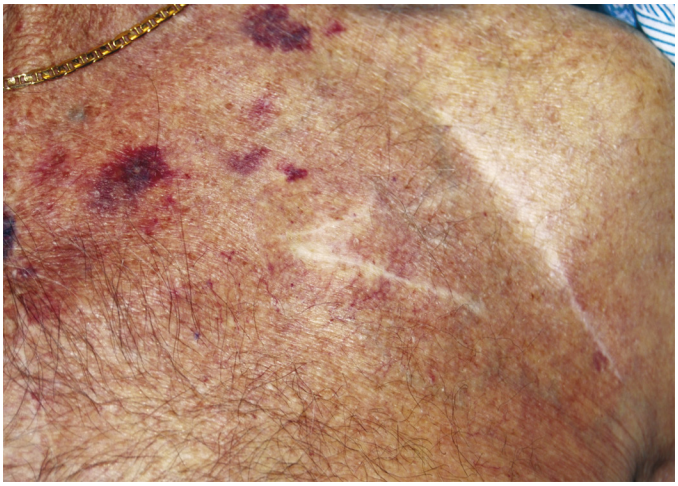
Patient preparation and draping are performed similar to an initial device implant. For patients with an ICD, tachycardia detection and therapy must be disabled before the use of electrocautery. In pacemaker-dependent patients, the use of an asynchronous pacing mode should be considered. Note that unless a specific "electrocautery mode" is available for an ICD, an asynchronous pacing mode may not be available.

If there are concerns regarding vessel patency, the contralateral side can be prepped in the event that it may be needed to complete an upgrade or lead revision procedure. Prevention of infection is important, and general principles of good surgical technique should be used, including the use of preprocedure intravenous antibiotics.

Generally, the incision for generator replacement is made over the existing surgical scar after administration of adequate sedation and local analgesia. Alternate incisions should only be made if the generator has significantly migrated or lead addition is planned and an alternate incision is required for vascular access. As much as possible, multiple incisions in different positions should be avoided (Fig. 7.39). If the previous scar is either hypertrophic or thin and wide, it may be excised to provide fresh skin margins for a more desirable wound appearance and to aid with closure (Fig. 7.40).

Using electrocautery or a PlasmaBlade, the operator must dissect carefully to the capsule containing the generator and leads, taking care to identify any leads that may be overlying the generator. As the dissection progresses, the position of the generator header block should be identified, including the direction of the leads as they exit (Fig. 7.41).

Dissection of the generator at the opposite side of the header block from which the leads exit is recommended to expose as much of the header block as is safely possible to carefully identify the leads. Occasionally, extensive fibrosis prevents the generator from being easily removed from the pocket. A useful tip is to place a heavy suture through the generator header block's suture hole. This can then provide traction to pull the generator more easily out of the pocket (Fig. 7.42).



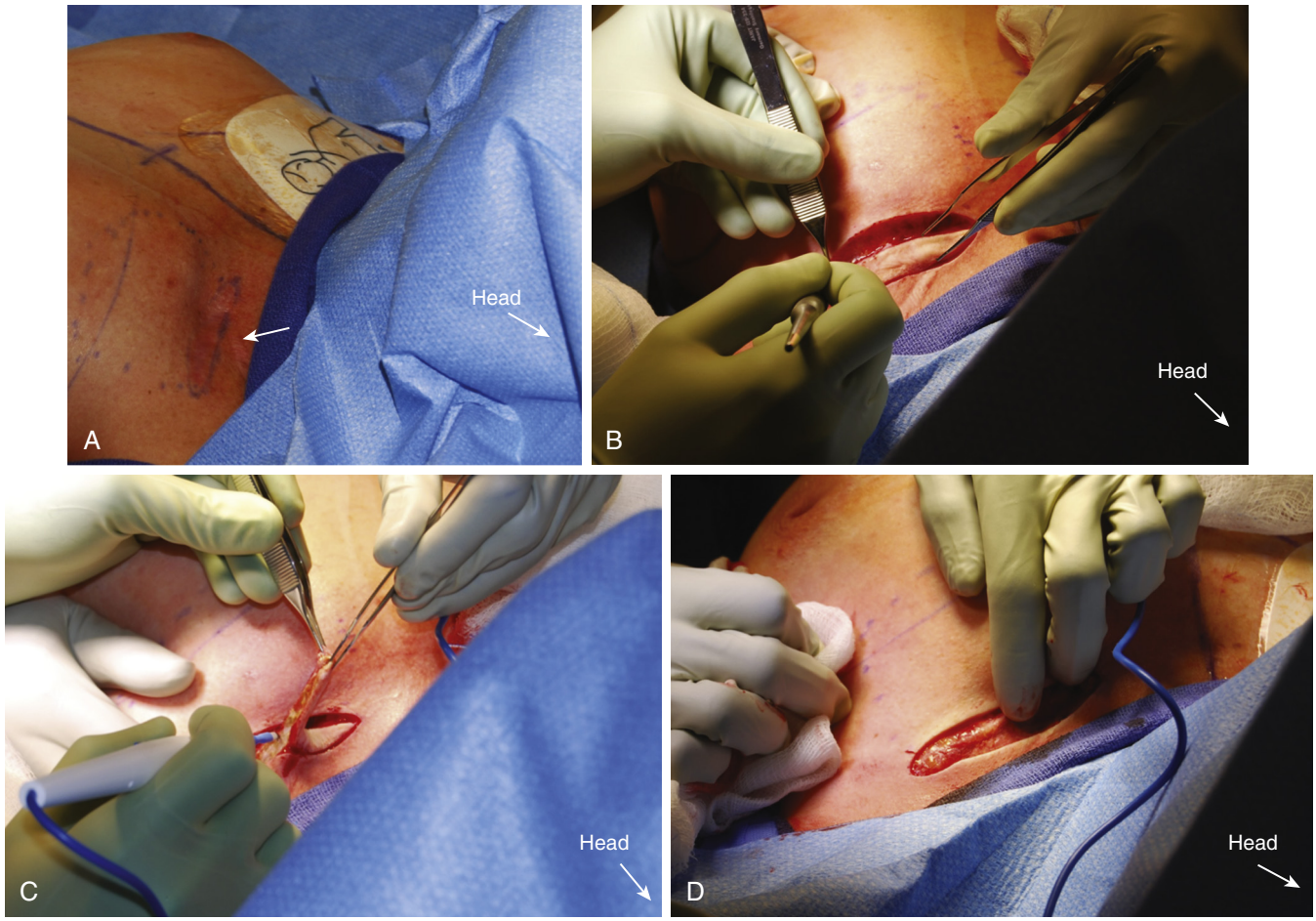
• **Fig. 7.39** This patient had three separate incisions made for implantation and generator replacements. Attempts should be made to perform generator replacement through the original incision if reasonable.

If the leads are incorporated within the fibrotic capsule, they should be dissected free of the capsule fibrosis sufficient to allow for testing and reconnection to the new pulse generator. This may be relatively easy to accomplish unless a significant amount of fibrosis is encountered. In this case, extensive dissection may be required, and the risk of damage to the leads will increase. Electrocautery can be used in short bursts but not enough to damage the insulation.

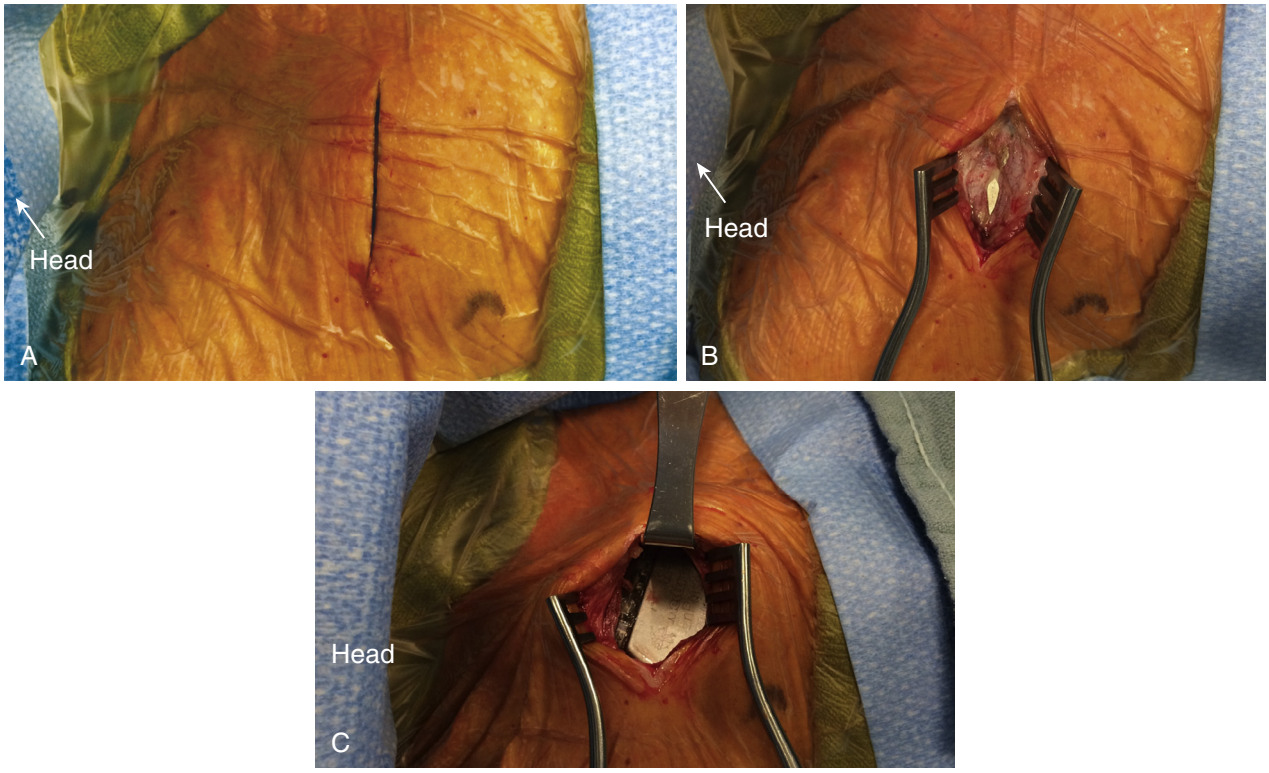
Some implanting physicians have recommended removing as much of the capsule as possible at the time of generator replacement to decrease the risk of infection. This is based on data that have demonstrated the presence of bacteria in pocket cultures in the absence of clinical infection.[9–12] No randomized data support routine use of this approach.

If the operator chooses to remove the capsule, dissection of the anterior capsule should be limited or avoided altogether to prevent compromising the vascular supply of the subcutaneous tissue; this could result in tissue necrosis. An approach that may be less risky to the leads is to remove the generator and capsule in toto (versus first dissecting into the pocket) and then carefully remove the capsule off of the generator and as much of the lead pedicle as is considered safe (Fig. 7.43). Other physicians will "score" the pocket to allow blood to enter the pocket space under the assumption that this will allow white blood cells into the pocket that could decrease the infection risk; however, data are not available to support this or other capsule modification approaches as preventing infection.

Dissection around leads requires extreme care. The electrocautery pen (or PlasmaBlade) should be used parallel to the leads and with short bursts to dissipate potential thermal energy over a larger surface area. Electrocautery should not be applied perpendicular to the lead as thermal energy will be concentrated in a smaller area and increase the risk of thermal damage to the insulation. It is advisable to decrease the output of the coagulation setting while dissecting near leads and to avoid use of the cutting function altogether near any lead[13] (see Chapter 3).

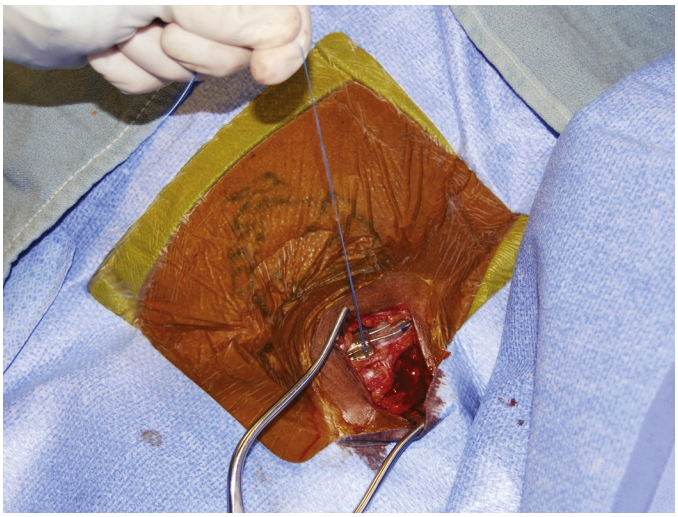


• **Fig. 7.40** Scar revision in a patient who had a thinned and widened scar. (A) Preoperative appearance of the scar *(arrow)*. (B) Resection of the scar. (C) Removal of the thinned scar tissue. Dissection should include the epidermis and the dermis. (D) Complete removal of the scar demonstrating healthy tissue beneath and fresh skin edges.



• **Fig. 7.41** Right-sided pacemaker generator replacement procedure. (A) Incision made through prior surgical scar. (B) Weitlaner retractor in place to aid dissection down to the capsule, which has been incised showing the generator below. (C) Capsule fully opened, exposing the generator lying within the capsule.

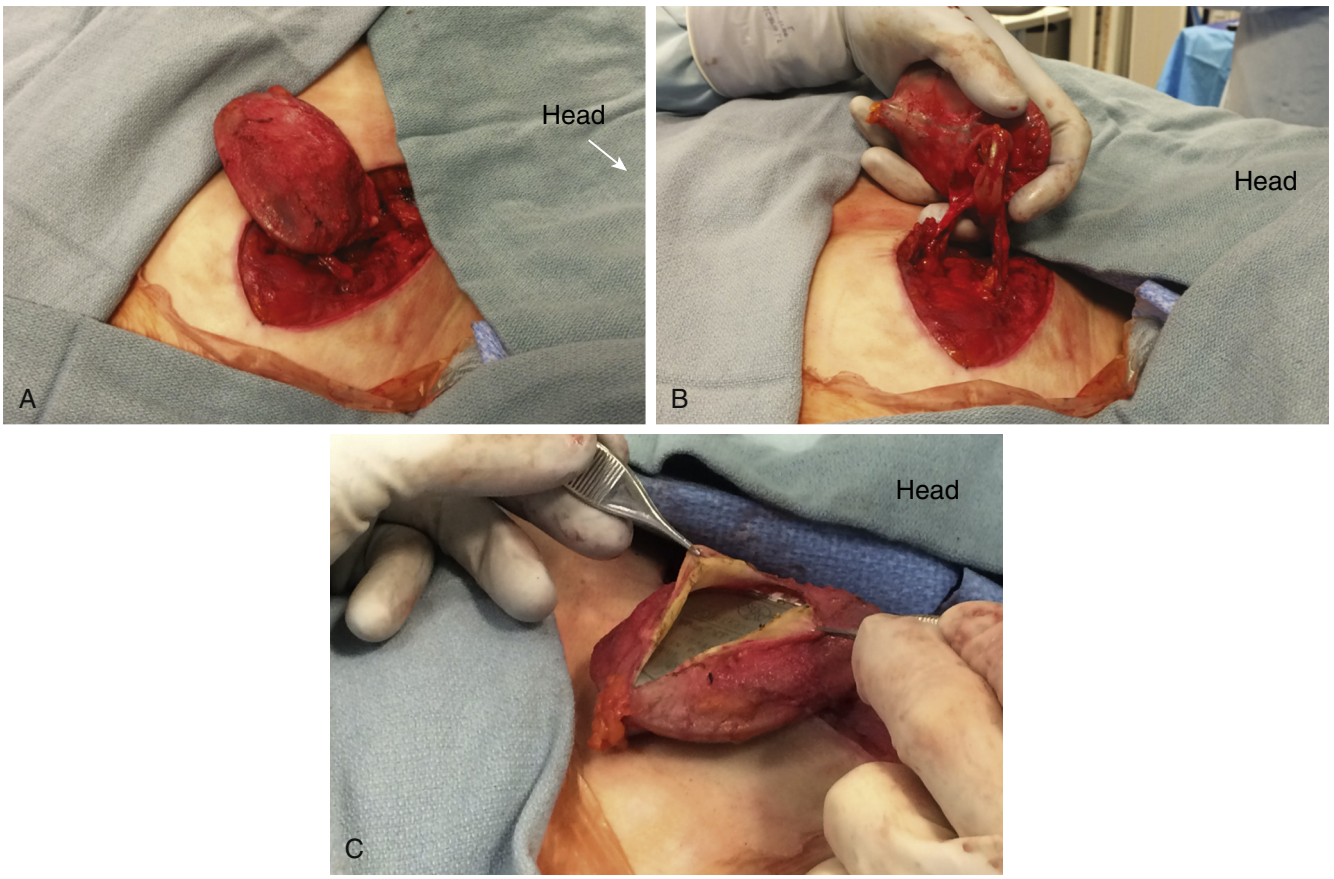
Occasionally, the capsule may have developed significant calcification ("porcelain capsule") (Fig. 7.44). The operator must proceed cautiously as the calcified edges may be sharp and could tear the sterile gloves, cut the operator's finger, or irreversibly damage the lead insulation. In such cases, performing an entire capsulectomy with the generator contained within the capsule as described earlier can be a safer approach. The capsule can then be more easily removed from the generator and leads.



• **Fig. 7.42** Placing a suture through the header block suture hole is a useful technique to provide countertraction, allowing for easier dissection of the generator from the capsule and for removal of the generator out of the pocket.

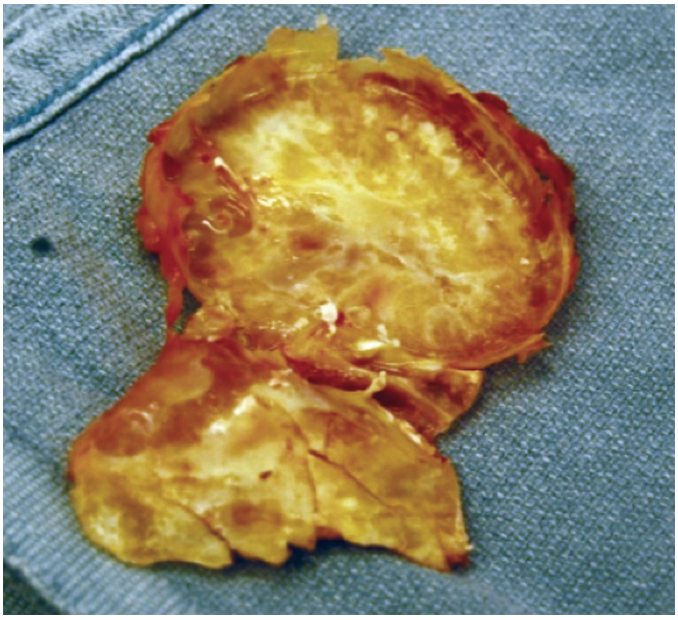
If leads are densely looped or twisted, patience when dissecting is highly recommended to avoid inadvertent damage to the lead insulation (Fig. 7.45). Additionally, if an abandoned lead is known to be present, it should be avoided unless it is truly necessary to free it from its pocket adhesions, as there may not be a protective cap present or it may have slipped off the end of the abandoned lead. The authors recommend reducing the electrocautery output to 20 W when dissecting the leads from any adhesions.[13]

Once the capsule has been entered and sufficient lead length has been freed to deliver the pulse generator out of the capsule, the leads can then be removed from the header block and tested for electrical integrity (sensing, threshold, impedance). In some instances, the leads are difficult to remove from the header block and the implanter should proceed with caution to avoid damage to the lead. All leads should be examined carefully for any evidence of lead damage. This may be apparent as an insulation breach or conductor fracture.



• **Fig. 7.43** Technique to remove the capsule from around a cardiac implantable electronic device generator. (A) The capsule and generator are removed in toto first. (B) The lead pedicle. Care should be taken not to damage the leads and to only remove as much fibrosis as necessary. (C) Taking the capsule off of the generator.

If the insulation is damaged, consideration can be given to repairing the lead depending on the clinical situation and the extent of the breach. The repair is only applicable to leads with silicone insulation. The repair involves applying sterile silicone adhesive over the area of the lead insulation breach and then securing a silicone sleeve over the area with sutures. The device manufacturers can provide lead repair kits for this purpose. It should be noted, however, that this may be a temporizing measure only and lead replacement must be considered as an alternative (Fig. 7.46).

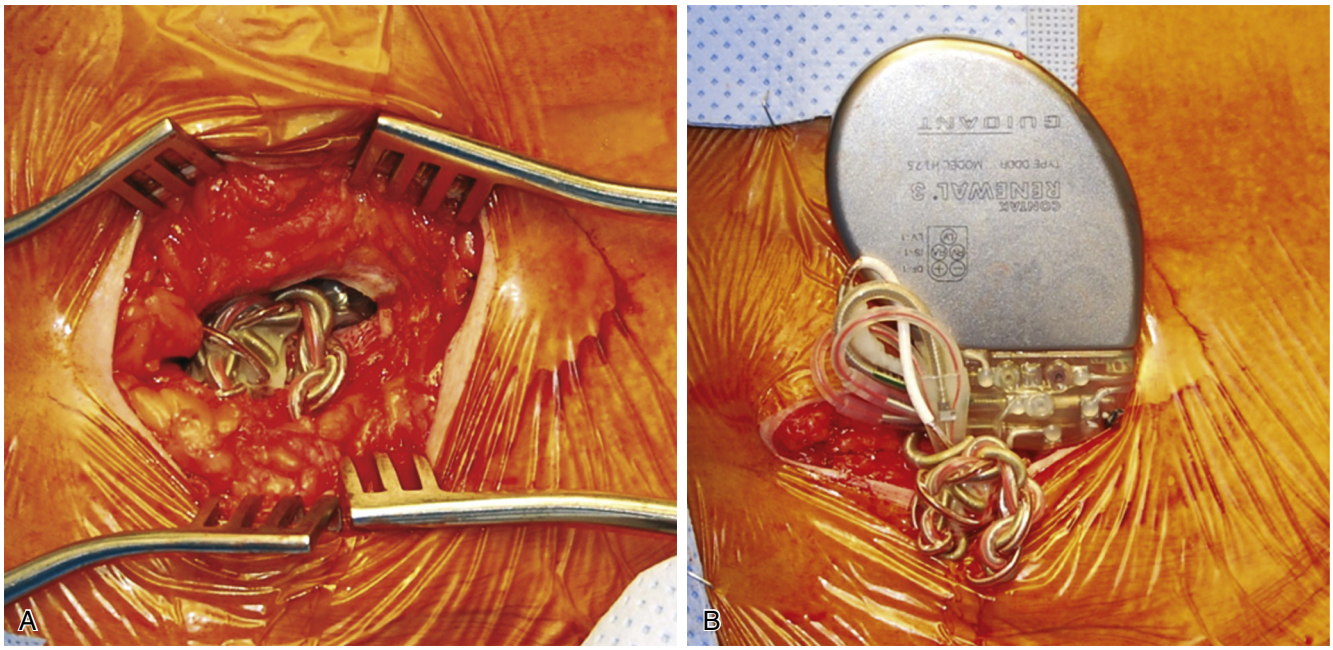


• **Fig. 7.44** "Porcelain" capsule that has been removed from a pacemaker generator.

When multiple leads are present, care should be taken to identify the serial number on each lead as they are removed from the header block. This will ensure the placement of the leads into the proper portals of the new pulse generator header block.

Occasionally, fluid is encountered when opening a pocket. If present, an indolent infection must be considered as the patient may present without classic findings of pocket infection (Fig. 7.47). The fluid should be sent for urgent Gram stain and culture. If the Gram stain does not suggest infection, the operator may proceed with the generator replacement. If the Gram stain or the appearance of the pocket is suspicious for infection, the implanter may have to consider one of two options: reconnect the prior generator and await the final cultures (considering externalizing the generator) while scheduling the patient for lead extraction, or move directly to extraction of the entire system if the operator is experienced in performing lead extraction and the appropriate resources are readily available.

The risk of infection associated with generator replacement has generally been reported to be higher than with de novo implants, likely owing to the presence of indolent *Staphylococcus epidermidis* bacteria (or other bacterial forms). Placing the generator and leads in an antibiotic-impregnated pouch (minocycline and rifampin) is also an option; however, this approach is not used routinely and there is a lack of available randomized data supporting this approach.[14] Pouches with these antibiotics should be avoided in patients with allergies to tetracycline or rifampin. Of course, all patients undergoing implantation of a CIED, either de novo or replacement, should receive preprocedure IV antibiotics, the single antibiotic intervention that has been shown to reduce surgical site infection.[15]



• **Fig. 7.45** (A) Entering a device pocket with observation of entangled leads. (B) The generator and leads are removed from the capsule, illustrating the severity of entangled leads (often called twiddler's syndrome).

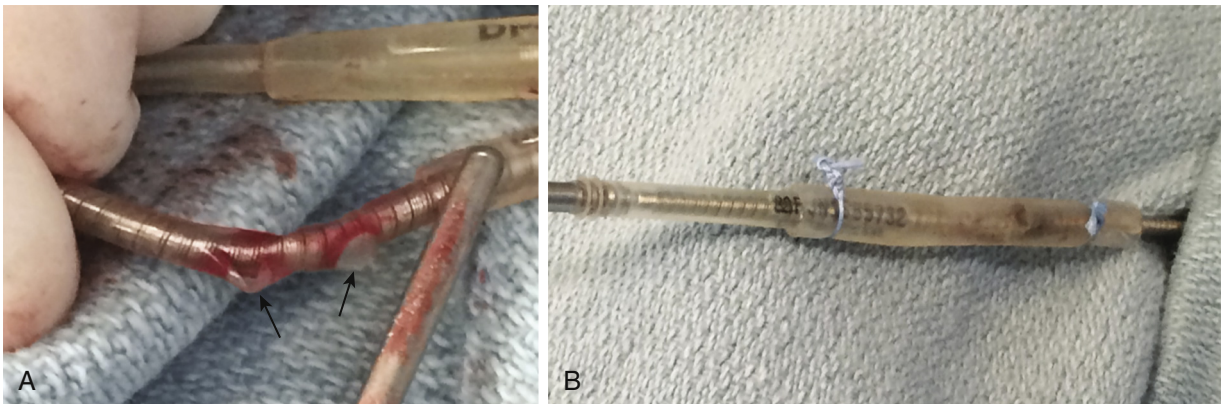
Special Considerations

Subcutaneous Implantable Defibrillator Generator Replacements

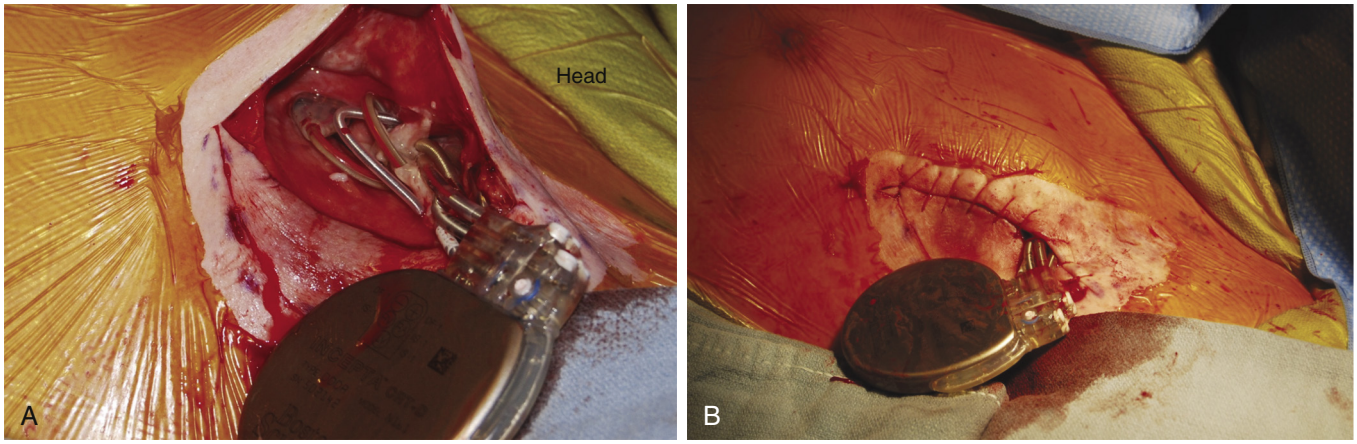
The surgical technique for replacement of the subcutaneous implantable defibrillator (S-ICD) generator is similar to that of the initial implantation (see Chapter 10). Without the need for either a new pocket formation or tunneling for lead placement, the operator may be able to perform the procedure with local anesthesia or conscious sedation. The patient is placed in the supine position with the left arm adducted between 45 and 90 degrees to provide comfortable access to the generator pocket. Appropriate monitoring electrodes and defibrillation pads are placed, and the surgical field is widely prepped and draped incorporating the generator and previous implantation scar. Once tachyarrhythmia detection and therapies are inhibited, the surgical scar from the previous implantation is infiltrated with local anesthetic and dissection is carried down to the generator pocket, taking care to avoid damage to the lead. After delivering the generator from the pocket, the lead is removed from the old generator and attached to its replacement. Following confirmation of hemostasis, the pocket may be irrigated with antibiotic solution. If an antibiotic pouch is preferred by the operator, it is placed around the device at this point. The generator is then repositioned within the pocket and closed in the standard fashion. There should be little or no modification of the pocket required as the latest-generation device is slightly smaller than its predecessor. Interrogation of the lead may be performed through the new generator, and defibrillation testing may be performed based on the clinical status of the patient and at the physician's discretion. Unlike with transvenous ICDs, defibrillation testing is currently recommended for the S-ICD.

Generator Replacements in Patients With a Subpectoral Pocket

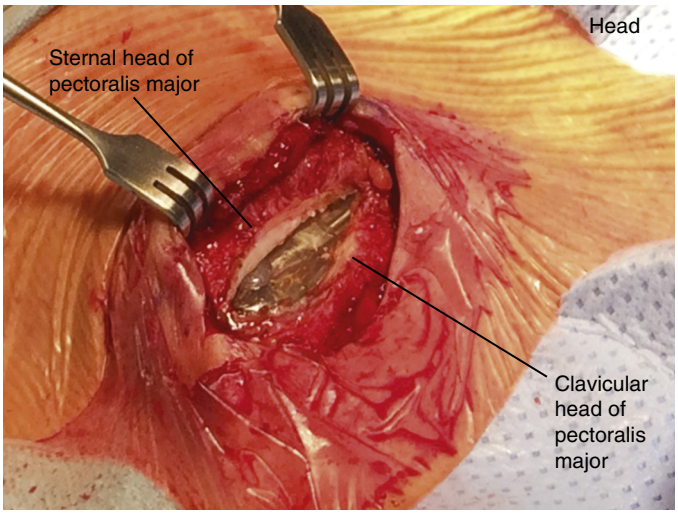
Patients who have undergone implantation of the device and leads below the pectoralis muscle (subpectoral implant) present



• **Fig. 7.46** (A) Identification of an insulation breach (*arrows*) of an atrial lead in a patient undergoing a dual-chamber implantable cardioverter-defibrillator replacement. The patient rarely used the lead for atrial pacing and was also noted to have subclavian vein stenosis. (B) The decision was therefore made to repair the lead.



• **Fig. 7.47** (A) Implantable cardioverter-defibrillator generator replacement procedure. Upon entering the pocket, gelatinous material was found encompassing the leads, which was suspicious for infection. (B) Gram stain confirmed suspicion of infection and the generator was externalized pending system extraction.



• **Fig. 7.48** Subpectoral implantable cardioverter-defibrillator generator replacement procedure. The pocket was originally formed via a transverse incision and separation of the plane between the clavicular and sternal heads of the pectoralis major muscle.

a unique challenge. For those originally approached through a transverse incision, it is imperative that the separation between the clavicular and sternal heads of the pectoralis major be identified to reduce the risk of bleeding.

Once identified, dissection can be directed through this plane without significant bleeding resulting from damage to the muscle fibers (Fig. 7.48). The generator capsule formed in the subpectoral space is typically thinner and less dense than that in the subcutaneous space. Care must be exercised when identifying the course of the leads and their anchoring sleeves, as placement may have originated above the muscle and advanced through the clavicular-sternal separation rather than from below the clavicular head of the pectoralis major at the time of initial implantation.

If the subpectoral space was entered via the deltopectoral groove, it is important to avoid lateral dissection of the generator pocket to prevent migration into the axilla. Additionally, scar tissue may obscure the deltopectoral groove sufficiently enough that identification of the separation between the pectoralis major and anterior deltoid muscles may be difficult, and significant bleeding may be encountered.

For patients in whom the generator was initially placed subpectorally through an axillary approach, referral to a plastic surgeon for assistance is recommended if the implanting physician is not experienced with this approach. In addition to the risk of injury to the axillary artery and vein, injury to the long thoracic nerve in this region can result in a winged scapula.

Pocket Revisions

Revision of an existing CIED pocket presents several unique challenges. Procedural planning and the surgical approach depend on the clinical indication, which may include generator migration; changes in the size, shape, or type of generator chosen at the time of a planned generator replacement; or addition of an absorbable antibiotic pouch. Other indications include chronic pain, impingement upon the clavicle or anterior deltoid, movement of a device inadvertently placed within breast tissue, or threatened erosion.

Postponing a pocket revision to the time of generator replacement is preferable as it avoids an otherwise unplanned procedure and the additional risk of infection from an interim surgical procedure. If the device is impinging upon the clavicle or anterior deltoid muscle, care should be taken to move the generator, leads, and, not infrequently, the anchoring sleeves sufficiently away from those structures to prevent further impingement. For generators that have migrated away from the initial implant site, careful consideration for the best surgical approach, either through the existing scar or through a new incision, is recommended. Closure of the pocket in these situations may require interrupted absorbable or nonabsorbable sutures to decrease the size of the pocket (enlarged owing to the spontaneous migration/dissection) to prevent migration back into the previous site.

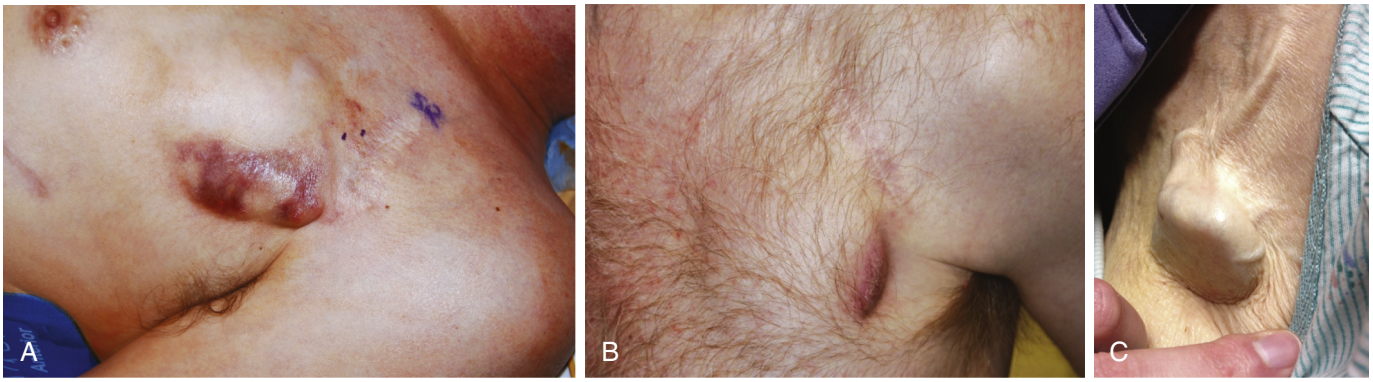
Occasionally, a generator has inappropriately been placed within the fibroglandular parenchyma of the breast tissue rather than in the correct surgical space (upon the prepectoralis fascia). This may result in significant pain, requiring pocket revision. In this circumstance, the generator and leads should be repositioned beneath the pectoralis major fascia or moved to a subpectoral location. Careful dissection of the device and capsule is critical to prevent damage to the glandular breast tissue, which could otherwise cause persistent and significant pain, as well as scarring.

A pocket revision performed for threatened erosion may prevent the generator/leads from eroding through the skin and necessitating extraction of the entire system. This may be possible if there is no evidence of pain, erythema, induration, or cellulitis and the skin over the area of concern slides easily over the device during palpation. If pain, erythema, or induration is present, infection should be suspected and will require complete system extraction (Fig. 7.49).

Regardless of the reason for revision, the procedure should be discussed with the patient beforehand, and the patient should be aware that the device may "feel" different following the procedure. For patients with threatened erosion, care should be taken to inform the patient that the device may be found to be infected; in that case, a revision alone would not be appropriate.

Situations Requiring Lead Tunneling

Occasionally, a patient is found to have subclavian vein obstruction when a new lead is required for replacement or upgrade. The options include lead extraction of a nonfunctional lead to gain venous access, the use of venoplasty techniques, or placement of a new lead on the contralateral side and tunneling to the ipsilateral pocket containing the generator. This is performed by gaining venous access and placing the new lead on the contralateral side. Then, the tunneling may be performed with commercially available vascular tunneling devices (e.g., Atrium Low Profile Tunneling System),

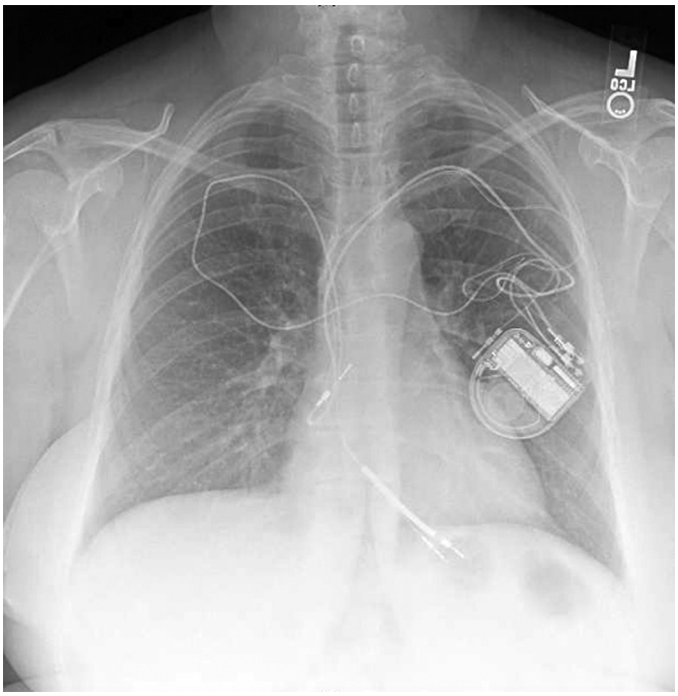


• **Fig. 7.49** (A) Patient referred for pocket revision for the indication of threatened implantable cardioverter-defibrillator generator erosion. Examination had shown induration and erythema consistent with a pocket infection. The figure is taken from the planned lead extraction procedure. (B) Another patient referred for threatened erosion. Note erythema and that the inferolateral border of the generator was adherent to the skin. This is an example of an infected pocket and not threatened erosion. (C) In contrast, a patient with minimal subcutaneous tissue and excess movement of the generator and leads within the pocket at risk for erosion and appropriate for a revision to place the system subpectorally.

a hemostatic sheath placed over a malleable tunneling tool (e.g., Medtronic subcutaneous lead tunneling tool, Boston Scientific SICD tunneling tool), or a long clamp such as a Pean forceps.

The tunnel should be formed within the subcutaneous tissue sufficiently enough to prevent erosion and should clearly avoid the sternocleidomastoid muscles to prevent movement of the lead extension with neck rotation. As such, a tunneling route over the sternomanubrial junction or superior aspect of the sternum is preferable. The new lead is fixed to the underlying pectoralis fascia with its anchoring sleeve similar to standard implantation prior to tunneling the lead. Once the tunnel is formed and the lead has been anchored, the lead may be passed through and connected to the generator in the old pocket (Fig. 7.50). In patients with large chests, a lead extender may occasionally be required to transverse the breadth of the chest. However, lead extenders are not available for ICD leads.

Another situation in which lead tunneling may need to be considered is focal radiation of the breast, chest wall, or lung near the vicinity of the CIED device. This situation could irreversibly damage the circuitry of the CIED. Therefore, the CIED may need to be placed on the contralateral side to the focus of radiation, and the leads tunneled to the new device implant site.



• **Fig. 7.50** Chest radiograph of a patient with a tunneled atrial pacemaker lead from a right-sided access to a previously formed left subcutaneous pocket. Note that the tunneled lead is performed at the level of the sternal–manubrial junction. Tunneling should avoid the insertion of the sternocleidomastoid muscles and sternal wires if present.

Summary

Successful implantation of any CIED device depends on a thorough understanding of the surgical fundamentals of the proposed procedure, efficiency in its execution, innovation when faced with unexpected challenges, and safety for both the patient and the surgical staff. Thoughtful preprocedure planning, sound surgical technique, and familiarity with the dynamic relationship of anatomic structures are equally as important as the indication for the procedure and postprocedure device programming.

Surgical fundamentals begin with patient positioning, prepping, and draping and extend through the incision, pocket formation, vascular access, lead positioning and anchoring, positioning of the generator and leads within the pocket, and wound closure. Surgical efficiency is a product of procedural planning and execution with well-organized and proficient movements, minimizing unnecessary steps; it is not a product of speed. When unexpected findings or anatomic variances

are encountered during the procedure, an understanding of anatomy and its dynamic relationships with surrounding structures will allow the implanting physician to overcome such obstacles. Finally, situational awareness and careful attention to detail during all aspects of the procedure help minimize risks to the patient and the operating physician and ensure the best possible outcome.

Acknowledgment

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8

Transvenous Lead Placement

ANDREA M. RUSSO, ULRIKA BIRGERSDOTTER-GREEN

Introduction

Cardiovascular implantable electronic device (CIED) utilization has grown substantially in response to broadening indications. It is estimated that 10 million cardiac rhythm management (CRM) devices are in use worldwide, corresponding to 7 million leads with a lead implantation rate of 1.4 million/year.[1] The addition of wireless remote monitoring, improved battery life, reduction in device size, and advanced pacing algorithms has substantially contributed to improved quality and outcomes. Lead technology has also advanced with regard to lead size, insulation material, implantable cardioverter-defibrillator (ICD) coil coating, and durability. However, there have been few changes in the traditional transvenous approach to lead placement, which remains the standard pacemaker and form of ICD implantation. This chapter covers implantation strategies for transvenous lead placement, emphasizing the practical aspects of venous access, lead location, and management of potential complications. Multiple techniques are discussed to allow the operator to develop familiarity with several approaches that allow adaptation of the procedure to a specific patient.

Venous Access

Several approaches exist to insert permanent transvenous pacing or defibrillator leads, including (1) cephalic vein cut-down, (2) subclavian vein venipuncture, and (3) axillary vein venipuncture. Less common or rarely utilized approaches include access through the internal or external jugular vein; however, these approaches require tunneling of the lead over or under the clavicle to the site of the pulse generator. This approach may be useful in the case of a more distal venous occlusion. In the presence of limited venous access, insertion of leads through the iliac vein has also been described, but this is very rare.[2] Epicardial lead placement may also be considered as an alternative approach in patients with limited venous access, recurrent endocarditis, or prosthetic tricuspid valves.

Each implantation approach has specific advantages and disadvantages. A relatively common acute complication related to venous puncture is pneumothorax, which may require chest tube placement. This complication is not seen with cephalic vein cut-down, as direct visualization of the vein protects against this. The cephalic vein approach is also associated with better long-term lead longevity. Insulation breaks or fractures due to mechanical injury of the lead and subclavian crush are most often seen with subclavian implantation.

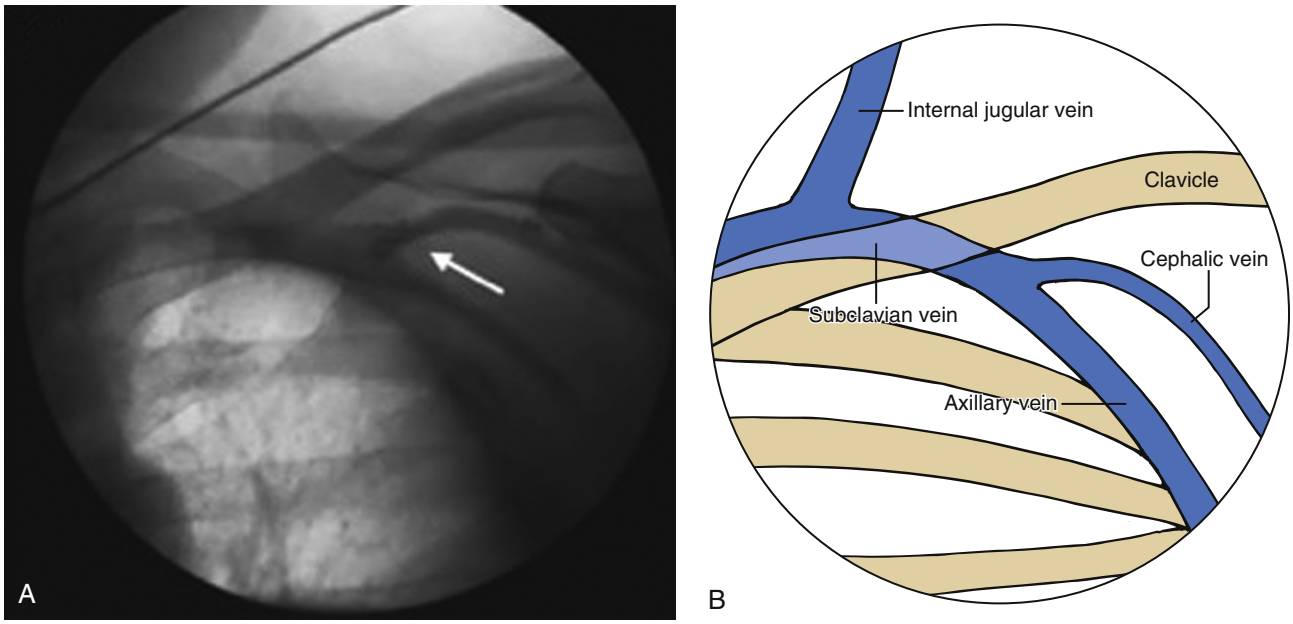
Operators may have greater comfort and expertise with one approach or another. Implanting physicians should be familiar with more than one approach as this will provide options for venous access in difficult cases or when patient-specific factors limit access using a single approach.

Tip: Transvenous pacemaker and defibrillator lead implantation can be performed successfully by multiple approaches using various techniques of which the implanting physician must be aware. On the surface, transvenous lead implantation may appear to be simple, but it is anything but that.

Subclavian Vein Approach

The commonly used subclavian venous approach is often rapid and allows placement of multiple leads through a single subclavian venous stick. Fig. 8.1 illustrates the relationship between the subclavian vein, the clavicle, and the internal jugular vein. A modified Seldinger technique is used to gain access using an introducer sheath inserted over a guidewire. An 18-gauge needle is frequently used to puncture the subclavian vein, although some operators utilize a micropuncture needle. Most operators will make an incision and may even create a pocket before the puncture, although venous access could be performed before the incision with subsequent tunneling of the guidewire into the pocket. Attention to appropriate positioning of the patient is important; multiple pillows beneath a patient's head may make access more challenging. In addition, placing the patient in a Trendelenburg position or administering fluids may facilitate entry if the subclavian vein is not distended—in a recumbent position, for example, a patient is hypovolemic.

Although traditional teaching is to access the vein at the junction of the middle and inner third of the clavicle, this medial approach can predispose the lead to crush injury between the clavicle and first rib (see Fig. 8.1).[3–5] A more lateral approach is now preferred. In fact, depending on the site of access, a lateral approach may provide entry into the axillary vein, which is actually extrathoracic, reducing the risk of pneumothorax.[6] The most consistent landmarks to align the

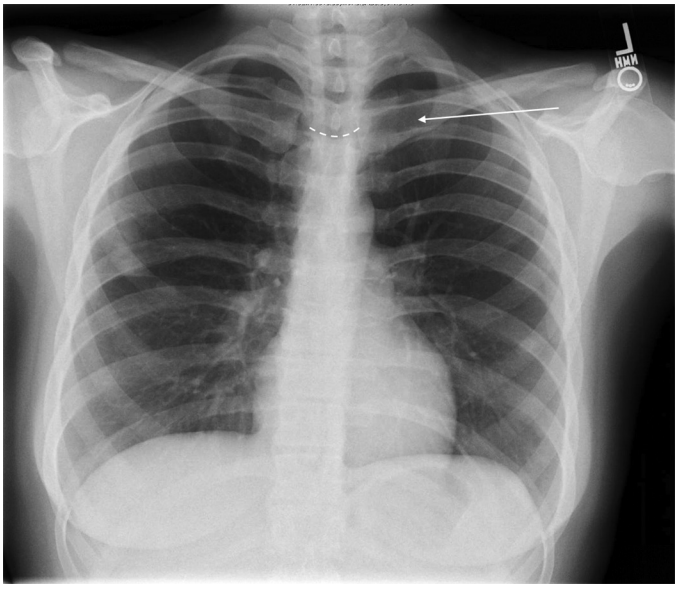


• **Fig. 8.1** Peripheral venography performed to identify location of subclavian vein. (A) The relationship between the axillary vein, cephalic vein, and subclavian vein is shown. The entry of the cephalic vein into the axillary vein is identified *(arrow)*. (B) A schematic also illustrates the relationship of the subclavian vein to the internal jugular vein and clavicle.

needle are the suprasternal notch and the lower one-third of the medial clavicle (Fig. 8.2). As the subclavian vein courses medially, it joins the brachiocephalic (innominate) vein, which lies immediately posterior to the manubrium (see Chapter 2). Many use the clavicle as the primary landmark; however, the fluoroscopic relationship of the clavicle to the lateral subclavian vein can vary depending on patient positioning and body habitus (Fig. 8.3A–C).

A 10- or 12-mL syringe containing 3–5 mL normal saline (and, sometimes, lidocaine) is attached to the needle. The needle is placed through the pocket at the lateral aspect of the clavicle. Once the tip is engaged beneath the clavicle, it is then directed toward the suprasternal notch. While advancing the needle, the implanter aspirates the syringe with a gentle vacuum until a flash of blood is seen in the syringe. If air is seen, this may indicate that lung tissue has been punctured. Once successful access to the vascular system is obtained, the operator should confirm that access is venous rather than arterial. This may be obvious when the syringe is disconnected if pulsatile arterial flow is noted. Alternatively, in the case of hypotension or desaturation, O_2 saturation can be performed from the aspirated blood in equivocal cases. Once the guidewire is inserted into the needle, fluoroscopy can confirm the course of the wire into the right atrium. If inadvertently placed into the arterial system, the wire and needle should be withdrawn with manual compression of the tissues around the vessel to obtain hemostasis.

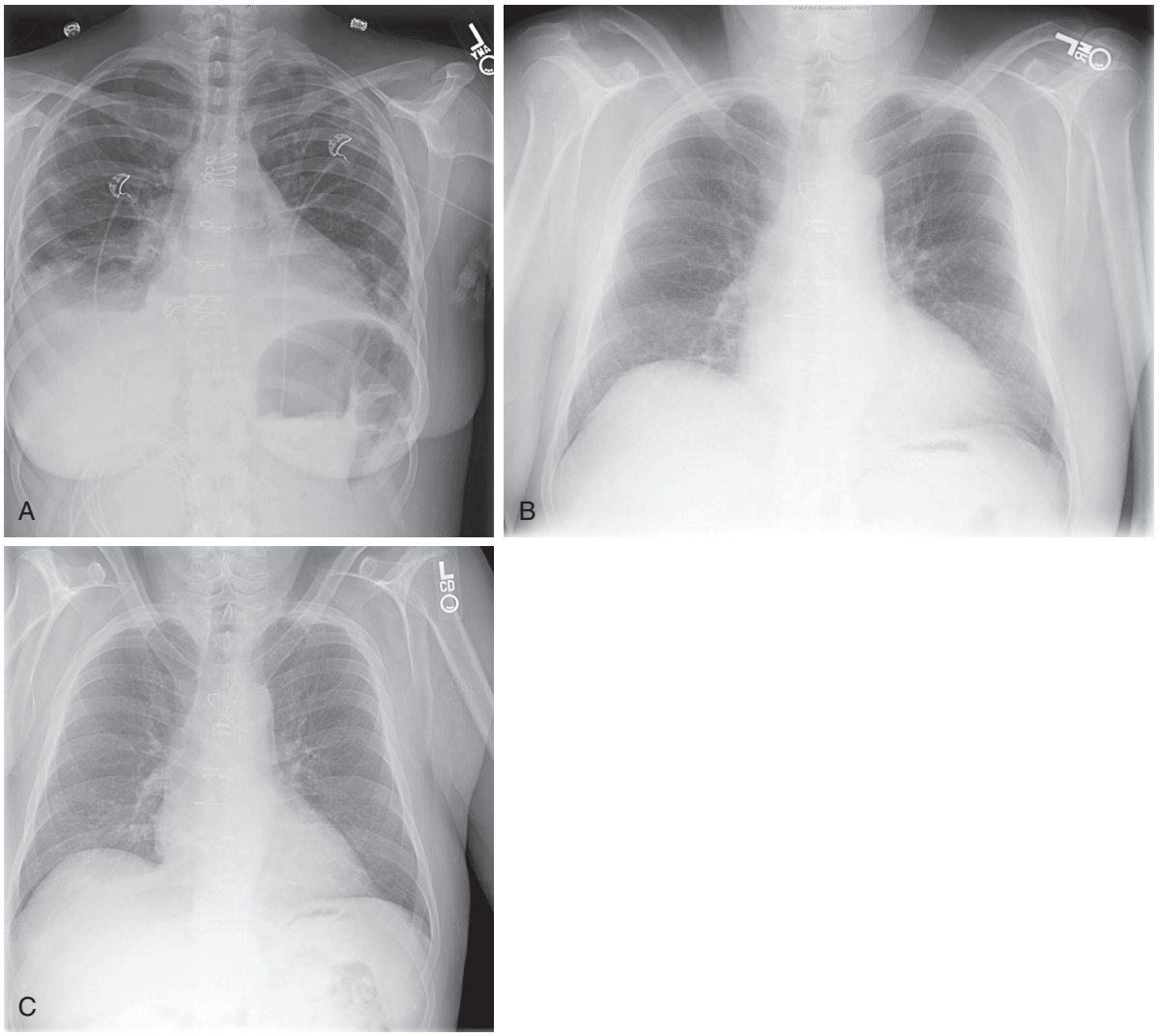
The guidewire should advance toward the right atrium under fluoroscopy (Fig. 8.4). Occasionally, the guidewire may course superiorly into the internal jugular vein, and can generally be redirected using fluoroscopy. In some instances, particularly in the case of preexisting collaterals, a 5-Fr dilator can help steer the guidewire in the proper direction.



• **Fig. 8.2** Chest radiograph (anterior-posterior) demonstrating the suprasternal notch *(dotted line)* and the appropriate direction at which an 18-gauge needle (*arrow*) should be advanced when subclavian venous access is obtained. The needle is advanced in the subclavicular space and along the lower third of the clavicle toward the suprasternal notch.

After the guidewire is placed, the needle is removed and a peel-away sheath is advanced over the wire (Fig. 8.5). The sheath used will depend on the size of the lead or leads used; typically sheaths range from 7 to 10 Fr. If more than one lead will be inserted into the heart using a single subclavian venous stick, the introducer sheath must be large enough to allow retention or reintroduction of a guidewire in the initial sheath.

One method is to use a larger-diameter sheath than required for the lead and, after advancing the sheath over the guidewire,



• **Fig. 8.3** (A–C) Chest radiographs demonstrating variations in relationship of the clavicle to the chest wall and lung apex. The lateral clavicle cannot be relied on as a consistent fluoroscopic landmark for the course of the axillary and subclavian veins.

to remove the vein dilator, leaving the guidewire and sheath in place. A hemostat is placed on the guidewire to secure it onto the sterile drape, preventing inadvertent loss of the guidewire in the venous system. The first lead is placed through the sheath alongside the retained guidewire. After successful placement of the lead, the sheath is removed as described later, and a second sheath is advanced over the retained guidewire to use for the next lead.

A second method includes removing the dilator and the guidewire from the larger-diameter sheath. After the first lead is successfully positioned in the heart, the guidewire can be reinserted into the sheath, and the first sheath can then be peeled away. The appropriately sized sheath is then placed over the newly placed wire. This avoids the need to use a hemostat for securing the wire onto the sterile drape, preventing potential loss of the guidewire while positioning the first lead.

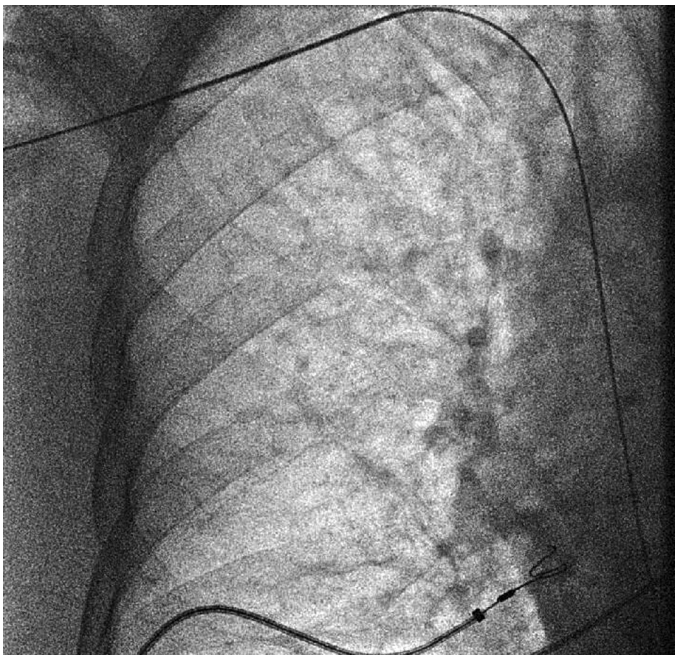
A third method is very similar but avoids upsizing the sheath. After the sheath (sized for the lead) is advanced over the guidewire, the vein dilator is removed, leaving the guidewire and sheath in place. A second guidewire is then advanced through the sheath. The sheath is removed, leaving the two guidewires in place (Fig. 8.6), and a sheath with a dilator is advanced over one of the guidewires after the other guidewire is secured to the sterile drapes (Fig. 8.7).

A fourth approach to multiple lead placement is to perform separate venipunctures for each lead. This has the advantage of decreasing lead-on-lead movement during implantation but increases pneumothorax risk. Implanting physicians will have variations on these techniques depending on their own training and clinical experience. Regardless of the approach used to retain a guidewire for a second (or third) lead, the guidewire(s) must be secured until used.

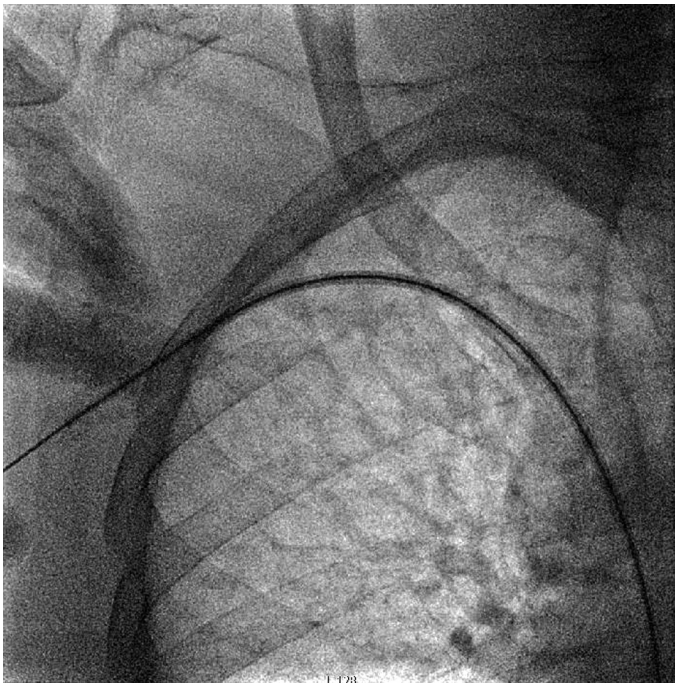
Unlike older technology, current sheaths now have hemostatic valves that prevent back-bleeding. More importantly, these sheaths are essentially airtight and reduce the risk of air embolism during lead placement.

After the first (often a ventricular) lead is positioned, the sheath is then peeled away, holding the lead in a stable position. While peeling away the sheath, care must be taken to keep air

from entering into the heart through the sheath. The first lead can be secured to the underlying fascia using the suture sleeve and nonabsorbable suture. Other implanting physicians will secure the lead after all leads are in place so that final adjustments for redundancy can be made.



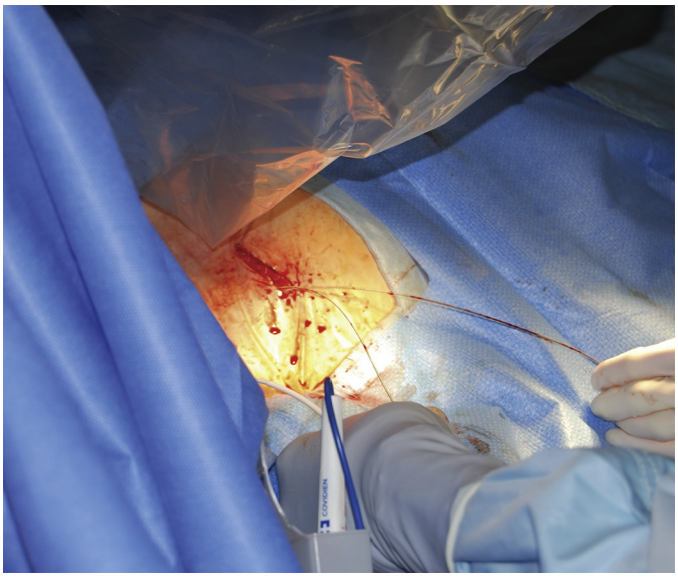
• **Fig. 8.4** Fluoroscopic image of a right-sided cardiac implantable electronic device implant demonstrating advancement of the guidewire into the right atrium. An 18-gauge needle was used to access the right subclavian vein.



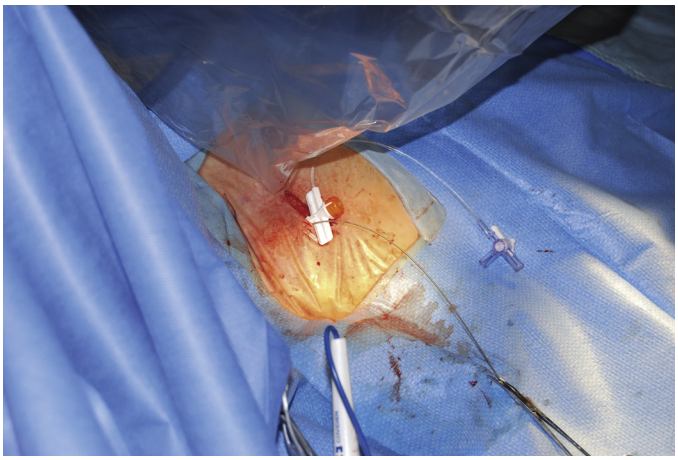
• **Fig. 8.5** Fluoroscopic image of a right-sided cardiac implantable electronic device implant demonstrating advancement of a hemostatic sheath over the guidewire through the right brachiocephalic vein and superior vena cava.

After leads are placed and sheaths are peeled away, each lead needs to be secured to the underlying fascia or muscle using nonabsorbable suture. Manual compression or an absorbable pursestring suture can be used to address any residual bleeding from the access site, if necessary.

Acute complications of subclavian venous puncture include pneumothorax, hemopneumothorax, arterial puncture, air embolism, and brachial plexus injury. Another complication of left-sided approaches with a very medial approach is inadvertent entry into the thoracic duct. The use of intravenous contrast and fluoroscopic visualization may reduce the risk of pneumothorax. After confirming the absence of a contrast allergy, approximately 10 mL of contrast can be injected through an intravenous peripheral catheter in the ipsilateral arm, which is often followed by a saline bolus to flush the contrast. This



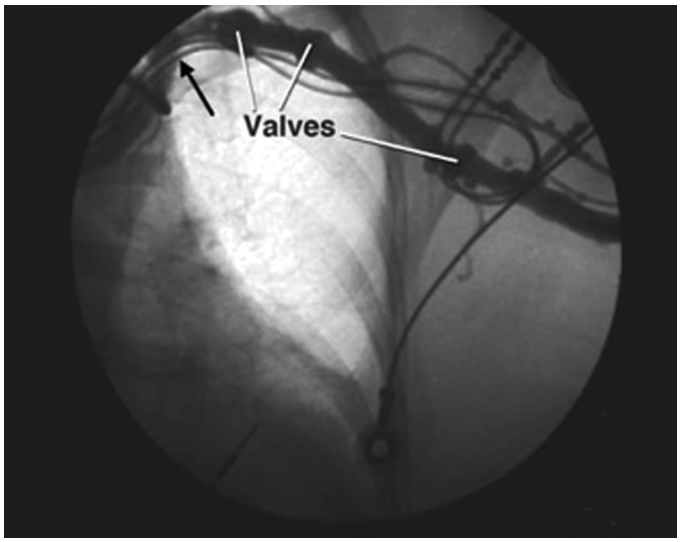
• **Fig. 8.6** Placement of two guidewires into the right subclavian vein using a single venipuncture for a dual-chamber pacemaker.



• **Fig. 8.7** Single venipuncture technique for implantation of a dual-chamber pacemaker. Two guidewires have been placed using the technique described in the text. A hemostatic sheath has been previously advanced over the first guidewire with subsequent removal of the guidewire. The second, retained guidewire is secured to the sterile drape to be used for the second hemostatic sheath and subsequent lead placement.

provides direct visualization of the venous system, and a roadmap can be used to help guide subsequent venous puncture (Fig. 8.8). This is particularly useful in cases where previous leads are in place and subclavian occlusion (Figs. 8.9 and 8.10) or stenosis without complete occlusion (Fig. 8.11) is suspected. Chest wall collaterals may also be visualized (Fig. 8.12A–B). Alternatively, venous ultrasound can help guide venous punctures (discussed later in this chapter). There are sufficient data supporting the safety and efficacy of ultrasound guidance in the case of internal jugular venous access,[7] with less data available for subclavian venous access.[8]

Although subclavian venous access is a relatively safe and quick technique, long-term complications, including subclavian crush phenomenon, can occur.[3] Bony or soft tissue entrapment of the lead by the costoclavicular ligament or subclavius muscle can lead to damage of the lead caused by repeated flexing of the leads during movements of the upper extremity.[4] Avoiding a medial stick can help to reduce or prevent this type of injury.[3–5]



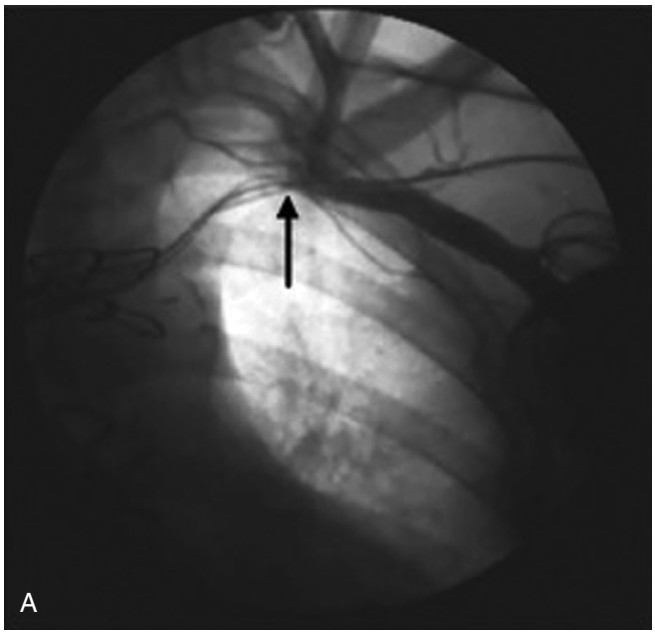
• **Fig. 8.8** Patent axillary and subclavian venous systems. Venous valves are clearly illustrated on this peripheral venogram. Despite the presence of two previously implanted transvenous leads, there is no evidence for significant venous obstruction, and a new lead was easily placed. The initial venous stick appears to have entered the vein in a more medial location *(arrow)*.

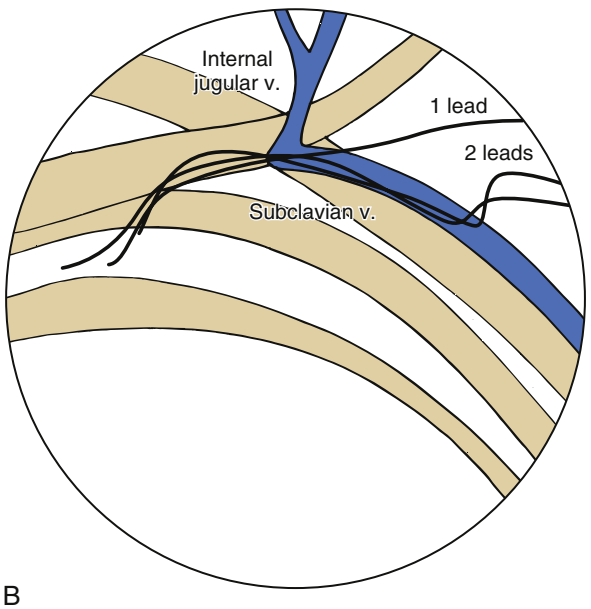
Tip: Although each implantation technique requires separate sets of skills that develop over years of practice, not all can be mastered by every implanter. Nevertheless, it is important for a clinician to be aware of the risks and benefits of each approach and know when to select one over the other. The subclavian approach is the preferred technique for some, but the risks specific to this make other approaches that do not put patients at risk for pneumothorax somewhat preferable.

Axillary Vein Approach

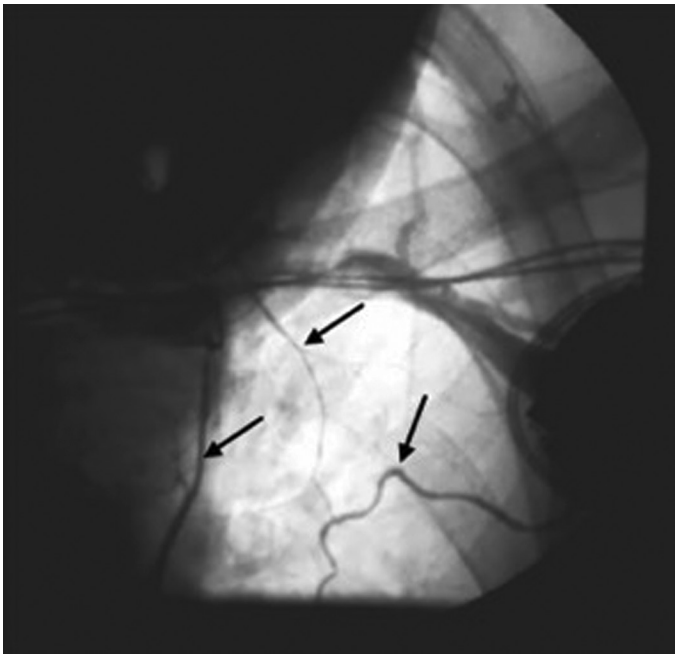
Axillary vein access has become a desirable approach to insert transvenous leads. This approach allows for an extrathoracic venous entry, minimizing risk of pneumothorax and subclavian crush syndrome.[9,10]

The axillary vein begins at the lower border of the pectoralis major tendon as a continuation of the basilic vein. It courses *under* the pectoralis minor at its tendinous insertion onto the coracoid process. It continues toward the clavicle where, at the lateral border of the first rib, it is referred to as the subclavian vein (Fig. 8.13). At the fluoroscopic horizontal level of

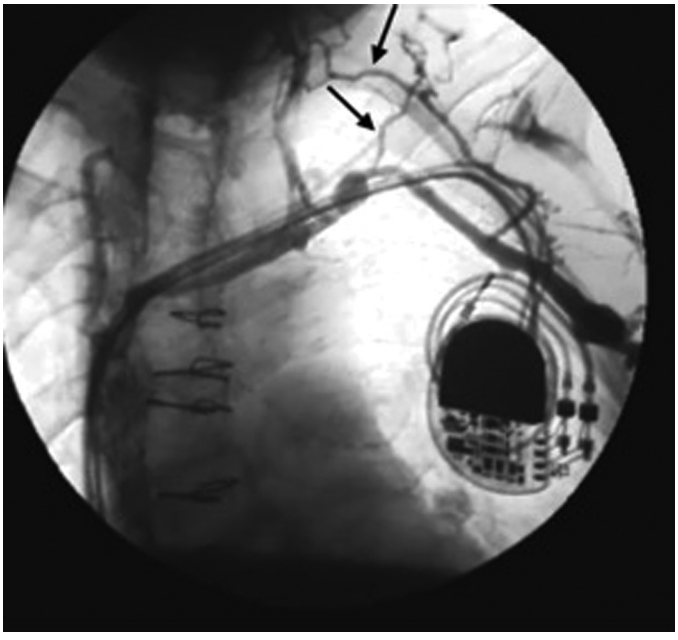




• **Fig. 8.9** Totally occluded subclavian vein. (A) In a patient with previously implanted transvenous leads, the subclavian vein is completely occluded *(arrow)* after the takeoff of the internal jugular vein. (B) A schematic illustrates the relationship between the subclavian and internal jugular veins, highlighting the occlusion.



• **Fig. 8.10** Collateral veins *(arrows)* are noted in this patient with an occluded left subclavian vein. With previously implanted transvenous leads, a segment of subclavian venous occlusion is noted with multiple chest wall collaterals and subsequent patency more proximally.



• **Fig. 8.11** Venous narrowing without obstruction. Despite the presence of collaterals *(arrows)*, complete venous obstruction does not appear to be present. This may be because of recannulation or anticoagulation.

the coracoid process, the cephalic vein, which courses on top of the pectoralis minor muscle, joins the axillary vein. The axillary vein courses anterior and medial to the axillary artery in the axilla and can often be palpated in the deltopectoral groove.

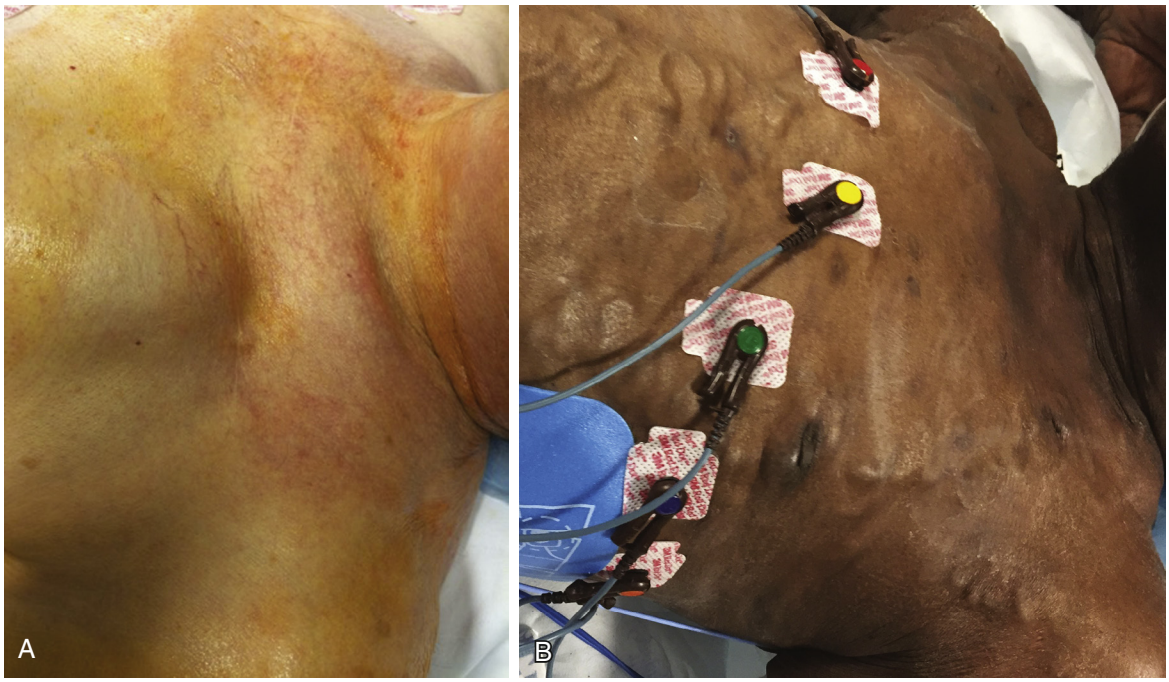
Like subclavian venous access, a venipuncture is performed using a modified Seldinger technique with an 18-gauge needle and fluoroscopy to identify bony anatomic landmarks. Important landmarks for access into the axillary vein include the clavicle, coracoid process, first and second ribs, and deltopectoral groove. The deltopectoral groove is formed by the lateral border of the pectoralis major and the medial border of the anterior deltoid. It can be palpated from the skin surface before making the skin incision (Fig. 8.14A–B). The axillary artery may occasionally be palpable in the deltopectoral groove as the axillary vein runs medial and anterior to this artery.[10] Fig. 8.1 also illustrates the relationship of the axillary vein and the cephalic vein, which drains into the axillary vein.

Several approaches have been described for venipuncture of the axillary vein using the anatomic landmarks noted earlier.[4,9–13]

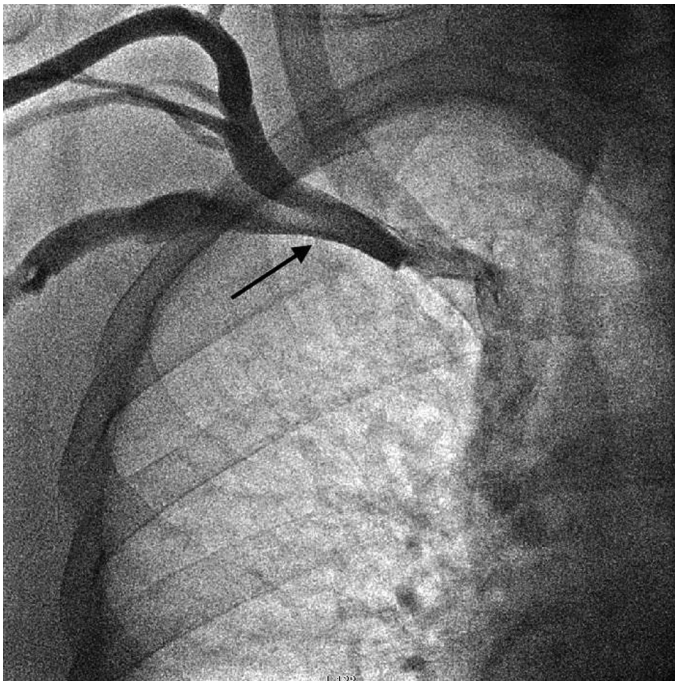
One approach to the axillary vein uses the anterior first rib as its primary anatomic landmark. The needle is inserted through the incision or pocket at a 45-degree angle to the patient's chest and advanced medially toward the fluoroscopic first rib shadow. The target is between the lateral and medial borders of the first rib until the rib is contacted (Fig. 8.15A–B). Again, the needle is then withdrawn while aspirating until blood return is documented, at which time the guidewire is inserted, followed by a sheath. If the vein is not encountered, then the needle is withdrawn and the angle adjusted slightly and the procedure repeated. The needle should never be bent once the tip touches the first rib, which could result in inadvertent "jumping" of the needle, causing trauma to the surrounding tissues. The needle should never be advanced past the medial border of the first rib, as again, the needle could enter the pleural space. Care must be taken to maintain the needle at a 45-degree angle. This will help avoid the needle slipping under or over the first rib and into the pleural space. There is minimal risk of pneumothorax as long as the needle angle is correct and neither too shallow nor too perpendicular to the chest wall, which can also result in entry into the infraclavicular space.[9] If this vein is not easily accessed, contrast venography or ultrasound techniques can be utilized.

As with all venipuncture approaches, factors such as dehydration and anatomic variations can affect successful vessel access.

Review of axillary venograms performed during lead insertion demonstrates that the axillary vein usually courses at the level of or slightly cephalad to the intersection of the inferior border of the second rib and the superior border of the third rib at the margin of the rib cage. Another technique for axillary venipuncture with or without venography was developed. This approach utilizes these fluoroscopic landmarks to position the needle within the deltopectoral groove directed at the shadow of the posterior second rib. The needle is held at an angle of 60–90 degrees to the skin, pointing along the rib cage margin, and advanced while aspirating until blood return is obtained[11] (Fig. 8.16A–B). The course of the needle should not be directed medially beyond the margin of the ribs, as the needle may enter the pleural space and create a pneumothorax. One of the drawbacks of this more caudal approach to the axillary vein is that the needle tends to course through both the pectoralis major and the pectoralis minor muscles/tendon, placing the lead at risk for opposing muscle contractile forces, which increases lead fracture risk. Additionally, because the angle of entry into the vessel is steep, the lead may be subject to acute angles, also increasing the risk of lead fracture.



• **Fig. 8.12** Examples of chest wall collateral veins in two patients with complete occlusion of the subclavian vein. (A) Subtle spider veins and (B) gross varicosities.



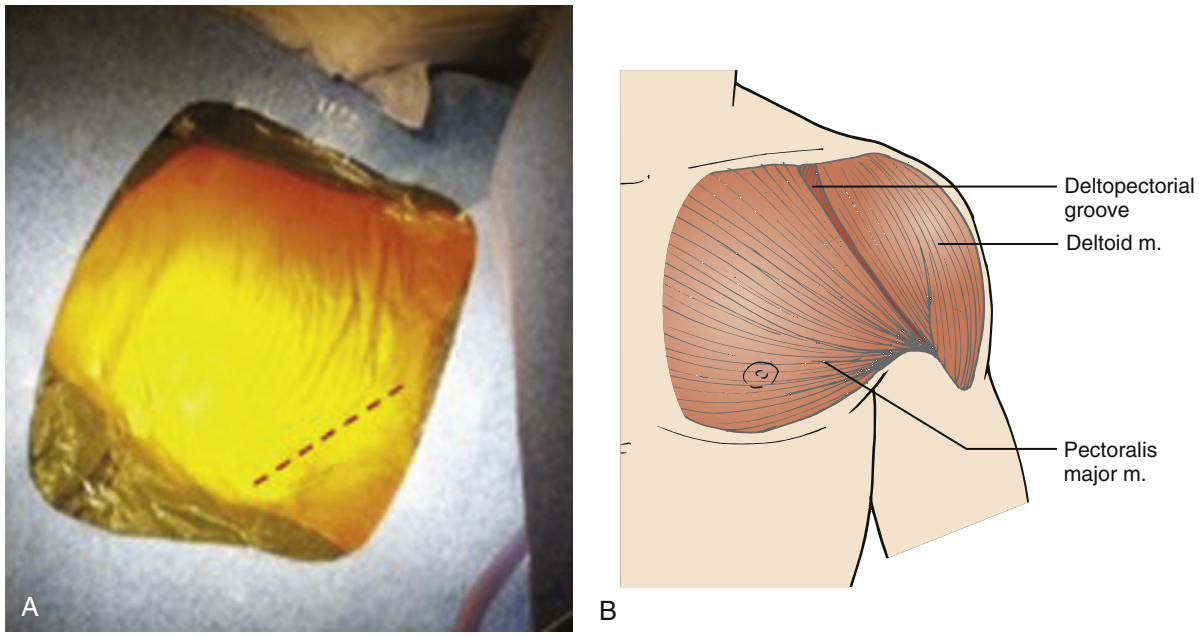
• **Fig. 8.13** Course of the axillary vein, which becomes the subclavian vein at the lateral border of the first rib *(arrow)*. The cephalic vein in this example is unusually large and joins the subclavian vein more medially than in most situations.

A nonrandomized study suggests similarly high success rates of subclavian and axillary vein techniques when performed by experienced operators, although the choice of technique was left to the discretion of the operator and bias cannot be excluded.[14]

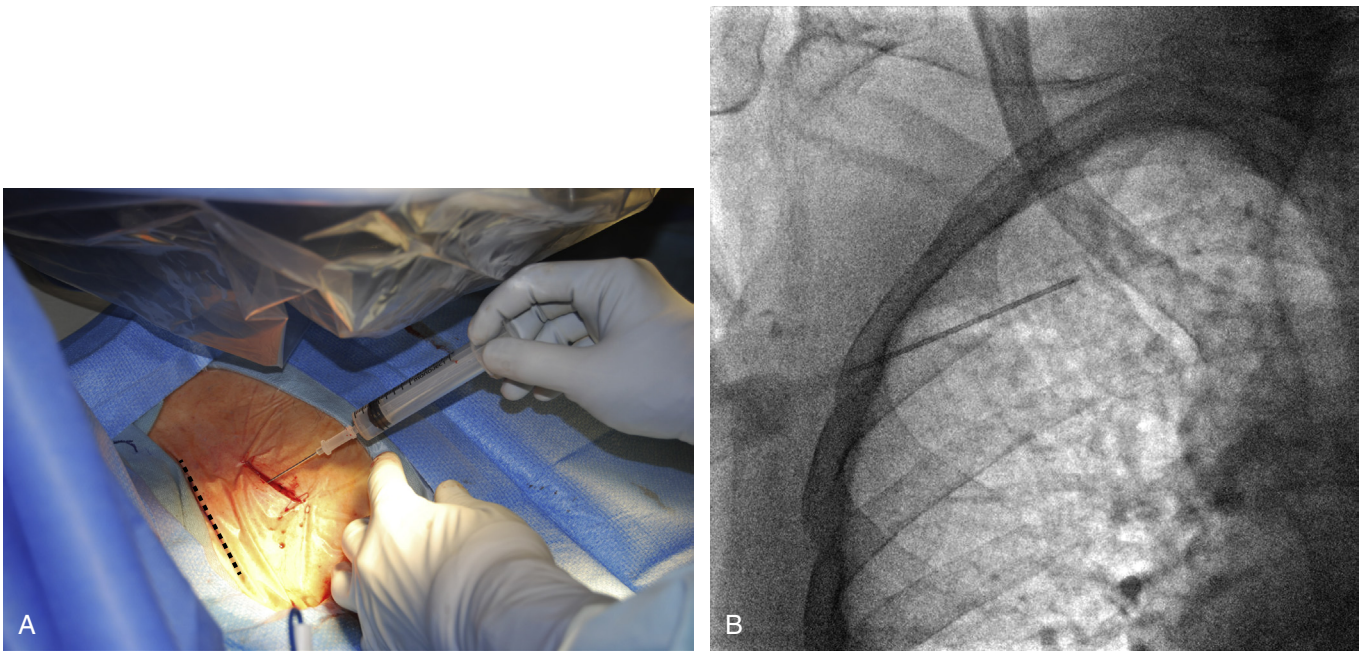
Peripheral venography can be very helpful in identifying the venous anatomy and can be used to aid in cannulation of this vein, if access cannot be easily obtained with fluoroscopic landmarks alone. Many implanting physicians now routinely use venography for venous access. Some implanting physicians prefer to use a 5-Fr micropuncture introducer system for contrast-guided venous puncture of the axillary vein; this is safe and effective using a medial or lateral approach to the axillary vein.[15] Many implanting physicians prefer a modified Seldinger technique with a larger-bore needle over concerns that the 5-Fr micropuncture needle is more prone to bending and a stable direction of access can be more difficult.

Review of peripheral contrast venograms performed to identify radiographic location and facilitate venous access often illustrates some anatomic variations in axillary vein location, particularly in the cranial-caudal dimension.[16] The most common radiographic position of the axillary vein was found to be over the anterior third rib. Whites had a more caudal axillary vein position, whereas men and patients with higher body mass index (BMI) had a more cranial position of the axillary vein in this study.

Tip: The implanter should use one approach predominantly and consider a stepwise methodologic technique so that each step is performed with care and an understanding of the nuances of risk and benefit of that technique. The axillary vein approach is preferred by many implanters and is highly successful, but the implanter must understand the anatomic markers and the risk of this approach. If venous access is not successful with this approach, the implanter must have mastered at least one other approach.



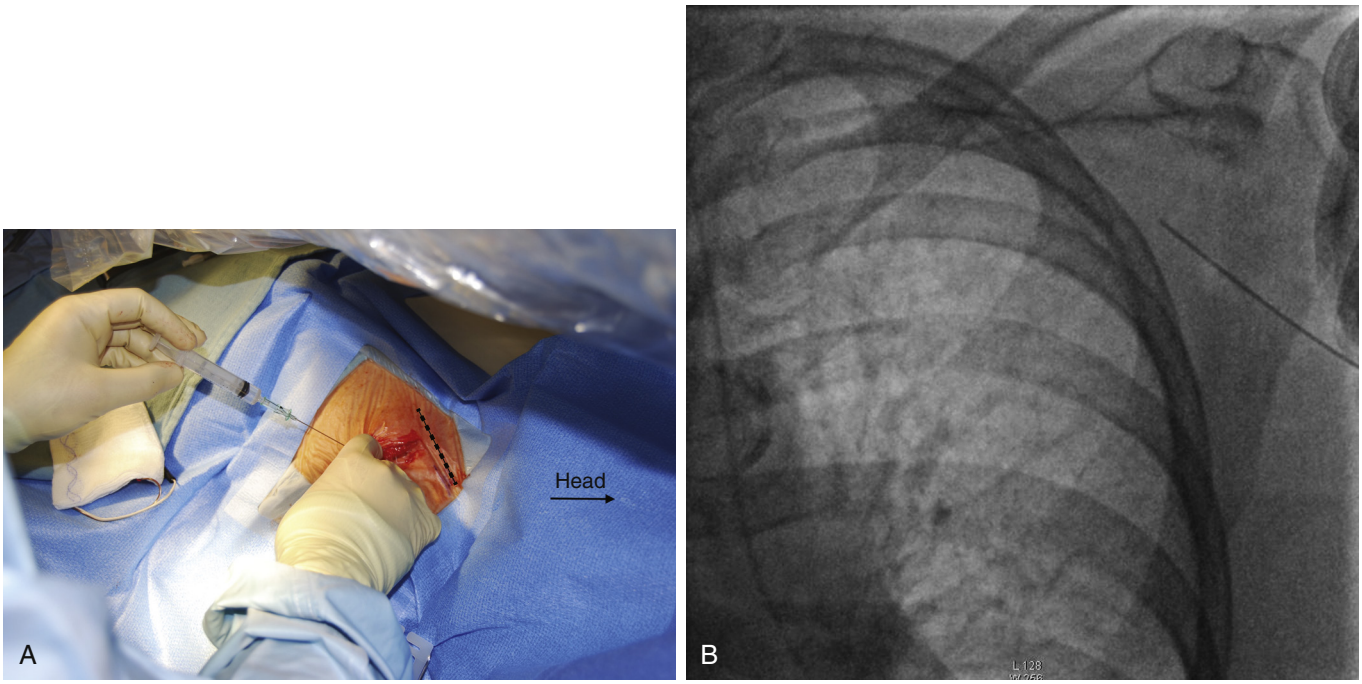
• **Fig. 8.14** Cephalic vein cut-down. (A) The cephalic vein lies in the deltopectoral groove *(dashed line)*, which can be palpated before creating an incision. (B) A schematic illustrating the relationship of the deltoid and pectoralis muscles.



• **Fig. 8.15** Right-sided cardiac implantable electronic device implant with subclavicular incision. The *dotted line* denotes the clavicle; the head of the patient is to the left. (A) The appropriate angle at which to hold the 18-gauge needle when performing an axillary vein approach targeting the fluoroscopic anterior first rib shadow. (B) A fluoroscopic image of the same venous access. The appropriate angle at which to hold the needle relative to the chest wall is between 30 and 45 degrees.

Cephalic Vein Approach

Cephalic vein cut-downs eliminate many risks associated with subclavian venous puncture, particularly the most common acute complication of iatrogenic pneumothorax. Anatomic landmarks are utilized to identify the cephalic vein, which lies within the deltopectoral groove. The deltopectoral groove is formed by the lateral aspect of the pectoralis major muscle and the medial border of the anterior deltoid muscle (Fig. 8.17A–C).[10] These landmarks can be palpated prior to creating an incision, which will help delineate this region and subsequently allow direct visualization of this vein during the surgical procedure. An incision is often created parallel to the deltopectoral



• **Fig. 8.16** Left-sided cardiac implantable electronic device implant with deltopectoral groove incision. The *dotted line* denotes the clavicle, and the head of the patient is to the right. (A) The appropriate angle at which to hold the 18-gauge needle when performing an axillary vein approach targeting the fluoroscopic posterior second rib shadow. (B) A fluoroscopic image of the same venous access approach. The appropriate angle at which to hold the needle relative to the chest wall is between 60 and 90 degrees.

groove (dashed line). Venography is not required to access this vein. Fig. 8.1 also illustrates the course of the cephalic vein.

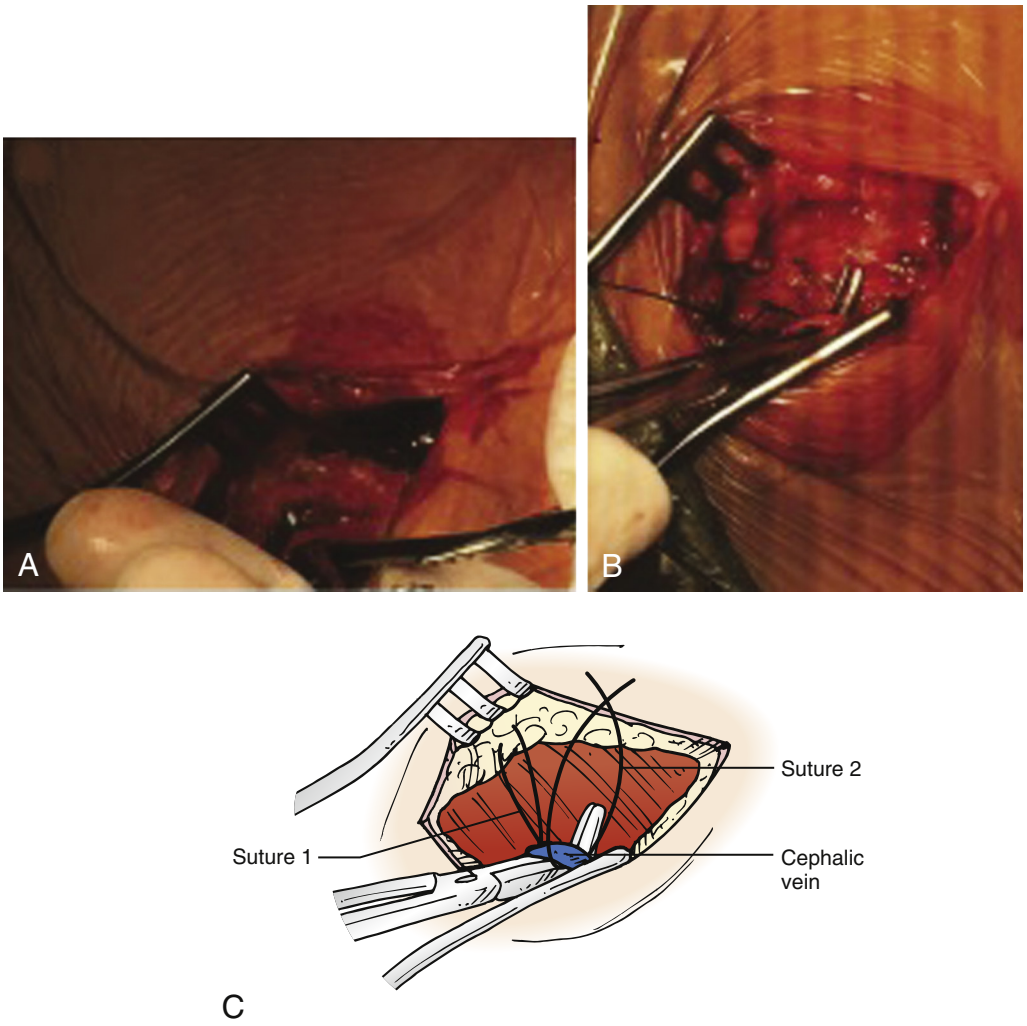
Fig. 8.17A illustrates the location and appearance of the cephalic vein in the deltopectoral groove. The vein is often hidden beneath the deltopectoral groove fat pad. The fat pad can be recognized by its pale color compared with subcutaneous fat, which tends to be a darker yellow. In some patients, this vein is deeper and more time may be required for dissection (Fig. 8.17B). After the vein is isolated, two nonabsorbable ties are typically placed around the cephalic vein (Fig. 8.17B–C). The more distal suture is often tied off for hemostasis, and a hemostat is placed on the suture. A second hemostat is placed on the more proximal suture, which is not tied. A No. 11 blade is used to nick the upper third of the vein to allow direct introduction of the lead into the vessel. The lead is then passed into the heart with fluoroscopic guidance.

If only one lead is required, the suture sleeve can be passed just into the cephalic vein and a tie placed over the vein and suture sleeve to obtain hemostasis. The lead can then be secured to the pectoralis muscle using nonabsorbable suture and the lead sleeve. Care must be taken to avoid securing the lead to the anterior deltoid muscle, which would result in patient pain and limit motion of the upper extremity. If a second lead is required and the vein is large enough to accommodate this, the lead can be inserted directly into the vein before the first lead is secured.

An alternative approach to placing lead(s) into the cephalic vein uses an introducer sheath to stabilize the vessel. This can be performed with an 18-gauge needle to access the cephalic vein under direct visual guidance and then advancing a guidewire, followed by advancing the sheath over the wires. Others may place a 16-gauge Angiocath into the vessel, followed by the guidewire and then the sheath (Fig. 8.18A–C).

If the vein appears too small to allow access of two leads, a separate subclavian or axillary vein puncture could be performed for the second lead. Rarely, the cephalic vein may take a supraclavicular course to join the jugular vein, making access difficult.[17] Two or more leads may be successfully inserted through the cephalic vein for cardiac resynchronization device implantation.[18] Failure to place leads using a cephalic vein cutdown approach may be related to failure to isolate the vein, a small cephalic vein, venous stenosis, venous tortuosity, or other anomalies.[19,20]

Studies have shown that the cephalic vein approach is preferable because of reduced acute and long-term complications. Because venipuncture is not required, the risk of pneumothorax is eliminated with direct visualization of the cephalic vein. Risk of lead failure, including fracture or insulation breaks, appears to be lower with the cephalic approach.[13,21] This may be due to a lower risk of subclavian crush because the lead does not pass *through* the subclavius muscle or costoclavicular ligament. Another advantage is that the presence of valves in the vein prevents introduction of air into the venous system (see Fig. 8.8). This vein can typically accommodate at least one lead, and two leads often can be inserted either under direct visualization of the vein or by adding an introducer sheath. However, if a guidewire and introducer sheath are utilized, care must be taken to prevent air embolism, as with subclavian venipuncture. If there is damage to the vein and bleeding occurs, the vein can be tied off for hemostasis, and subclavian or axillary



• **Fig. 8.17** Cephalic vein cut-down. (A) An example of a more superficial vein; it lies within the deltopectoral groove. (B) A deeper cephalic vein requiring more extensive dissection. A nonabsorbable suture is placed underneath this vein. The more distal suture is subsequently tied for hemostasis. A second suture (not shown) is placed more proximally and later used to secure the lead with the suture sleeve, also providing hemostasis. (C) A schematic of a cephalic vein cut-down approach.

venipuncture can then be performed. If the vessel is transected and the proximal portion retracts below the pectoralis major and does not stop with digital compression, a surgical consult may be required.

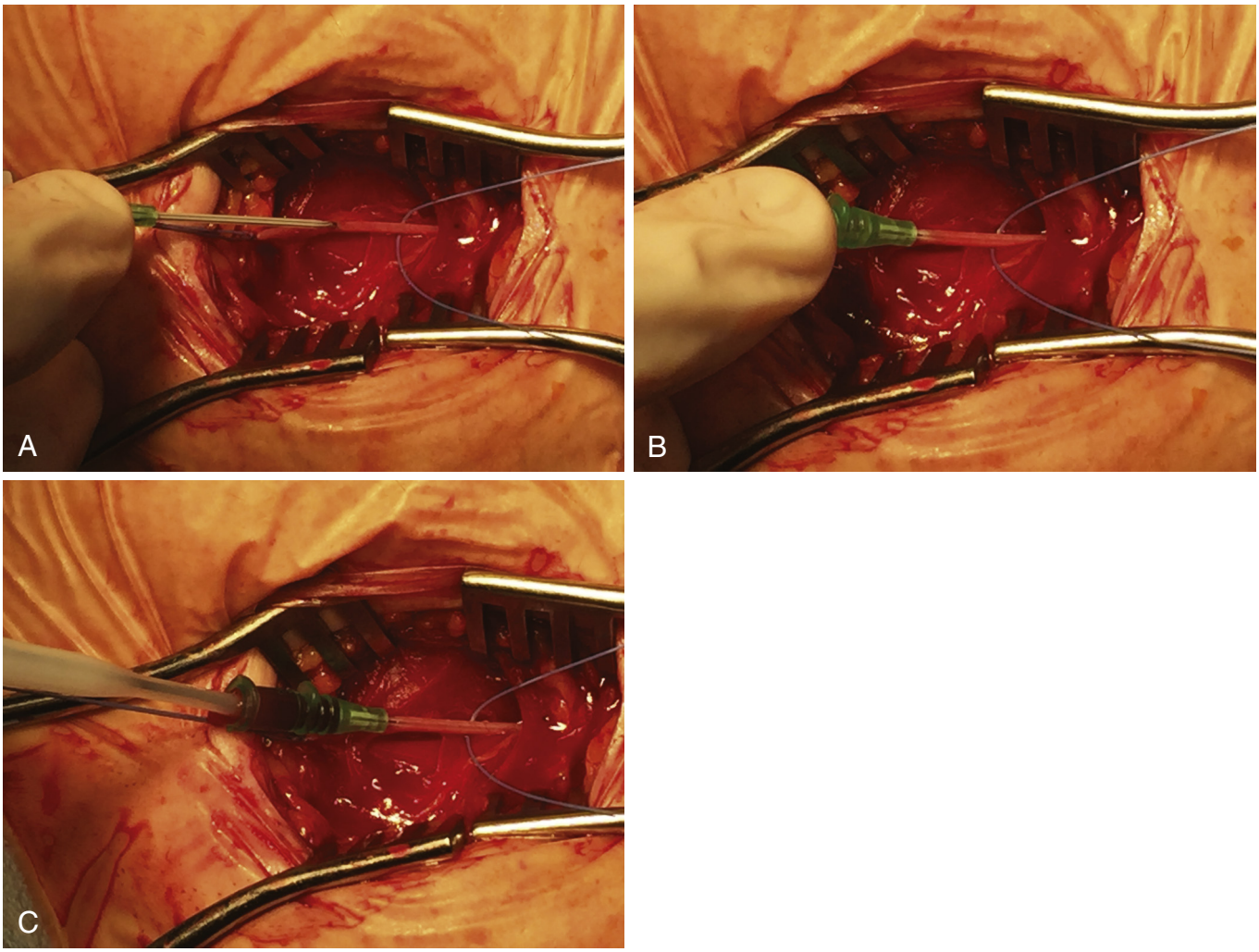
Tip: The cephalic vein approach has advantages over the subclavian approach because the risk of pneumothorax is low, but the implanter must become proficient in using this approach if it is to be performed routinely and successfully.

Venous Anomaly

The most commonly encountered thoracic venous anomaly is the presence of a persistent left superior vena cava (SVC). A left-sided SVC is reported to occur in 0.3% of the population.[22] The left SVC typically drains into the right atrium through the coronary sinus, which is dilated. Leads can still be passed into the right atrium and right ventricle via this route (Fig. 8.19A–C). Alternatively, right-sided access can be utilized to gain access to the heart via the right SVC. However, in extremely rare cases, a persistent left SVC may be present with an absent right SVC.[23]

Venous Obstruction

Venous obstruction occurs relatively frequently after ICD implantation,[24,25] with various degrees of obstruction found in 25% of patients undergoing elective generator replacement.[25] Complete occlusion may be found in 9% of cases with more proximal patency. Brachiocephalic (innominate) vein access has been described in the presence of axillary and subclavian venous occlusion when there is restoration of more medial patency.[24] Following venography, the needle is initially advanced under fluoroscopic guidance into a lateral infraclavicular location and then advanced under the clavicle in a horizontal plane toward the sternal notch to enter the ipsilateral brachiocephalic vein medial to the



• **Fig. 8.18** An alternative approach to cephalic vein access. A needle can be used to enter the cephalic vein under direct visualization (A). The 16-gauge Angiocath can be inserted into the vein (B), followed by a guidewire, which will then be exchanged for an introducer sheath large enough to allow passage of the lead (C).

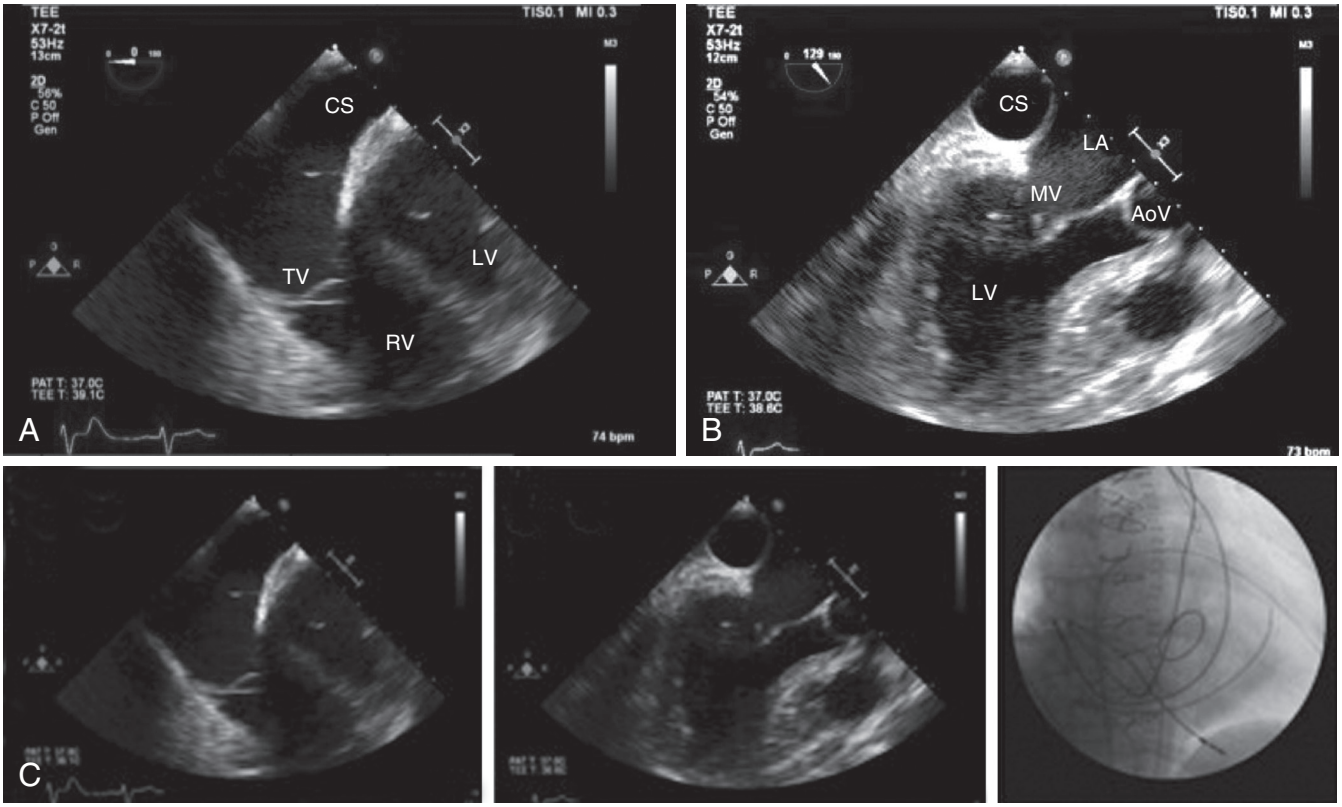
occlusion.[24] Risks of such an approach include lead placement through the subclavius muscle or costoclavicular ligament, a higher risk of pneumothorax, and inadvertent entry into the subclavian artery, carotid artery, or thoracic duct.

Patients who appear to have complete or nearly complete obstruction of the subclavian/axillary venous system may be candidates for venoplasty. Often a small channel can be crossed with appropriate hydrophilic wires; venoplasty can then be performed, allowing access to the vessel (Fig. 8.20A–C, Video 8.1).

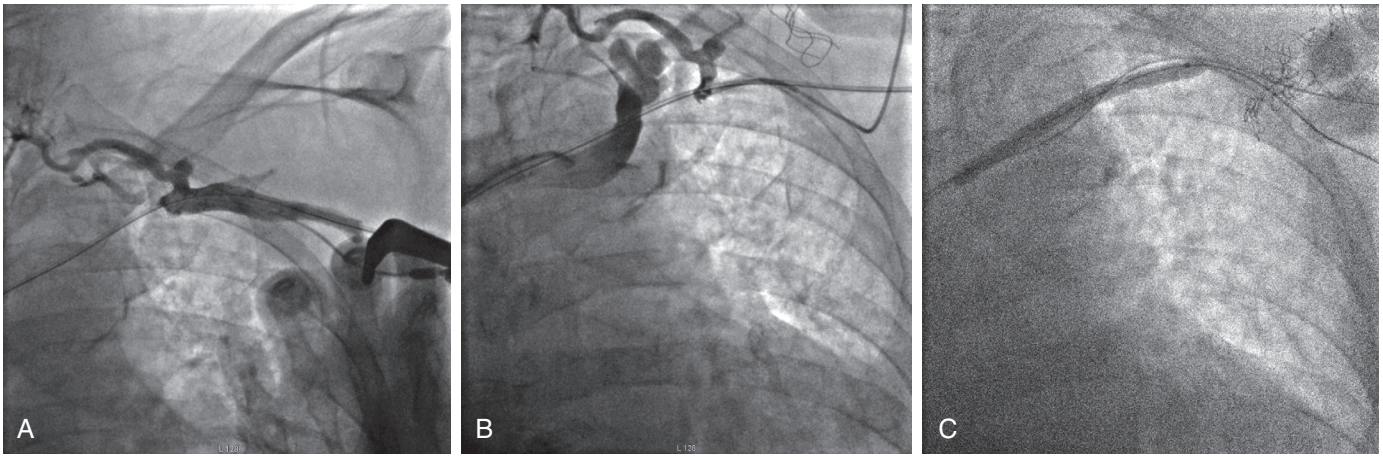
Venous Access During Lead Revision

If a lead revision procedure is performed to address lead malfunction (e.g., fracture, insulation breach) or stability (e.g., repeated dislodgement), a new venipuncture can be avoided using a "wire under insulation" technique. This approach can be used to maintain or access the venous system using the previously implanted lead. If the lead has been recently placed or is freely mobile (as demonstrated by the ability to advance the lead freely, documenting the absence of adhesions), the distal portion of the lead can be withdrawn to the cavoatrial junction. The insulation of the lead is incised with a No. 11 scalpel blade, and a guidewire is inserted between the insulation and conductor.[26] The lead is advanced back into the venous system until the location of the guidewire within the insulation is intravascular. The guidewire is then freed from the insulation and advanced into the superior vena cava. The lead to be removed is then withdrawn from the vein while the guidewire remains within the venous system. A new peel-away sheath can then be advanced over the guidewire.

This technique can also be used in patients with previously implanted transvenous leads and venous obstruction that prevents conventional access.[27,28] In this situation, if the chronically implanted lead cannot be easily withdrawn into the SVC, then a modification of the described procedure can be attempted. The lead may be pulled back a few centimeters, enough to place a nick in the insulation as medial as possible such that advancing the lead carries the guidewire intravascularly.



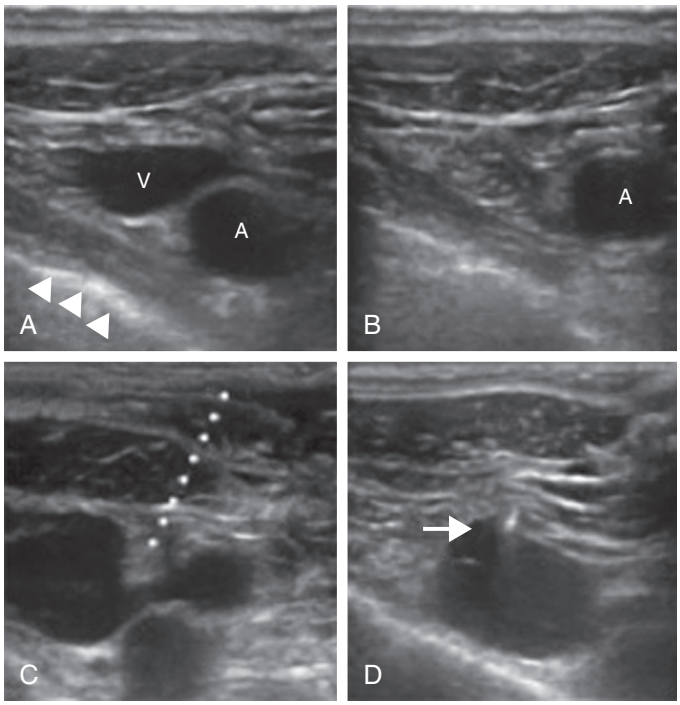
• **Fig. 8.19** Persistent left superior vena cava. (A–B) The appearance of a large coronary sinus on echocardiography. In this case, echocardiography was available before pacemaker insertion. The implanting physician elected to approach this case from the left side because the patient was right handed. (C) The course of the right ventricular active fixation lead from the left subclavian vein through the coronary sinus into the right atrium, and then across the tricuspid valve to the right ventricular apex. *AoV*, Aortic valve; *CS*, coronary sinus; *LA*, left atrium; *LV*, left ventricle; *MV*, mitral valve; *TV*, tricuspid valve.



• **Fig. 8.20** Fluoroscopic images from a patient scheduled for addition of a lead for an upgrade to a dual-chamber implantable cardioverter-defibrillator (ICD) from a single-chamber ICD. (A) Venogram performed before venous access, demonstrating apparent complete obstruction of the left subclavian vein. (B) Simultaneous contrast injections from both sides of the obstruction (the left brachiocephalic was accessed from the right femoral vein). (C) Venoplasty performed, which successfully allowed placement of the transvenous lead.

Techniques to Minimize Complications

As previously discussed, acute complications of venous puncture include pneumothorax, hemopneumothorax, brachial plexus injury, and air embolism; longer-term complications include lead fracture, insulation break, and "subclavian crush." In a large cohort study, patient factors associated with increased risk of pneumothorax in cardiac pacing included female sex, age greater than 80 years, and history of chronic obstructive pulmonary disease (COPD).[29] Young active patients are more prone to longer-term lead complications. The frequency of



• **Fig. 8.21** Axial approach for ultrasound-guided access. (A) Ultrasound image of a right subclavian vein *(V)* and artery *(A)*, showing proximity to chest cavity *(arrowheads)*. (B) Ultrasound image under compression showing collapse of the vein. (C) Indentation of the vein with the needle tip confirms its location. The angle of the needle *(dotted line)* is directed away from the artery to the extent possible. (D) A 0.035-inch guidewire *(arrow)* is inserted and can be followed medially to confirm venous location.

complications also appears to be related to choice of venous access and operator experience.

Although randomized studies are not available, an axillary vein puncture approach may help to reduce acute complications and long-term lead failure compared with the subclavian approach.[30] When subclavian access is obtained, the leads may become entrapped between the clavicle and the first rib.[4,13] Subclavian crush syndrome is thought to result from lead entrapment between the costoclavicular ligament and/or the subclavius muscle, which is a muscular-tendinous structure (see Chapter 2). This occurs when the subclavian vein access is medial such that the lead courses through this structure. The placement of multiple leads appears to increase this risk. To reduce the risk of crush injury, other venous access techniques, including cephalic vein cut-down,[13,31] or a more lateral approach to the subclavian/axillary veins is preferred.

In addition to use of anatomic landmarks, fluoroscopy, and contrast venography as already described, ultrasound guidance can identify the course of the vein. Doppler flow to identify the axillary vein for extrathoracic pacemaker lead placement was described more than 20 years ago.[32] Ultrasound-guided access offers an opportunity to reduce the complication risks of vascular access for device implantation. This involves using a vascular probe ranging from 5 to 13 MHz depending on the manufacturer. Most advocates of this technique obtain venous access first and subsequently create the device pocket 1 to 2 cm caudal to the site of access. This approach requires retrieval of the wire(s) from within the pocket to proceed with sheath insertion. Either axial (cross-sectional) (Fig. 8.21A–D) or longitudinal (parallel with the axis of the target vessel) (Fig. 8.22A–D) ultrasound guidance can be used depending on operator comfort and whether surface anatomy allows for proper orientation of the probe (a longitudinal approach may require more room). There have not been any randomized trial data investigating ultrasound guidance, but case series reports have reported a reduced risk of pneumothorax or inadvertent artery puncture and less fluoroscopy exposure time.[33] Preprocedure imaging of the proximal cephalic vein in the infraclavicular region has also been described.[34]

Tip: Despite the apparent simplicity of lead implantation, complications and adverse effects can occur at every step of the way; to prevent them, the clinician must exercise extreme vigilance and adhere to careful technique as well as develop a sensitivity toward issues that can create short- and long-term harm and risk to patients, especially if shortcuts are used.

The implanting clinician should learn to master at least one or two specific techniques to implant leads and place them appropriately but must be aware of the risks and benefits of the technique and know when to use an alternative approach.

Lead Positioning

Right Ventricular Lead Placement

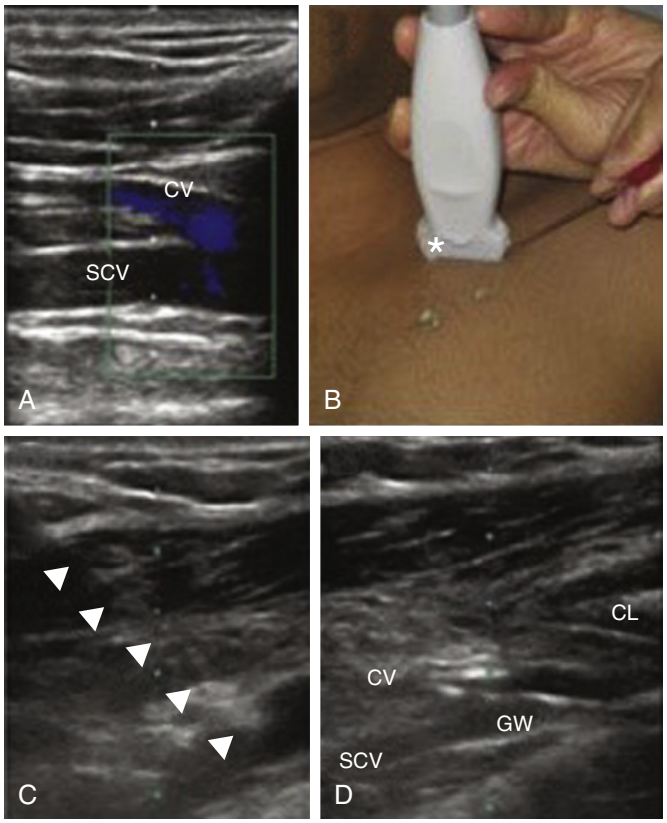
In dual-chamber systems, the right ventricular (RV) lead is typically placed first to provide backup pacing as needed throughout the procedure. After venous access has been obtained, a guidewire is positioned in the SVC and appropriately sized sheaths used as

described earlier. The traditional RV lead location is the RV apex, which generally enables a stable and easily accessible location. This location is achieved by curving a stylet to cross the tricuspid valve and reaching the right ventricular outflow tract (RVOT). The curved stylet is then replaced by a softer, straight stylet and prolapsed down to the RV apex. Often the lead drops down toward the apex as the curved stylet is removed and/or the straight stylet is being advanced. Care must be taken to avoid prolapse of the lead back into the atrium or inferior vena cava during the stylet exchange, which can be exacerbated in the presence of significant tricuspid regurgitation (Fig. 8.23A–C, Video 8.2). Reaching the RVOT confirms a true RV location of the lead rather than a possible coronary sinus (CS) location. Some physicians have incorporated a method to advance the lead from the atrium into the RVOT by prolapsing the lead across the tricuspid valve and then dropping the lead from the RVOT into the RV apex. This approach must be used cautiously in patients with a left bundle branch block because it can traumatize the right bundle, resulting in complete heart block. This approach may also be more prone to cause ventricular ectopy, although, as with any method, when used in experienced hands it can be performed safely.

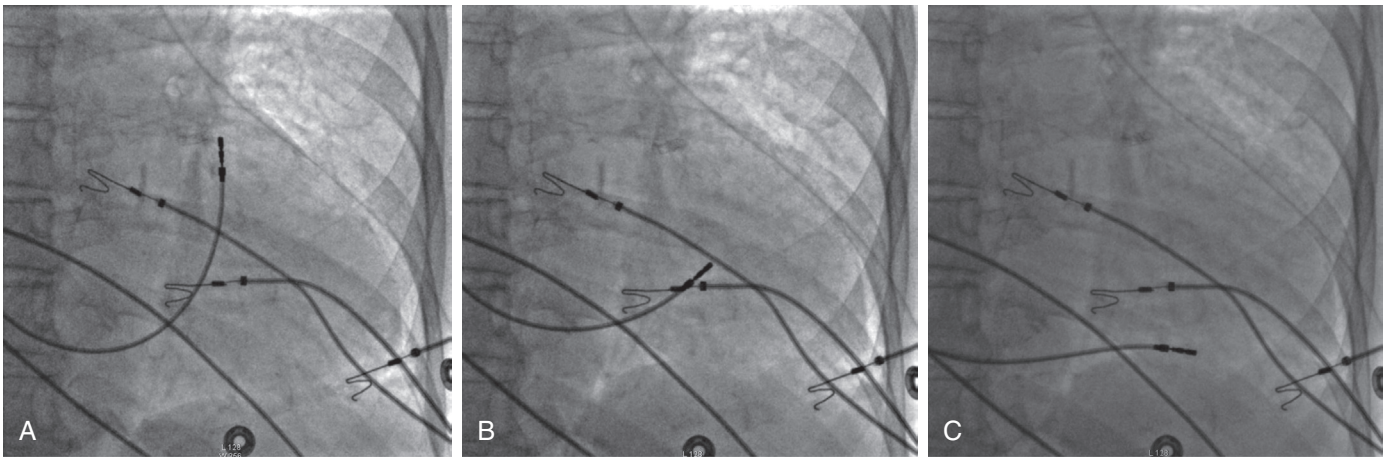
Prolapse can be useful when encountering a severely enlarged right atrium, significant tricuspid regurgitation, or elevated pulmonary artery pressure. Caution must be used to ensure that the lead does not become engaged in the tricuspid chordae with any of the methods. The lead tip should always be observed to freely move before advancing.

Once the lead is in the apical region, it can be slightly advanced with a soft tip (i.e., withdrawing the stylet slightly back from the tip of the lead) as needed to the desired final location. R-wave amplitude may be checked before extending the helix to confirm adequate myocardial contact. The active fixation mechanism should be deployed under fluoroscopic magnification, after which the stylet is withdrawn several centimeters and the lead is given adequate redundancy across the tricuspid annulus and into the low right atrium.

Pacing parameters can now be obtained. A so-called injury current can often be observed on the pacing system analyzer (PSA), suggesting good myocardial contact and the likelihood of improved pacing parameters after the initial "injury" (Fig. 8.24). Maximum output pacing at 10 V should be done to document the absence of phrenic nerve or diaphragmatic stimulation.



• **Fig. 8.22** Longitudinal approach for ultrasound-guided access. (A) Longitudinal image of the subclavian vein *(SCV)* and cephalic vein *(CV)*. The artery is not visible but is immediately adjacent to the vein. (B) Demonstration of needle entry position in longitudinal approach. The ultrasound probe is placed over the angle of the clavicle *(asterisk)*. (C) The needle *(arrowheads)* must be visible from the skin to the cannulation point in the vein to ensure that it is in the same plane as the ultrasound. (D) The guidewire *(GW)* entry point is seen to be 2 cm away from the chronic lead *(CL)*.

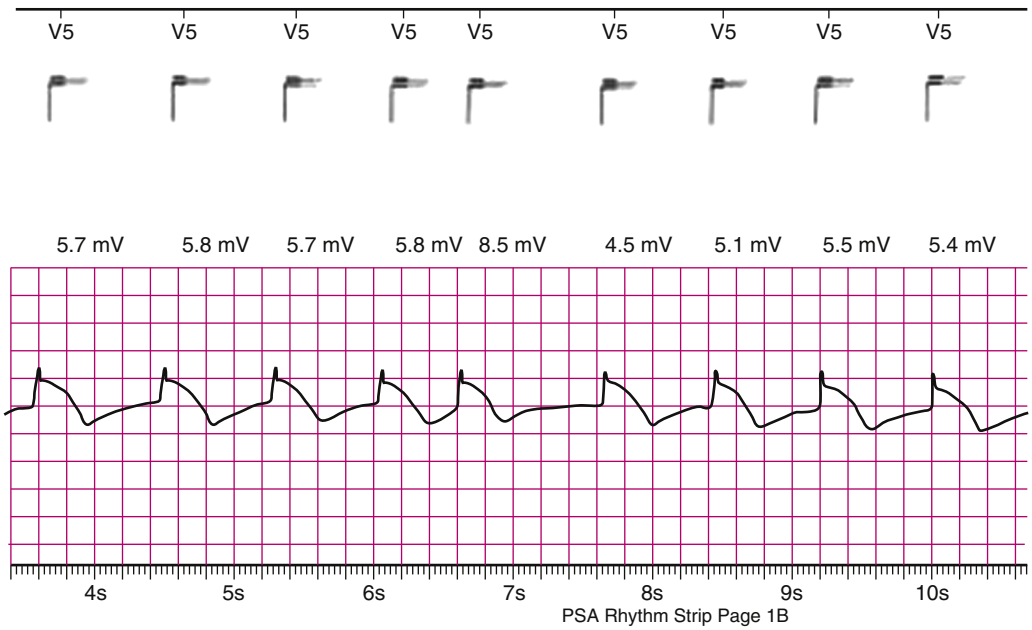


• **Fig. 8.23** (A) Fluoroscopic image showing a right ventricular (RV) pacing lead advanced into the RV outflow tract. (B) Fluoroscopic image showing moving the RV lead down the septum toward the RV apex, following exchange of a curved stylet for a straight stylet. (C) The RV lead has been advanced to the RV apex.

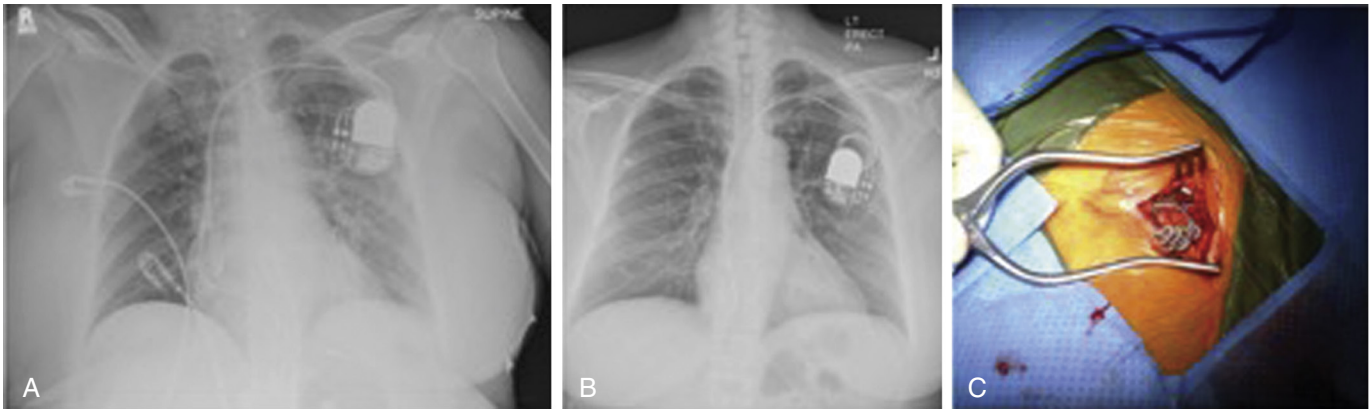
The lead can now be secured to the pectoral fascia using 0 nonabsorbable suture around the anchoring sleeve. Some implanting physicians leave the stylet in the lead while placing the sutures around the suture sleeve, considering this approach to minimize risk of lead injury from overtightening the sutures. Others will either partially or completely remove the stylet first before tying down the lead to visualize the final redundancy of the lead without the stylet. At least two sutures should be placed around each lead sleeve to maximize the stability of the lead. A gentle tug of the lead after sutures are in place should confirm a stable, nonmovable lead.

It is important to secure all leads to the underlying fascia or muscle using the suture sleeve. However, even with the use of a nonabsorbable suture, this does not necessarily prevent dislodgement resulting from positional changes caused by large breasts or pectoral muscle motion. This situation can present as "twiddler's syndrome" (Fig. 8.25A–C) when the lead progressively retracts and coils in the pocket accompanied by generator rotation.

The procedure for placement of a passive fixation lead (tined) is similar to that for an active fixation lead. The lead is initially advanced into the RVOT and then, with the stylet exchange technique, positioned into the RV apex. With the stylet withdrawn slightly from the tip, a slight forward advancement (1 to 2 mm) can help to stabilize the lead tip in position. An injury current will often be observed on the intracardiac electrogram on the analyzer, from direct contact with the lead tip to endocardium, when in proper position.



• **Fig. 8.24** Tracing of a large injury current recorded on the electrocardiogram directly after extending the helix of the right ventricular lead.



• **Fig. 8.25** Twiddler's syndrome. (A) Chest radiograph taken immediately postoperatively. The atrial and ventricular leads are in good position in the right atrial appendage and at the right ventricular apex. (B) Lead position at 3-month follow-up. The atrial lead is now pulled back to the superior vena cava/high right atrium junction, and the ventricular lead is pulled back to the right atrium. (C) Findings at the time of reoperation in a different patient. Marked twisting of the lead was noted, consistent with twiddler's syndrome.

Right Ventricular Apical Versus Right Ventricular Septal Location

The RV apical lead location has recently come into question owing to evidence of an association between traditional RV apical pacing and heart failure, atrial fibrillation, and increased mortality.[35,36] RV pacing may induce a dyssynchronous electrical activation leading to left ventricular (LV) remodeling.[37] RV septal pacing has therefore been brought up as an alternative to RV apical pacing, with the hypothesis that by pacing closer to the His-Purkinje system, a more physiologic LV activation may occur, thus improving patient outcomes. Multiple studies have compared RV apical to RV septal pacing with conflicting results. Acute hemodynamics, echocardiographic changes, and long-term LV systolic function[38] have been assessed with no clear benefit supporting a consensus practice change toward RV septal pacing. The lack of demonstrated benefit may result in part from a variable RV septal pacing location where a significant number of RV septal leads may in fact have been placed on the anterior wall of the right ventricle rather than a true septal location. Whereas fluoroscopy is useful to identify the RV apex, finding the true septum may not be as easy. In a recent study using conventional fluoroscopic criteria, only a minority of presumed RV septal leads were implanted on the true RV septum,[38] and these authors suggest additional criteria for RV septal pacing including aiming for the middle of the cardiac silhouette in the right anterior oblique (RAO) fluoroscopic view, confirming rightward orientation in the left anterior oblique (LAO) view, and having a paced QRS duration of less than 140 ms.

The impact of the right ventricular lead location on clinical outcome and the incidence of ventricular tachyarrhythmias in patients with a cardiac resynchronization therapy-defibrillator (CRT-D) system was studied in the Multicenter Automatic Defibrillator Implantation Trial with Cardiac Resynchronization Therapy (MADIT-CRT) patient population.[39] This study did not demonstrate any benefit in clinical outcome or echocardiographic response for the nonapical location. A nonapical RV lead location was associated with an increased risk of ventricular tachyarrhythmias. Most recently, the Comparison of Right Ventricular Septal and Right Ventricular Apical Pacing in Patients Receiving a CRT-D Device (SEPTAL CRT) study randomized 263 patients undergoing CRT to a septal versus apical RV lead location,[40] with no difference shown between the two groups in clinical outcome. Overall, large prospective studies are needed to confirm or deny the benefit of RV septal pacing over traditional RV apical pacing (Fig. 8.26A–D).

Tip: Although there may be a specific advantage of septal positioning, the data are not strongly in favor of one approach or another. The risk of a septal placement includes a possibly higher rate of dislodgement in inexperienced hands.

Right Ventricular Lead Selection

Consideration of lead type and length should be made before beginning the procedure. There are varying lengths of leads available, and using a lead long enough to provide adequate redundancy is important. This is particularly important in the pediatric patient population, in whom a lack of redundancy can cause pacing problems at a later stage (Fig. 8.27A–B). Generally, patients taller than 6 feet should receive a longer lead. Large dilated hearts and barrel-chested anatomy should also be taken into consideration. Fixation mechanisms vary as well. Active fixation leads are most commonly used today, and there is little need to consider a passive lead in the right ventricle.

In the case of ICD leads, the use of a single- versus dual-coil lead should be considered. Traditionally, dual-coil ICD leads have been used more frequently; however, recent concerns regarding ease of extraction and lack of documentation of clear benefit has led to a change in practice patterns and increasing use of single-coil ICD leads.[41]

A recent study of 129,520 ICD recipients enrolled in the LATITUDE remote monitoring program evaluated trends in single- versus dual-coil ICD lead implantation and differences in clinical outcomes. This study demonstrated a significant increase in single-coil use between 2004 and 2014. Single-coil lead implantation was associated with more frequent defibrillation testing and the lead being taken out of service but was not associated with increased mortality or more frequent defibrillation failure.[42]

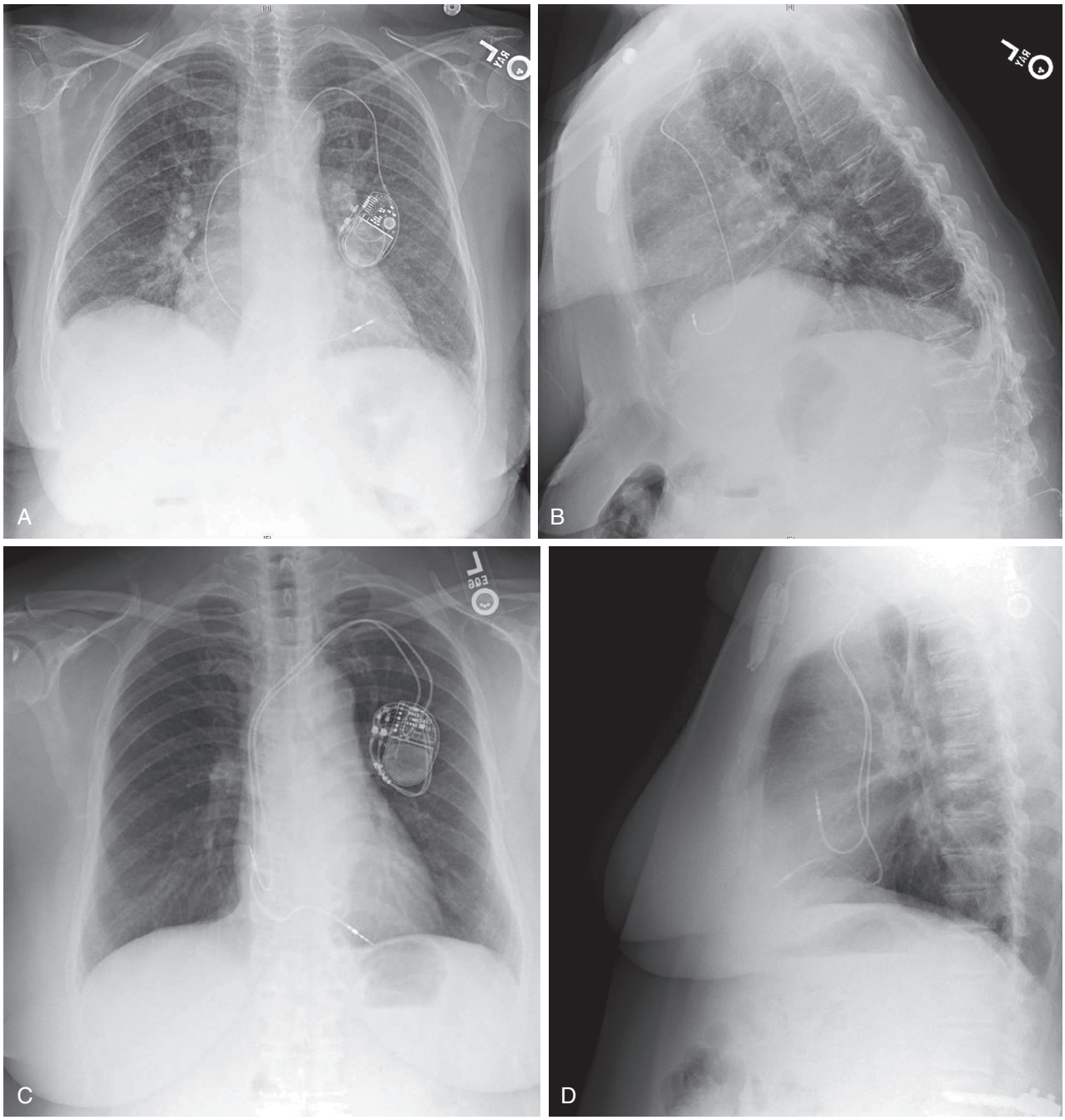
When the Right Ventricular Lead Is Not in the Right Ventricle

A rare complication of lead placement is that the RV lead is inadvertently placed in the left ventricle. This can occur through crossing an atrial septal defect (ASD) or ventricular septal defect (VSD) or directly accessing the subclavian artery (Fig. 8.28A–G). In the case of LV pacing, the paced QRS complex should demonstrate a right bundle branch block (RBBB) pattern as opposed to the typical left bundle branch block (LBBB) seen with correct RV lead placement. Fluoroscopy is also helpful when assessing lead positioning. A more superior than normal course of the lead into the ventricle should raise suspicion of crossing an ASD rather than the tricuspid valve. In the case of subclavian artery access, the guidewire will traverse to the anatomic left of the patient's spine and will not continue into the inferior vena cava.

Tip: It is important to check lead V1 to make sure there is not a manifest RBBB during pacing to avoid these complications.

Right Ventricular Lead Perforation

Lead perforation of the RV is a rare but potentially life-threatening complication that may occur during, shortly after, or late after implantation (Fig. 8.29A–B).[30] An acute perforation may be associated with hemodynamic compromise due to tamponade from a pericardial effusion; however, a delayed presentation is more typical. Lead characteristics may play a role in the risk

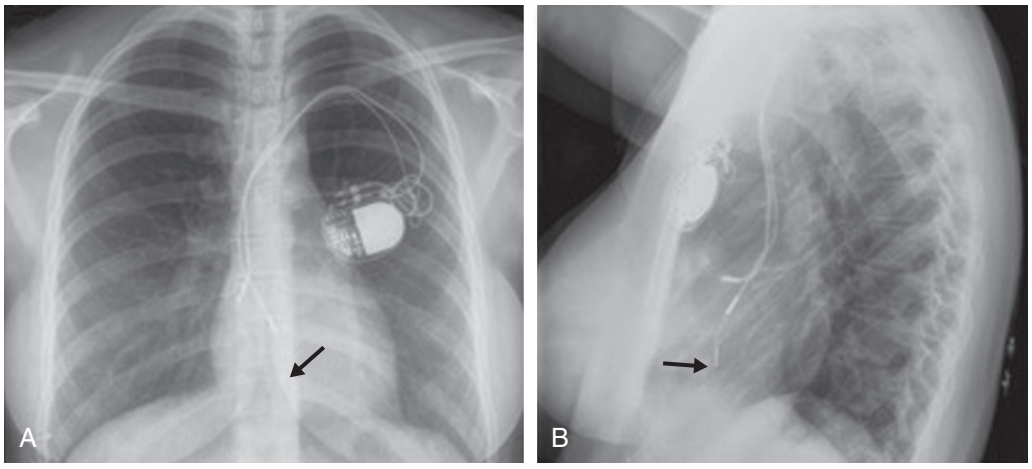


• **Fig. 8.26** Posteroanterior (A) and lateral (B) chest radiographs demonstrating a dual-chamber pacemaker with the right ventricular (RV) lead placement into a septal location. (C–D) A dual-chamber pacemaker with the RV lead placement into the RV apex.

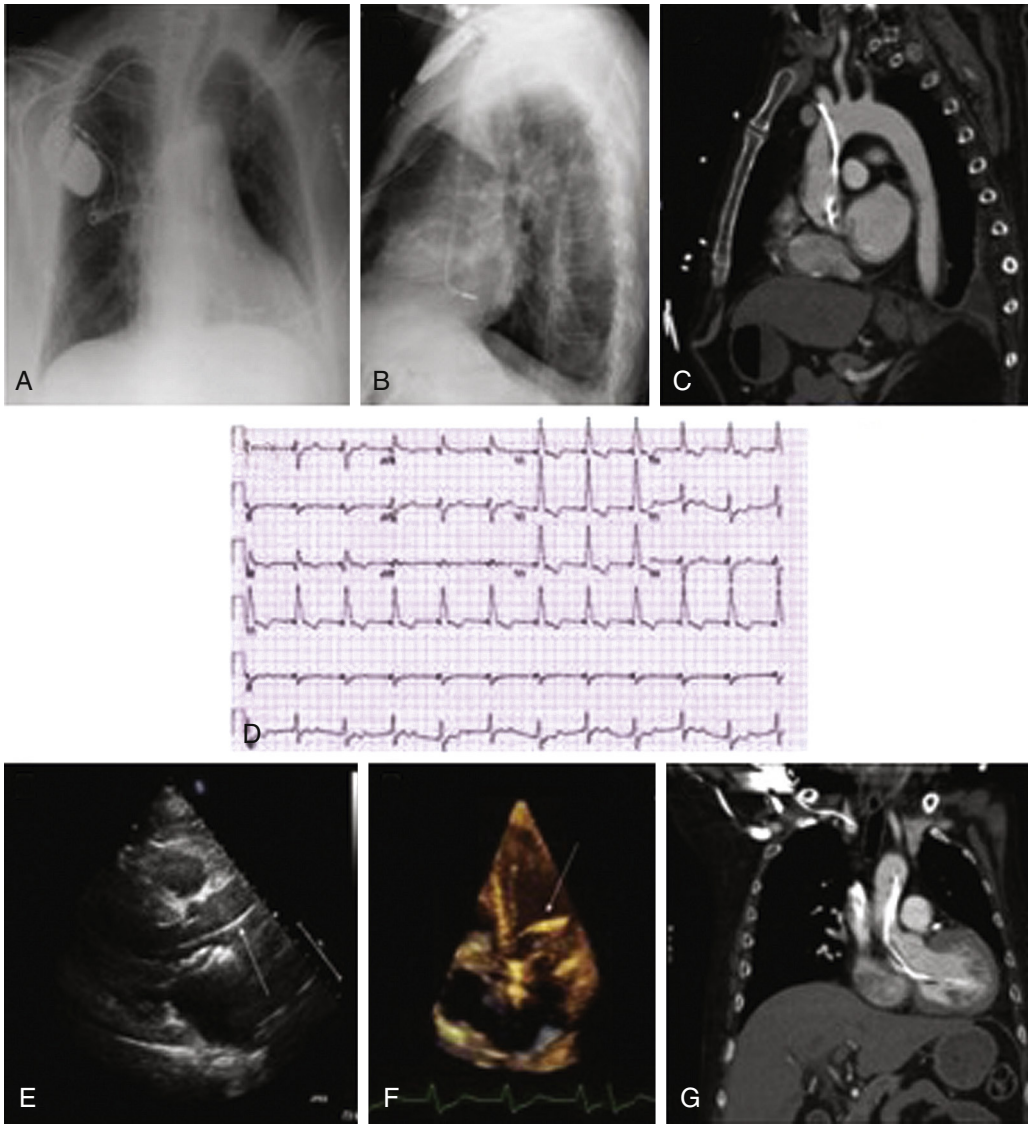
of perforation, with a higher rate of perforation seen in ICD leads compared with pacemaker leads. Active fixation leads have been considered more prone to perforation, although this has not been substantiated in more recent studies.[30] At the time of lead implantation, it is important not to overtorque the active fixation mechanism, which may increase the risk of perforation (Video 8.3). It is also helpful to check for diaphragmatic pacing, which may occur with perforation of the lead. Using multiple fluoroscopic views to confirm final lead location also helps establish a stable and secure final lead location.

Atrial Lead Placement

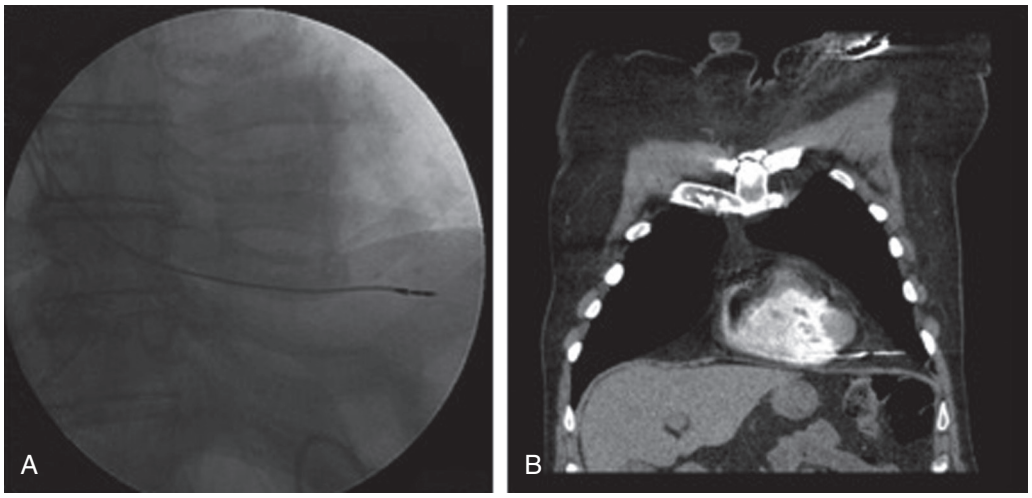
The right atrial appendage is generally the preferred location for the atrial lead. With the available retained guidewire, a new pacemaker introducer sheath is advanced over the wire into the SVC. In a similar fashion to the RV lead, the dilator is removed, the guidewire is left in place, and the atrial lead is positioned under fluoroscopic guidance. A straight stylet is used to reach the right atrium (RA) but then replaced with a preshaped "J" stylet, allowing for placement into the RA appendage. The



• **Fig. 8.27** Posteroanterior (A) and lateral (B) chest radiographs showing transvenous leads placed at age 4 years in a now 21-year-old woman. The *arrows* show the tip of the right ventricular lead pulled back to the tricuspid valve with no remaining redundancy.



• **Fig. 8.28** Right-sided pacemaker with right ventricular lead placed through the right subclavian artery as demonstrated on chest radiograph (A–B), chest computed tomography with lead in the aorta (C, G), and echocardiography (E–F). (D) A 12-lead electrocardiogram shows right bundle branch block consistent with left ventricular pacing.



• **Fig. 8.29** Cine image (A) and chest computed tomography (B) demonstrating a right ventricular lead perforation.

TABLE 8.1 Optimal Lead Parameters at Implantation

	Atrium	Right Ventricle	Left Ventricle
Sensing (mV)	≥1.0	≥5.0	≥2.0
Amplitude (V)	≤1.5	≤1.5	≤2.5
Impedance (Ω)	≥350	≥350	≥400–1600
Shock lead impedance (Ω)		>25 to ≤130	

Single-coil leads tend to have higher lead impedance owing to a decrease in electrode surface area compared with dual-coil configurations.

curved lead should move freely in the lower atrium, after which the lead can be gently rotated and pulled straight up to engage the appendage (Video 8.4). Fluoroscopy can sometimes help verify the appendage location by documentation of a so-called windshield wiper appearance of the atrial lead motion. P-wave amplitude may be checked before extending the helix to confirm adequate myocardial contact. Similar to RV lead placement, the active fixation mechanism should be deployed under fluoroscopic magnification, after which the stylet is slightly withdrawn and the lead is given adequate redundancy. Pacing parameters can now be obtained (Table 8.1). Maximum output pacing (10 V) is performed to check for capture of the phrenic nerve, indicating a too-lateral position, and the lead can then be sutured in place with nonabsorbable suture around the suture sleeve to the pectoralis muscle. In patients with previous cardiac surgery, the appendage may no longer be available. The atrial lead can then be placed on the septum or the lateral wall. An active fixation mechanism is required to place a lead in one of these locations. The LAO view is helpful when placing the lead on the septum.

A potential complication of atrial lead placement is the inadvertent placement of the lead across an atrial septal defect or patent foramen ovale into the left atrium. For this reason, an RAO view can be examined to document an anterior direction of the atrial lead when targeting the right atrial appendage. Also, atrial leads can be the cause of cardiac perforation, presenting with tamponade, pericardial effusion, pericarditis. and/or abnormal lead parameters.

A passive fixation atrial lead is no longer commonly used, but it remains an option. These leads are preformed into a "J" configuration and the stylets are straight. The lead is positioned into the low right atrium as described earlier. The straight stylet is removed, causing the lead to curve, which as it is pulled upward ideally will "catch" in the right atrial appendage.

Atrial Lead Selection

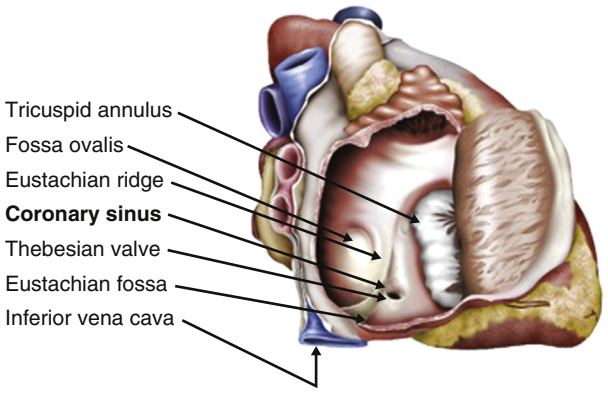
Atrial lead length is usually less critical than RV lead length, but for ease of pocket positioning, it is helpful to have the lengths of atrial and ventricular leads that exit the vascular space match up. Active fixation leads are most commonly used and needed in nonappendage positions. There are a variety of J-shaped curve stylets available. A longer-reach "J" curve may be needed in a large RA when a stable location is difficult to obtain. Occasionally it is necessary to form a custom-shaped curve with the stylet when unusual positions in the lateral or posterolateral RA must be sought.

Left Ventricular Lead Placement

Locating the Coronary Sinus Os

Patients who require CRT or biventricular pacing as part of their pacemaker or ICD system will need an additional transvenous lead to pace the left ventricle. LV pacing is achieved through placement of a lead in a branch of the coronary sinus. The RV lead is typically placed first to provide pacing in case of heart block from catheter-induced RBBB in the patient with LBBB at baseline. A separate venipuncture for the LV lead is recommended to minimize interaction and dislodgement of the RV lead while placing the LV lead. A 0.035-inch (150–190 cm) hydrophilic guidewire such as the Radiofocus Angled Glidewire (Terumo) is used

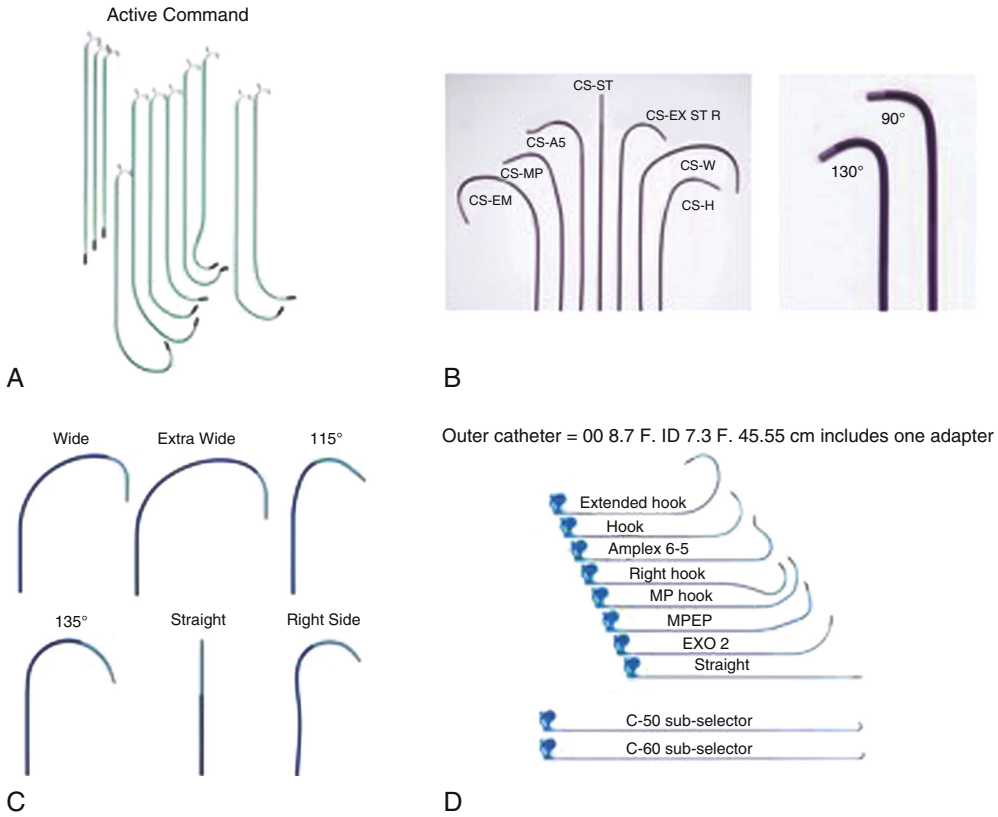
and a pacemaker introducer sheath, typically a splittable, hemostatic introducer system, is advanced over the guidewire into the SVC. The dilator is removed and a coronary sinus guide sheath positioned over the long wire into the right atrium. The coronary sinus (CS) os is typically located between the inferior vena cava and the tricuspid valve on the inferior aspect of the interatrial septum (Fig. 8.30). A variety of LV lead delivery systems are now available depending on heart size, anatomy, and whether it is a right- or left-sided implant (Fig. 8.31A–D). In most patients, the CS os can be located using an average-reach curve such as the extended hook, coronary sinus hook, or the multipurpose guides. In the case of a more vertical takeoff of the CS os or a large, dilated RA, a guide with longer reach may be needed. In the case of a prominent thebesian valve or eustachian ridge, some implanters have used an Amplatz AL2 sheath to gain access to the CS os.



• **Fig. 8.30** Anatomic rendering of the right atrium demonstrating the location of the coronary sinus os.

It is helpful to start with the same guide in each case (depending on preferred manufacturer), as this increases the familiarity with and handling of guides, letting the operator know at an early stage when a different guide sheath will be needed. Right-sided delivery systems are available for right-sided implants, allowing for a more stable introduction into the CS os by letting the curve of the sheath hug the lateral wall of the atrium. A right-sided curve may also increase the difficulty in positioning the sheath farther into the CS and an inner guide may be needed to deliver the lead.

A helpful technique is to advance the guide sheath across the tricuspid valve and slowly pull back while giving the sheath a slight counterclockwise turn, which facilitates reaching the posterior location of the CS os. Locating the CS os can be facilitated with 30-degree RAO and 30-degree LAO views. The RAO view often allows visualization of the atrioventricular groove fat pad. Often by withdrawing the guidewire/sheath apparatus from the ventricle to the atrioventricular groove, and with slight counterclockwise torque, the guide sheath and wire can engage the CS os. A 30-degree LAO view can also facilitate finding the CS os by using the spine as a landmark for the septum. Small 2-mL puffs of contrast can be given to help



• **Fig. 8.31** Guide catheters from Medtronic (A), Biotronik (B), St. Jude Medical (C), and Boston Scientific (D).

visualize the position of the CS os. The 0.035-inch wire is used to probe for the CS os and the guide sheath advanced over the wire into the CS. The 0.035-inch wire is removed and a small amount of contrast given to confirm a CS location.

Alternatively, some implanters engage the CS with a diagnostic electrophysiology (EP) catheter (such as a decapolar catheter). Any of the starting guide sheaths noted previously can be used with the EP catheter or a straight sheath, with a gentle curve created manually. Once the CS os is engaged, the guide sheath can be passed over the diagnostic catheter to enter the CS. This approach may eliminate the need for contrast injection while accessing the CS as the CS position can be confirmed by LAO fluoroscopy.

Tip: The CS os is the most posterior structure in the heart. LAO positioning and counterclockwise torque of the sheath can help with positioning. Make sure pacing is available from the RV lead before CS lead positioning to avoid atrioventricular block due to trauma of the right bundle during implantation.

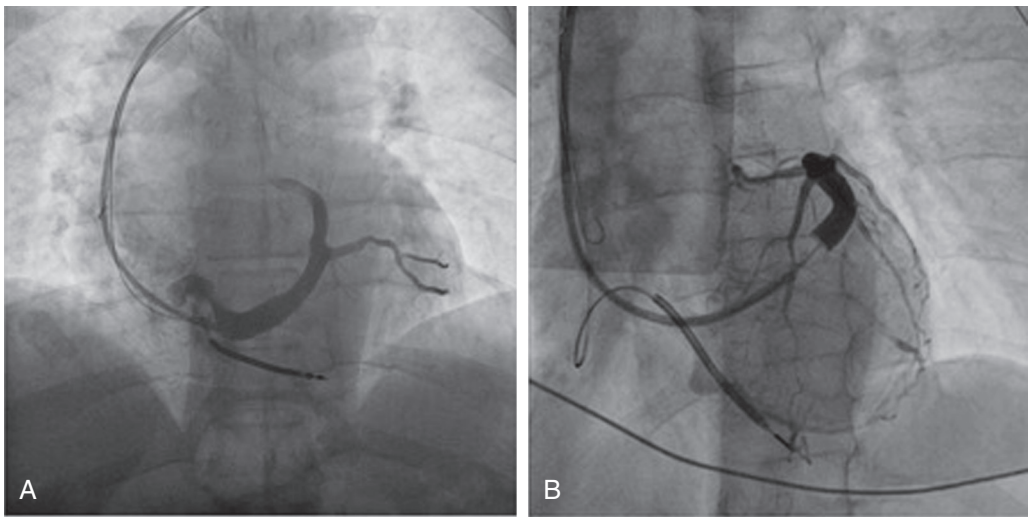
Visualizing Coronary Venous Anatomy

Once CS cannulation is obtained, a venogram will help visualize coronary venous anatomy. Five to 10 mL of contrast are delivered while using cine imaging. This is often enough to establish the availability of adequate branches for the LV lead. If no branches are seen, an occlusive venogram (Fig. 8.32A–B) should be performed using a balloon occlusion catheter. The balloon should be placed in the proximal portion of the CS so as to not inadvertently occlude takeoff of proximal branches. Dissection of the CS can occur with balloon dilation, particularly when using a noncompliant balloon. Venogram projections should be obtained in anteroposterior, LAO, and RAO views. It is helpful to stay on cine long enough to image late filling vessels.

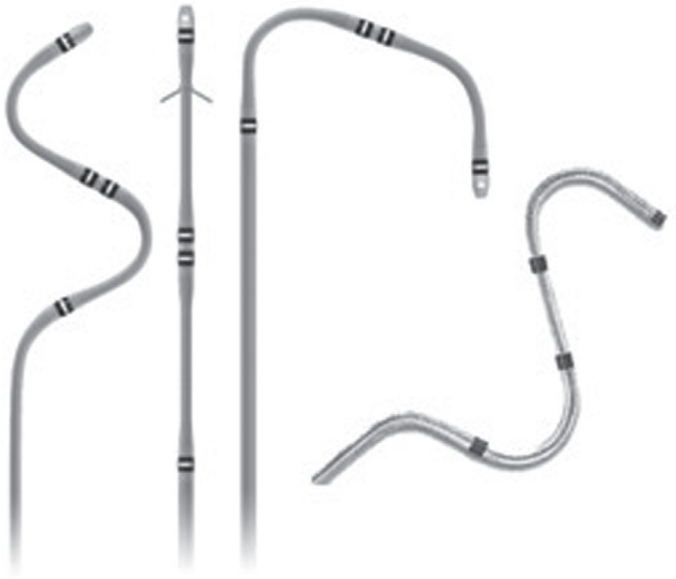
Lead Selection and Placement of the Left Ventricular Lead

At this point, the CS guide sheath should be well situated in the proximal portion of the CS and target branches identified. Before the availability of the quadripolar LV lead, a careful assessment of CS anatomy was needed to identify the best suitable LV leads considering both vessel size and tortuosity. The quadripolar LV lead has now emerged as the first choice in most implants and in many cases allows for a more "forgiving" anatomy. LV leads with four electrodes enable pacing from multiple pacing configurations (Fig. 8.33), thus increasing programming options in cases of diaphragmatic pacing or high pacing thresholds,[42] ultimately reducing the need for LV lead revisions. Available quadripolar leads can be delivered through an inner guide, further adding to the flexibility of placement.

Various guidewires and inner guides are available to facilitate placement. It is helpful to be familiar with the standard guidewires used for LV lead placement (Table 8.2). Similar to always starting with the same guide catheter, it is also helpful to always start with the same guidewire. As with CS guide catheters, it is important to develop your "go to" 0.014-inch guidewire with the understanding of the different support profiles: coating (hydrophilic), parabolic grind, and radiopaque polymer for improved visualization. Table 8.2 classifies 0.014-inch wires into the following three categories: (1) light support or floppy, (2) medium support, and (3) heavy support wires. Medium support 0.014-inch wires (Whisper DS and BMW) are preferred starter wires for engaging most target vessels. The medium support wires offer excellent support, steerability, and trackability even without knowing the venous branch vasculature. Heavy support 0.014-inch wires



• **Fig. 8.32** (A) Cine image of a nonocclusive venogram with an immediate subselection of a lateral branch, making the need for an occlusive venogram less likely. (B) An occlusive venogram with small-diameter branches and late filling.



LV Test Results Report

LV Pace Polarity	Relative Longevity	Capture Threshold	Last Impedance	Phrenic Nerve Stim Present?
LV1 to RVcoil	2 months less	.075 V @ 040 ms	627 ohms	Not Tested
LV1 to LV2	3 months less	1.25 V @ 040 ms	1063 ohms	Not Tested
LV1 to LV3	4 months less	1.25 V @ 040 ms	1045 ohms	Not Tested
LV1 to LV4	Maximum	1.00 V @ 040 ms	969 ohms	Not Tested
LV2 to RVcoil	1-9 years less	3.00 V @ 040 ms	927 ohms	Not Tested
LV2 to LV1	3.6 years less	5.00 V @ 040 ms	1083 ohms	Not Tested
LV2 to LV3	2.4 years less	4.50 V @ 040 ms	836 ohms	Not Tested
LV2 to LV4	2.2 years less	4.50 V @ 040 ms	969 ohms	Not Tested
LV3 to RVcoil	1.1 years less	1.75 V @ 040 ms	570 ohms	Not Tested
LV3 to LV1	1.2 years less	3.00 V @ 040 ms	1045 ohms	Not Tested
LV3 to LV2	1.5 years less	3.00 V @ 040 ms	836 ohms	Not Tested
LV3 to LV4	1.2 years less	2.50 V @ 040 ms	950 ohms	Not Tested
LV4 to RVcoil	3.4 years less	4.50 V @ 040 ms	494 ohms	Not Tested
LV4 to LV1	– – –	>6.00 V @ 040 ms	969 ohms	Not Tested
LV4 to LV2	– – –	– – –	969 ohms	Not Tested
LV4 to LV3	3.8 years less	5.00 V @ 040 ms	950 ohms	Not Tested

• **Fig. 8.33** The Medtronic left ventricular leads. *Left to right,* Attain Performa model 4598 (5.3 Fr, offset S), Attain Performa model 4298 (5.3 Fr, dual bend), Attain Performa model 4398 (5.3 Fr, straight with tines). At *right* is the St. Jude Quartet lead model 1458Q (5 Fr). The left ventricular test result report demonstrates available vectors with a significant range in capture thresholds, impedance, and longevity.

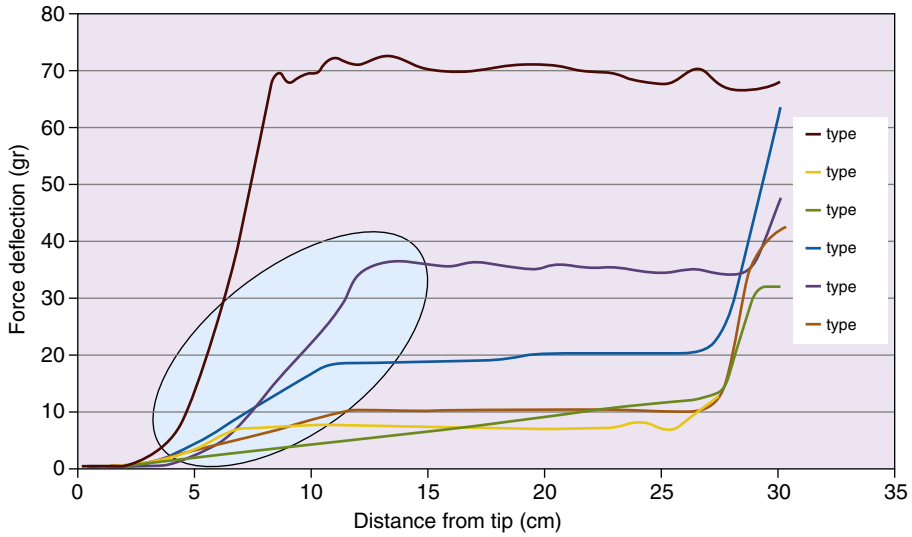
TABLE 8.2 Frequently Used Guidewires in Left Ventricular Lead Implantation

Light Support	Medium Support	Heavy Support
0.014 HT Floppy*	0.014 HT BMW*	0.014 HT Ironman †
0.014 HT Balance*	0.014 HT Balance TREK*	0.014 HT BHW*
0.014 HT Whisper LS†	0.014 Whisper MS†	0.014 HT Whisper ES†
		0.014 ChoICE PT†
		0.014 Spartacore 14*

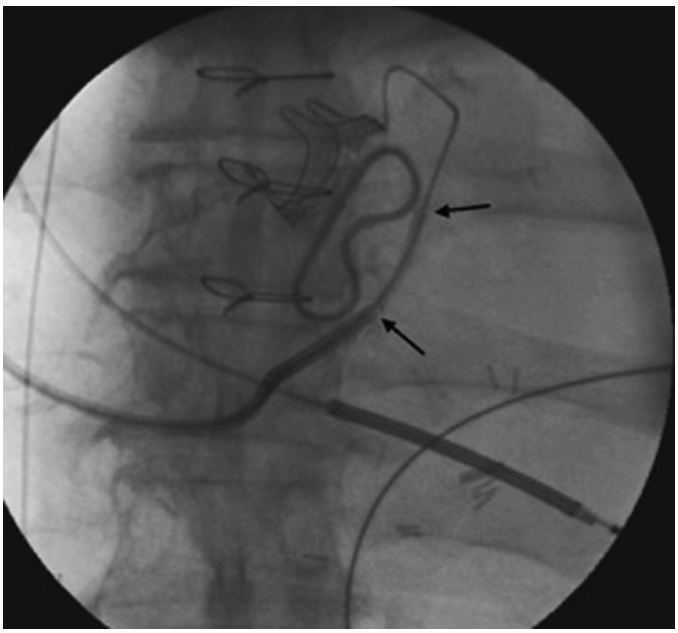
*Abbot Vascular.
†Boston Scientific.

are excellent for additional backbone support and alleviate the potential of prolapsing, giving the operator excellent pushability when attempting to deep-seat the LV lead, thereby reducing the potential for LV lead pullback. Familiarity with a specific guidewire will help the implanter in the decision process of when to switch to a different wire (Fig. 8.34). When advancing LV leads that are low profile with distal fixation mechanisms, the appropriate method is to subselect a tortuous target vessel to seat the lead tip. There is a potential for increased friction when advancing the 0.014-inch wire through the distal tip of the lead. Our recommendation is to slightly retract and readvance the 0.014-inch wire.

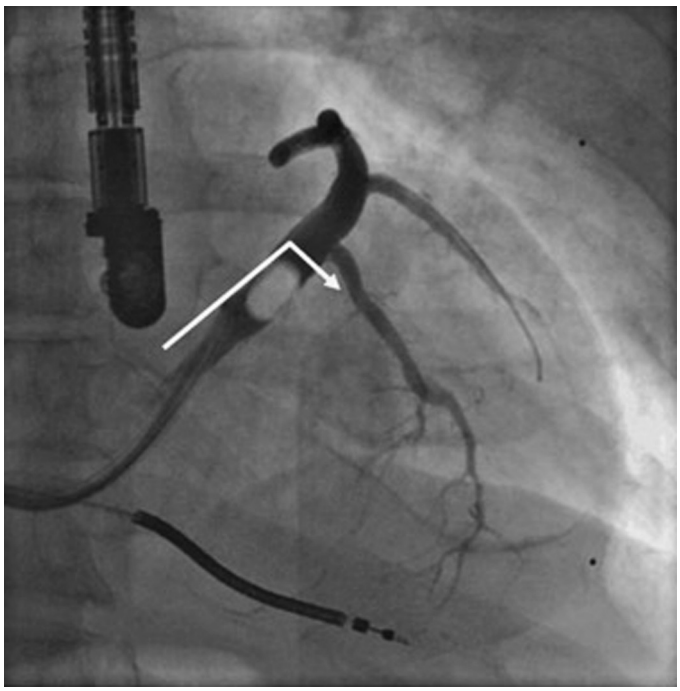
Inner guides or subselectors are useful in many circumstances. As with outer guide sheaths, there are numerous different shapes to choose from, with some variation depending



• **Fig. 8.34** Guidewire comparison chart illustrates the three support levels. The *x*-axis represents the distance from the tip; the *y*-axis represents the deflection force in grams. The Ironman and EDS wire illustrate extra support toward the distal tip, whereas the BMW and ES show decreased support with the same amount of tip pressure on the *y*-axis. Ironman and Whisper models, Boston Scientific; BMW, Abbott Vascular.



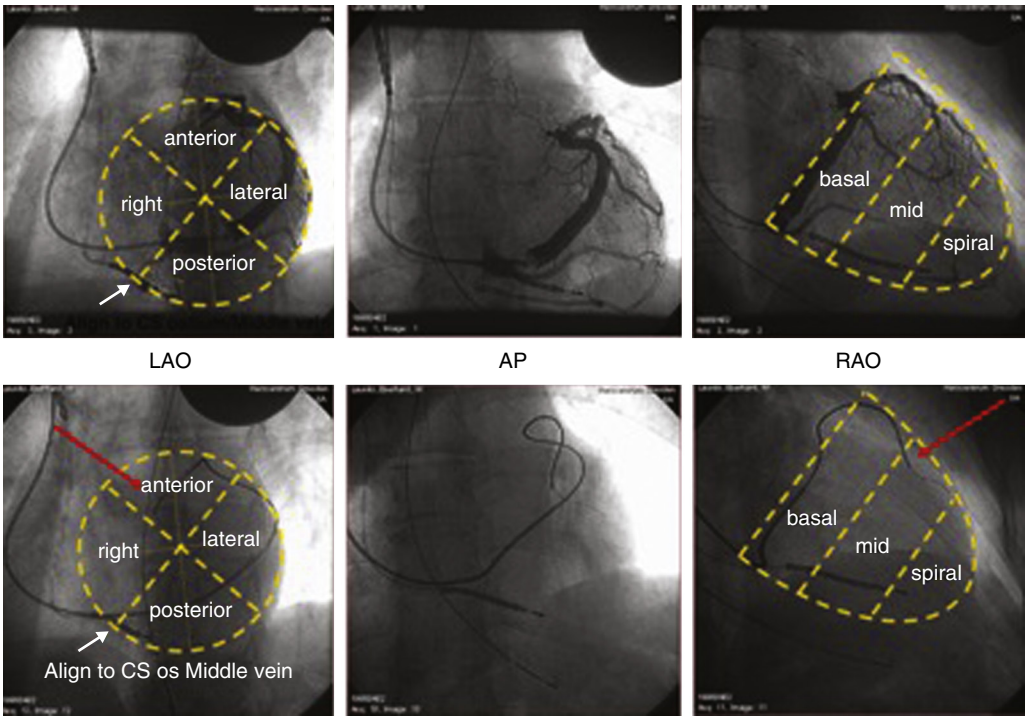
• **Fig. 8.35** Cine image of a steep coronary sinus takeoff requiring the addition of an inner guide *(top arrow)* to stabilize the outer sheath *(bottom arrow)*. Placement done over the guidewire.



• **Fig. 8.36** An angled takeoff of a lateral coronary sinus branch is seen where an inner guide may facilitate lead placement.

on the manufacturer. An inner guide may be used to aid in cannulation of the CS os and, perhaps more often, help stabilize the outer sheath. This is helpful in a more challenging takeoff of the CS os when the outer sheath barely reaches within the CS. In those cases, it is often difficult to place the LV lead without risking immediate dislodgement of the guide catheter. An inner guide/subselector in these circumstances will help deep-seat the guide catheter (Fig. 8.35). This is particularly true with the use of guide catheters designed for right-sided implants.

The subselector can often be very helpful when the target branch has an angled takeoff (Fig. 8.36). The 0.035-inch wire is positioned beyond the takeoff of the target branch; the subselector is advanced and then pulled back while delivering small amounts of contrast. Once subselection is confirmed at the level of the target branch with another small contrast injection, the wire is advanced into the branch as distally as possible. The subselector can now be positioned over the wire into the desired branch. The wire is removed and the LV lead delivered.



• **Fig. 8.37** The left anterior oblique 30-degree view helps localize the wire and leads to an anterior, lateral, or posterior area. The right anterior oblique 30-degree view is useful to determine if the wire and lead are placed in the basal, mid, or apical area. *AP,* anteroposterior; *CS,* coronary sinus; *LAO,* left anterior oblique; *RAO,* right anterior oblique.

Pacing and sensing thresholds are tested, as is impedance (see Table 8.1 for acceptable range). Maximum output pacing (10 V) is performed to check for diaphragmatic stimulation. Multiple fluoroscopic views are needed to confirm lead location (Fig. 8.37). An LAO view will confirm a lateral location, whereas an RAO view will confirm a basal, mid, or apical location. Video 8.5 demonstrates placement of a transvenous LV lead for CRT.

Tip: Positioning of the lead deep in branch for stability may require paradoxic movement of the sheath and the guidewire. Further, torqueing the sheath can help with positioning or may cause the entire system to eject from the CS. All movements of the lead, sheath, and guidewire should be deliberate and may work against the other movements.

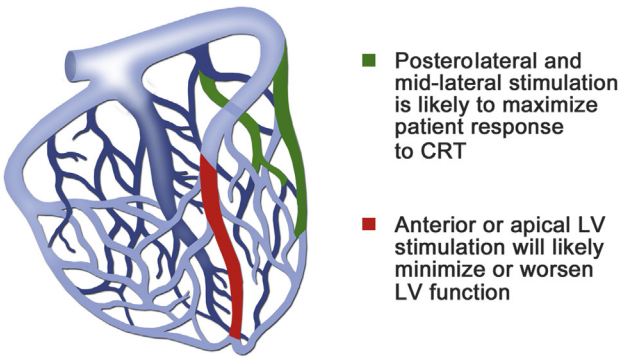
Left Ventricular Lead Location

Ideally, the optimal LV lead location for an individual patient should be easily identified before and during the procedure, thus potentially increasing the chance of CRT response. While this process has not yet been fully refined, data regarding lead location and outcome are emerging. Several studies have now shown that an apical LV lead location has a higher nonresponder rate and less favorable outcome from CRT,[43,44] suggesting that this location should be avoided.

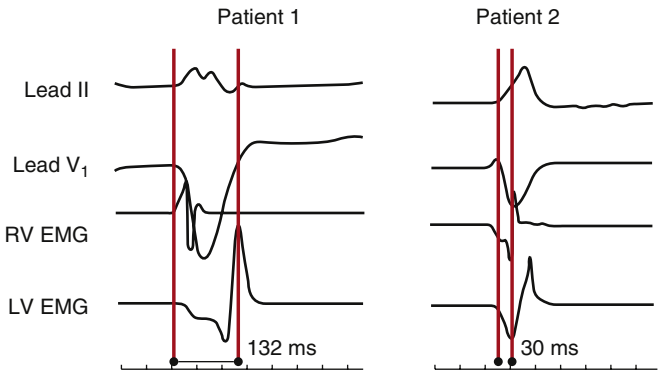
Placement of the LV lead appears most optimal in the region of the midlateral left ventricle, typically reached through a lateral or posterolateral branch. An anterolateral branch may in some circumstances be adequate if the lead can be placed distally enough to reach the lateral wall. Although a midlateral LV location overall appears to provide the best CRT response, an individualized approach to lead placement is clearly ideal. The targeted LV lead placement has generated significant interest, producing a large number of studies aiming to further define the targeted approach.

Imaging methods have been used to improve site-specific pacing. The Resynchronization Therapy for Electrode Region (STARTER) trial randomized 187 patients with heart failure, QRS duration 120 ms or greater, and LV ejection fraction 35% or less to LV lead placement guided to the site of the latest mechanical activation by speckle tracking radial strain compared with routine, nonguided LV lead implantation.[45] In this study, a strategy of echo-guided LV lead placement improved the patient survival rate free from defibrillator therapy in CRT-D recipients.

In the Targeted Left Ventricular Lead Placement to Guide Cardiac Resynchronization Therapy (TARGET) study, 220 patients with standard indications for CRT were randomized to unguided LV lead placement versus placement at the site of latest activation using speckle tracking radial strain. Targeted LV lead placement yielded a greater proportion of responders at 6 months.[46] As demonstrated in these studies, preprocedural identification of a target area may be helpful; however, it lacks the obvious correlation with available cardiac venous anatomy, which rarely is as straightforward as depicted in textbooks (Fig. 8.38). Imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI) have the potential to provide both anatomic and functional information but will require further studies to fully assess usefulness.



• **Fig. 8.38** The preferred and less preferred positions for placement of the left ventricular lead into the coronary sinus. *CRT,* cardiac resynchronization therapy; *LV,* left ventricular; *RV,* right ventricular.



• **Fig. 8.39** Examples of QLV measurements obtained in two different patients. A QLV >95 ms is desirable to increase likelihood of cardiac resynchronization therapy response. *EMG,* electromyogram.

Pacing from a site with more delayed electrical activation as assessed by an LV lead electrical delay (LVLED) greater than half the width of the baseline QRS (LVLED >50%) has been associated with a beneficial acute hemodynamic response and an improved long-term outcome.[47]

In a substudy of 426 patients from the SmartDelay Determined AV Optimization: A Comparison of AV Optimization Methods Used in Cardiac Resynchronization Therapy (SMART-AV) study, a QLV interval (onset of the QRS width to the first large peak of the LV electrogram) of 95 ms was associated with improved reverse remodeling and quality-of-life improvement.[48] Baseline electrical dyssynchrony, as measured by the QLV interval, may therefore be a useful tool to predict CRT response (Fig. 8.39).

Preliminary data suggest that QLV measurement can be obtained during the implant using a 0.014-inch Kinetic guidewire (Boston Scientific), subselecting primary and secondary target vessels using a far-field LV electrogram measuring the QLV (Fig. 8.40).[49] Whether QLV is feasible as determined during the implant procedure and ultimately helpful will, however, require larger, prospective studies.

Left Ventricular Guide Catheter Removal

Once the LV lead has been placed in the best available location and testing has confirmed appropriate pacing and sensing, the guide sheaths will need to be removed. This is done using a so-called slitting tool, which engages the hub of the subselector (if one has been used) and slits the sheath while the sheath is pulled back with the opposing hand. The introducer sheath is removed next, peeled in a standard fashion. The CS guide catheter is removed last, again with the slitting tool used similarly to the subselector removal. It is important to observe this under fluoroscopy to verify a stable lead tip location. If the lead should pull back but still be in the CS branch, the distal location can sometimes be secured by using a more supportive guidewire (Spartacore Choice PT, BHW) or, if possible, a stylet. LV lead redundancy is best observed in the RAO view and should not be excessive as this can contribute to LV lead dislodgment. The lead is sutured in place as described previously.

Left Ventricular Lead Complications at the Time of Implantation

Coronary Sinus Dissection

Coronary vein dissection occurs in 1.3% of implants as reported in a meta-analysis of randomized clinical trials.[50] CS dissection is related to both use of a balloon catheter for a contrast venogram (Fig. 8.41) and sheath and lead manipulation within the CS. The sheath should never be advanced without being preceded by a guidewire to reduce the risk of dissection. A large dissection in the proximal portion of the CS may lead to hemodynamic compromise associated with a pericardial effusion. Monitoring of vital signs, fluoroscopy of the cardiac silhouette, and use of echocardiography should aid in the diagnosis. A more distal dissection of a smaller branch as demonstrated by staining of contrast outside the lumen is typically not associated with any hemodynamic consequences but will likely preclude placement of a lead in this location.

Tip: While a dissection is to be avoided, a small dissection does not necessarily rule out positioning of a CS lead. Also, consider placement in another venous vessel instead (e.g., middle cardiac vein).

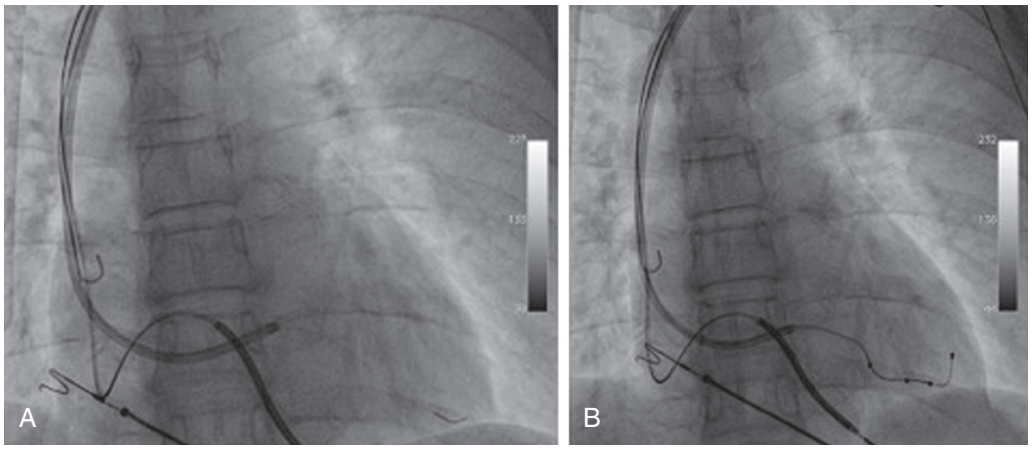
Phrenic Nerve Stimulation

The left phrenic nerve courses over the pericardium of the LV free wall, often in the target area for a mid-lateral LV lead placement. A lead placed in this region may therefore stimulate the phrenic nerve when pacing, causing diaphragmatic stimulation. The advent of quadripolar LV leads with multiple options for pacing vectors has significantly decreased the need for LV lead revisions owing to phrenic nerve stimulation,[43] making this less of a concern during the implant.

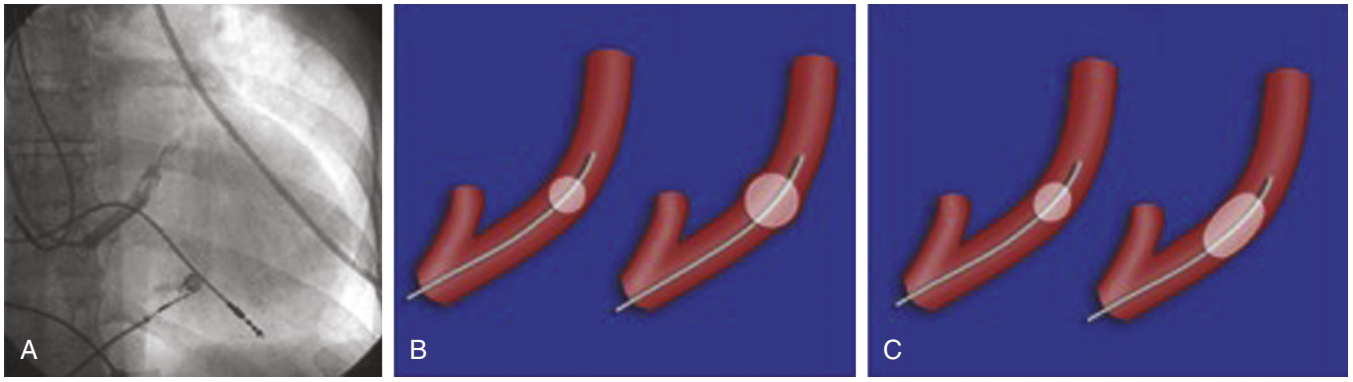
Lead Placement in Nonstandard Cases

Lead Placement in Right-Sided Implants

Most pacemakers and ICDs are implanted on the patient's left side; however, under certain circumstances, the patient's right side may be appropriate. Right-sided implants may be performed when the patient's dominant hand is the left hand

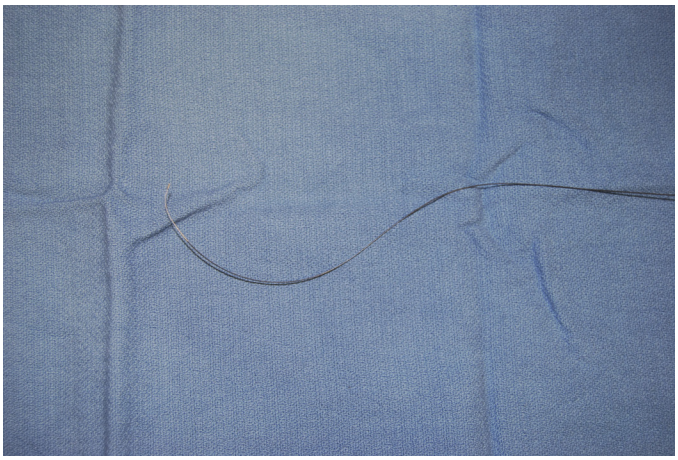


• **Fig. 8.40** Intraprocedural QLV measurement with the wire (A) and then the lead (B) documenting a QLV of 124 ms.



• **Fig. 8.41** Contrast venogram of dissection noncompliant balloon (A) and the difference between a compliant versus a noncompliant balloon. A noncompliant balloon (B) will expand to a predetermined shape, potentially compromising the intimal wall of an undersized coronary sinus os. A compliant balloon (C) will expand longitudinally without disrupting the intimal wall of the coronary sinus os.

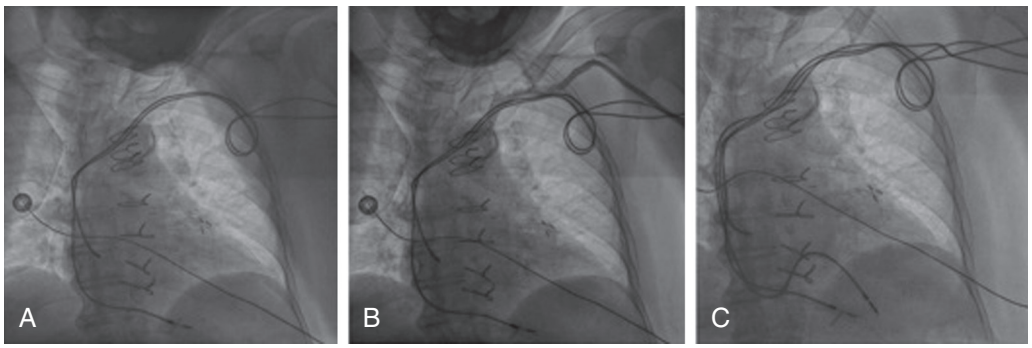
to minimize risk of motion-induced injury to the lead(s). Restriction of venous access due to hemodialysis catheters, arteriovenous fistulas, and infusion ports also limits site availability. Right-sided implants have generally not been correlated with higher complication rates but are anatomically somewhat more challenging. Whereas the transition from the left subclavian vein into the SVC is smooth, the transition on the right side is more angulated, making the insertion point of the needle for the venipuncture even more important. A medial insertion point will further aggravate lead positioning as it traverses the turn into the SVC. Once the RV lead reaches the RA, it will need to curve across the tricuspid valve (TV). This can be achieved by gently torqueing the stylet in the lead to point toward the TV, similar to the left-sided implant. Alternatively, the lead can be gently positioned on the RA lateral wall, the stylet slightly withdrawn, and the lead then given a small push to curve and prolapse across the valve. The use of a custom-curved S-shaped stylet can be useful to manipulate the lead across the tricuspid valve. This allows for stabilization against the lateral right atrial wall (Fig. 8.42). Sheath removal and suturing are done similar to the left-sided implant. Longer leads are rarely necessary in right-sided implants.



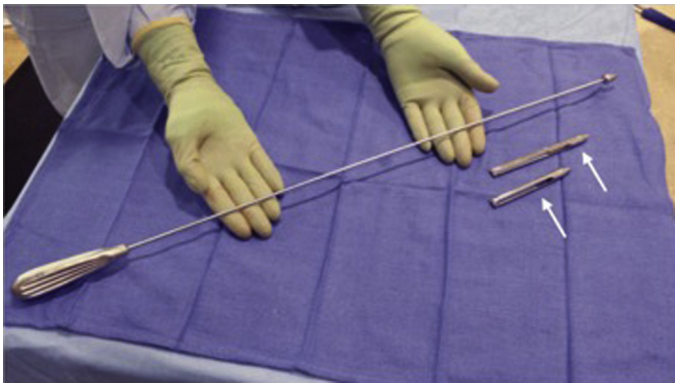
• **Fig. 8.42** S-shaped curved stylet to be used in right-sided implants. The shape aids in stabilizing the lead against the lateral atrial wall and advancing the lead across the tricuspid valve.

Lead Placement in Upgrade Cases

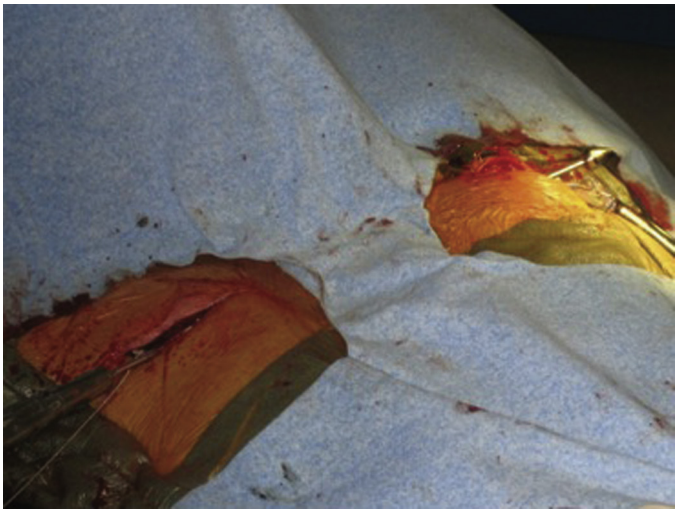
The need for a device upgrade is likely to occur more frequently as patients are being considered for an upgrade from a single- or dual-chamber pacemaker to CRT-pacemaker, from an ICD to a



• **Fig. 8.43** An additional right ventricular (RV) lead is needed in an elderly woman with a dual-chamber pacemaker placed 9 years ago. (A) Leads before procedure. (B) A contrast injection is given with documentation of a patent subclavian vein. (C) A new RV lead is placed with a slightly more lateral insertion.



• **Fig. 8.44** Tunneling tool used in upgrade cases when the contralateral side needs to be accessed.



• **Fig. 8.45** Tunneling tool placed subcutaneously. The handle is removed and replaced with one of the tools shown in Fig. 8.44 *(arrows)*. The leads can now be attached, secured with cotton umbilical tape, and pulled through.

CRT-D, or from a pacemaker to a CRT-D. The current rate of CRT upgrade varies widely among studies: 17.5% underwent an ICD-to-CRT upgrade in the RAFT substudy[51]; in the European Cardiac Resynchronization Therapy Survey of 2367 CRT implant procedures, 29.2% were identified as having an upgrade from a pacemaker to CRT-pacemaker or an ICD to CRT-D.[52]

High success rates are reported in upgrade cases; the initial success rate of an upgrade from an ICD to CRT-D in the Cardiac Resynchronization Therapy for Mild-to-Moderate Heart Failure (RAFT) study was 91% during RAFT and 90% in the substudy.[51,52] In recent studies, complication rates appear similar in upgrade cases as compared to new implants,[51] overall suggesting the high feasibility of upgrades. Data from the Implantable Cardiac Pulse Generator Replacement (REPLACE) registry[53] showed a higher medical and procedural major complication rate in patients who had an upgrade to or a revised cardiac resynchronization therapy device over the course of 6 months. The early complications were largely driven by difficulties in LV lead placement.

Improved LV lead and delivery systems and increased implanter expertise may account for more recent improved complication rates. There are, however, specific aspects of the lead implant that need to be considered. Previously placed leads may have caused a venous obstruction, and an assessment of patency needs to be done before the upgrade either through a venous ultrasound, chest CT, or peripheral intravenous contrast injection (Fig. 8.43A–C).

In case of an obstruction/occlusion, options include a contralateral lead implantation with tunneling across the chest, extraction of a redundant lead, and venoplasty. An individualized approach should be taken based on operator and center expertise. In the case of tunneling, there are several options. One is to use a vascular tunneling tool set as shown in Figs. 8.44 and 8.45 to cross the sternum subcutaneously. This can be somewhat more difficult in a patient with a previous sternotomy but overall is essentially always doable.

Another option for some implanting physicians is to perform venoplasty. This approach was successful in 371 of 373

TABLE 8.3 Procedural Approaches in Upgrade Cases

	Pros	Cons
Tunneling	Implanters are knowledgeable Tools are easily available	Available vascular access is used up More leads are added Consider lead length, adaptors Future potential revisions or needed extraction will be more difficult Cosmetic concerns
Extraction	Redundant leads can be removed System can stay on one side Guideline indications for the procedure exist Excellent safety data from clinical trials	Should only be done at experienced extraction centers Requires preplanning of procedure
Venoplasty	Allows implanter to use the same side for access No need for rescheduling	Requires additional training More leads through one vein Not covered in guidelines

patients as reported by Worley et al. in 2011.[54] Total angiographic occlusion was demonstrated in 65% of cases by peripheral venogram but in only 20% of cases by contrast injection at the site of obstruction; 86% were crossed with a hydrophilic wire. There are advantages and disadvantages to each of these approaches, as summarized in Table 8.3.

Summary

With the expansion of indications for CIED implantation, an increasing number of patients are receiving initial devices or system upgrades. Extensive knowledge of venous anatomy and radiographic landmarks is needed for safe and successful device implantation. Although the approach to transvenous access has not significantly changed over the years, implanting physicians should be familiar with more than one approach to allow an alternative plan for access in difficult cases. The implantation approach can impact the risk of short-term complications, as well as affecting long-term lead failure.

Implanter knowledge, skill, and experience with transvenous lead positioning within the cardiac chambers and coronary sinus are important in improving patient outcomes and minimizing complications. This is particularly the case for LV lead placement. The development of tools for more expedient CS access and delivery of LV leads into precise sites within the coronary venous system has improved outcomes. Advancements in LV lead technology, including availability of quadripolar leads that provide greater flexibility with pacing configurations, has provided a more patient-specific approach to CS pacing aimed at improving response to CRT, minimizing phrenic nerve stimulation, and reducing the need for reoperation.

Despite improvements in technology, the transvenous lead has been considered the "weakest link" in CIED systems. It is hoped that continued advancements in lead technology, including the development of leadless pacing and totally subcutaneous ICD systems, will provide even greater options for more patient-tailored lead placement with continued improvement in lead longevity and long-term outcomes.

Although lead placement may appear to be a rather simple procedure, it is anything but that; complications can occur at every step of the way, so implanters must be vigilant and be cautious at each step. We thank Michelle Williamson for her excellent photographic assistance.

Acknowledgment

We thank Michelle Williamson for her excellent photographic assistance.

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9

Considerations for Novel or Alternative Lead Placement

NAZEM AKOUM, JOSHUA HERMSEN

Introduction

Cardiac rhythm devices have been an essential part of the patient care arsenal for a number of decades. Initial devices were bulky and required cardiac surgery for implantation. In addition, these older devices had limited programmable features. Significant technological advances affecting essentially all the components of the pacing and defibrillation systems have made for smaller, smarter, and longer-lasting devices. In recent years, cardiac rhythm device implants have largely moved from the hands of the cardiac surgeon into the hands of the invasive cardiac electrophysiologist.

Despite their significantly improved reliability, cardiac rhythm devices still have some acute and chronic failings. Acute failings are most commonly procedure-related complications, whereas late failings are more commonly related to lead-related wear and tear, faulty software, and battery depletion. Other problems to contend with are not related to the device hardware per se, but rather to vascular and cardiac pathology. These problems are the basis for consideration of alternative lead placement.

This chapter discusses specific case scenarios in which alternative lead placement may be considered or may possibly be the only option. These include azygos vein or subcutaneous shocking-coil implants for patients with elevated defibrillation thresholds, as well as femoral, epicardial, and leadless pacing for patients with limited conventional transvenous options.

Considerations for Alternative Shocking-Coil Placement

Historically, defibrillation threshold testing was an integral part of the initial implant or replacement of an implantable cardioverter-defibrillator (ICD) system. Current practice trends show that the percentage of implants in which this is done is decreasing. This is driven by registry data as well as randomized trials showing no benefit of testing in shock efficacy or reduction in arrhythmic death.[1,2] Generally, patients receiving ICDs for secondary prevention of sudden death receive defibrillation testing. Other patient groups that are more likely to receive defibrillation testing include those with a low sensed R-wave amplitude (concerning for undersensing of ventricular fibrillation) and those chronically treated with amiodarone therapy, which can cause an elevation of the defibrillation threshold.[3]

When defibrillation testing is performed, induction of ventricular flutter or fibrillation is achieved through rapid pacing, T-wave shock, or direct current application. The purpose of inducing ventricular arrhythmias is to ensure that these are properly sensed and terminated by the implanted device. Most operators aim for a 10-J safety margin above the defibrillation threshold. About 5% of initial ICD implants are found to have elevated defibrillation thresholds (DFTs). Severe heart failure, dilated cardiomyopathies, and chronic therapy with amiodarone are associated with high DFTs.[4] This commonly occurs when the defibrillation vectors between the active generator, or "can," and the shocking coil(s) do not adequately "sandwich" the bulk of the fibrillating ventricular myocardium. A more posterior shocking-coil location is needed to lower the defibrillation threshold. The azygos vein is one option for placing a defibrillation coil, and a subcutaneous single shocking coil or an array of posteriorly directed shocking coils is another.

Azygos Vein Lead Placement

The azygos vein tributaries originate around the renal veins. As it continues its course cranially, it receives tributaries from the subcostal veins. The azygos vein runs superiorly and rightward to the vertebral column and is also joined by the hemiazygos vein. The opening of the azygos vein normally has a diameter of about 1 cm and is found on the posterior aspect of the superior vena cava (SVC). It joins the SVC just superior to the level of the right mainstem bronchus (Fig. 9.1).

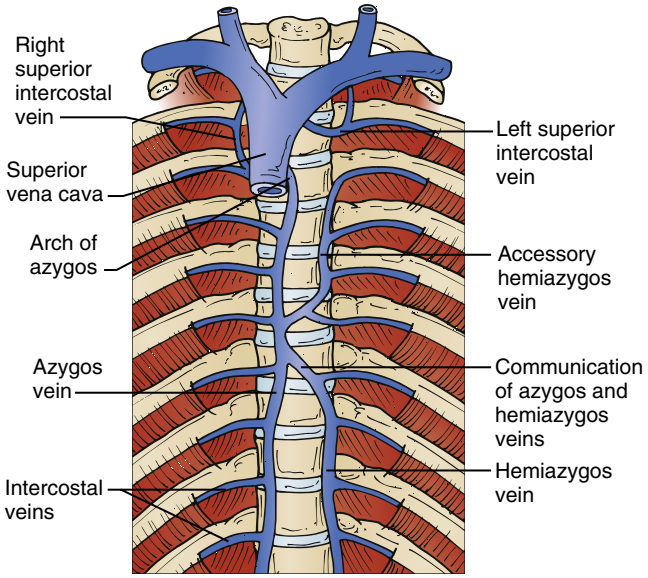
With this as a radiographic landmark, the azygos vein is engaged using a Judkins right or a multipurpose diagnostic coronary catheter and a hydrophilic wire. With fluoroscopic guidance, the wire is typically seen taking a posterior trajectory for a few centimeters before it courses inferiorly and posteriorly to the heart (Fig. 9.2).

Once the azygos vein is engaged, a 9-Fr sheath is advanced as distally as possible over the wire. After the wire is removed and the sheath flushed, the shocking coil is then advanced through

the sheath into the azygos vein. Once the azygos coil is incorporated into the defibrillator system, typically in place of the SVC coil, the shocking impedance increases slightly. The improvement in the defibrillation threshold is therefore not attributed to a decrease in shocking impedance but rather to the improved shocking vector with the more posterior azygos coil.[5]

Subcutaneous Shocking-Coil Placement

Another option for patients with an elevated defibrillation threshold is a subcutaneous shocking coil or an array of coils. The goal here also is to improve the shocking vector by placing a coil posterior to the heart. A tunneling tool is used to create the path for the additional coil starting from the bottom of the prepectoral pocket. A good technique for tunneling is to shape the tunneling tool along the curvature of the chest wall. This helps avoid breaking through the skin as well as the chest cavity. The direction of tunneling is inferior and posterior starting in the bottom of the generator pocket. The operator should aim to achieve a posterior lead position such that the tip of the lead or tunneling tool is as close to the vertebral column as possible. The addition of a subcutaneous coil reduced DFTs by 8 to 11 J.[4,6] Fig. 9.3 shows an example of a subcutaneous shocking coil used with a cardiac resynchronization therapy defibrillator device.[6]

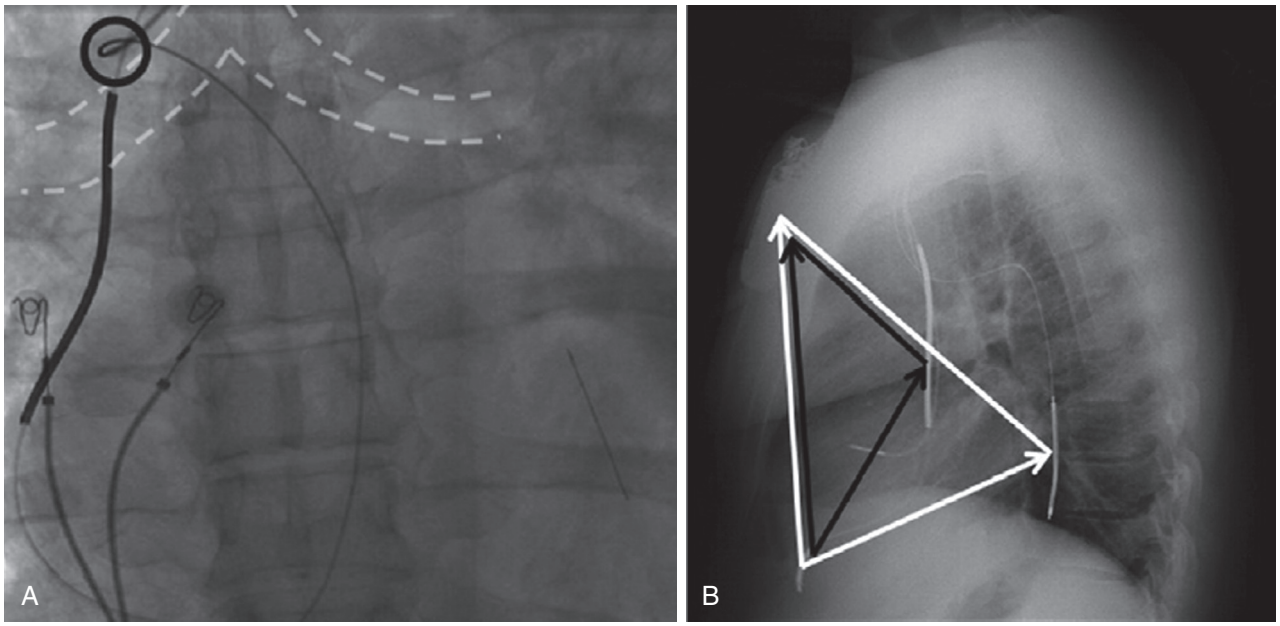


• **Fig. 9.1** Position of the azygos vein in the chest. The azygos vein originates from the posterior upper superior vena cava.

Considerations for Alternative Pacing Lead Placement

The most common approach for lead placement is upper extremity axillary or subclavian veins. With venous occlusions or multiple leads overcrowding and impeding blood flow, the first alternative considered is implanting on the contralateral side. Other options include performing angioplasty to the occluded or stenosed subclavian or innominate veins, which has been shown to be a safe option without significant consequences on preexisting leads.[7]

When options to use upper extremity veins are exhausted, alternative approaches are sought. With leadless pacing, for example, an argument could be made that this approach can potentially prevent vascular and valvular complications.



• **Fig. 9.2** (A) Azygos vein cannulation using a JR4 or multipurpose diagnostic coronary catheter. Note the posterior direction of the initial portion of the vein trajectory, which then courses inferiorly. (B) Lateral radiographic view shows the posterior position of the shocking coil. (Modified from Cooper JA, Smith TW. How to implant a defibrillation coil in the azygous vein. *Heart Rhythm.* 2009;6:1677-1680.)

Permanent Pacing Leads Implanted Through the Jugular Vein

The jugular vein approach is the most common for temporary transvenous pacing and can be used to insert permanent pacing leads as well. With this approach, the pacemaker pocket is in its usual prepectoral position. Access to the internal jugular is obtained using ultrasound guidance from the anterior triangle and a wire is placed and secured. A 1-cm incision is then made to create space for tunneling and securing the lead. The pacing lead is inserted through a sheath placed into the vein and the venous sheath is split and removed. A trochar is then used to create a tunnel from the pacemaker pocket to the jugular incision. After the lead is secured with sutures over the suture sleeve, its proximal end is pulled back through the tunnel to the prepectoral pocket and attached to the permanent pacemaker generator.

Permanent Pacing Leads Implanted Through the Femoral Vein

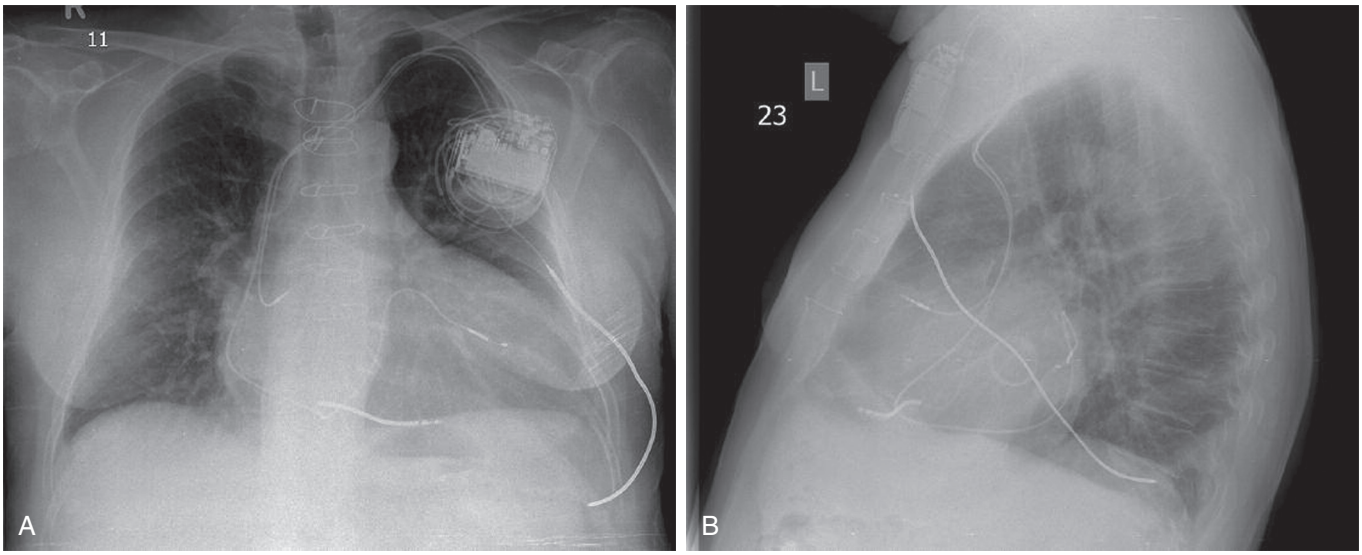
Insertion of a femoral or iliac pacing or defibrillator lead is similar to insertion of a temporary wire. A horizontal incision is made cranial to the groin skin crease but caudal to the inguinal ligament. Dissection is carried down to the deep fascia and a pacemaker pocket is created cranially from the incision. The femoral vein is accessed using anatomic landmarks or ultrasound guidance. A long sheath with a steerable or fixed curve can be used to guide the lead across the tricuspid valve into the right ventricle. Once the lead is in place, it is sutured over a suture sleeve in the femoral region and reflected back up toward the pocket. Once the leads are placed and connected to the generator, the pocket is closed, preferably with a strong suture to prevent caudal migration of the generator.[8] The challenge with this approach is usually with lead length depending on the patient's body habitus. Long leads 75 or 85 cm in length may be needed. Occasionally lead extenders can also be used in the case of pacing leads but not ICD leads. Fig. 9.4 shows a single-chamber ventricular pacemaker system with the distal lead actively fixated to the ventricular myocardium and the proximal portion connected to a pacemaker placed in an abdominal pocket.[9] In addition, care should be taken to keep the incision sites in the femoral region clean and infection free.[8] Other potential complications include lead dislodgement and lead-related deep or inferior vena cava thrombosis and embolization.

Temporary-Permanent Transvenous Leads

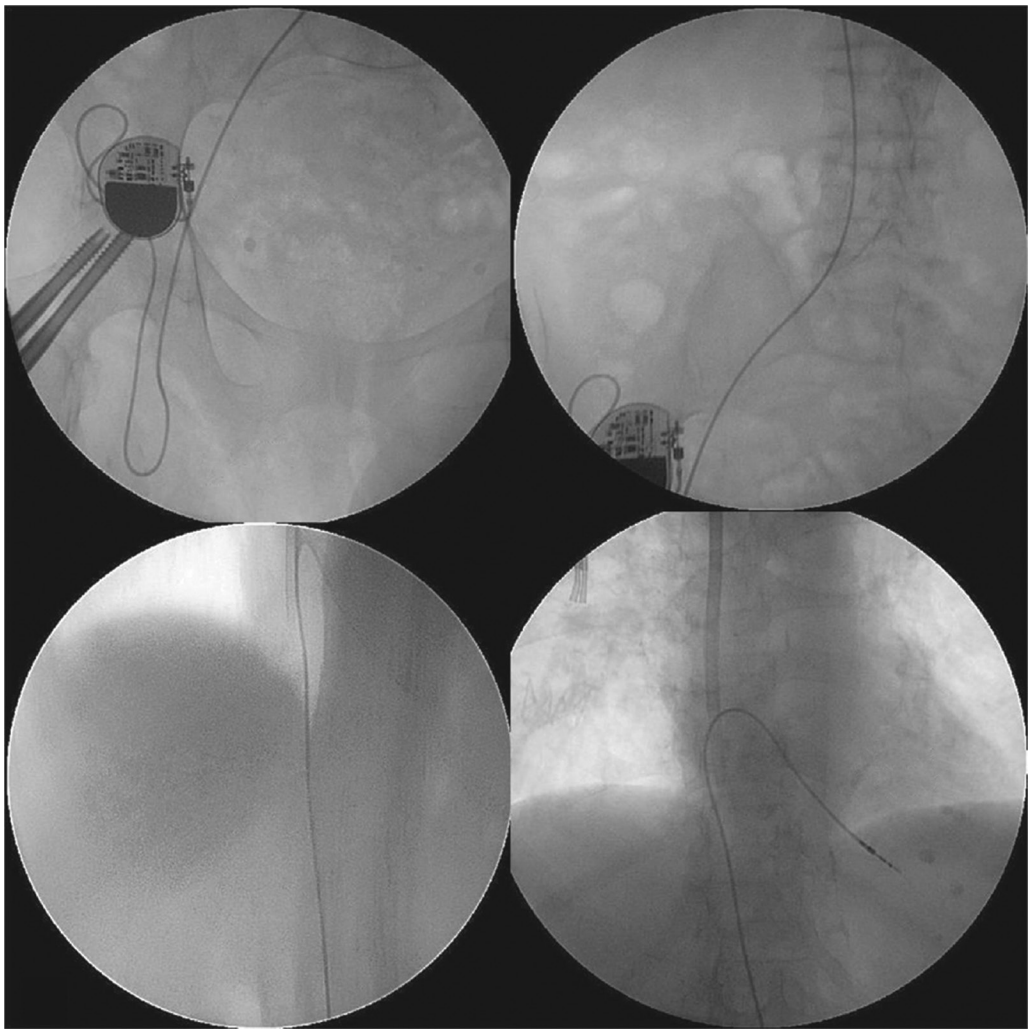
In situations where a patient must be managed with a temporary pacemaker for several days, the use of a permanent pacemaker lead with active fixation attached to a sterilized permanent pacemaker generator may be considered. A permanent pacing lead is less likely to dislodge than the typical balloon-tipped temporary wire. The lead is fixed to the skin using its anchoring sleeve, and the pulse generator may be secured to the external chest using a nonabsorbable suture placed through the anchoring hole in the device header block and covered with an occlusive bandage. Patients on whom this approach may be used are those who have had leads extracted and require pacing, but a new implanted device will be delayed for antibiotic therapy. Other situations appropriate to consider are when comorbid conditions must be stabilized, delaying implantation of a permanent pacemaker, and when a common temporary pacemaker lead is unstable.

Epicardial Pacing Leads

Placement of pacing leads directly onto the surface of the heart, so-called epicardial leads, can be a useful and sometimes necessary option. The decision to place epicardial leads is usually dependent on non–rhythm-related factors. That is, epicardial leads offer no significant advantage in rhythm management



• **Fig. 9.3** Posterior-anterior (A) and lateral (B) radiographic views showing a subcutaneous shocking coil coursing inferiorly and posteriorly following the curvature of the chest wall. (Modified from Kempa M, Budrejko S, Drelich L, et al. Implantation of additional subcutaneous array electrode reduces defibrillation threshold in ICD patients: preliminary results. *Arch Med Sci.* 2013;9:440-444.)



• **Fig. 9.4** Radiograph showing a pacing lead placed through the femoral approach. The lead is reflected cranially from the vein access site to an abdominal pacemaker generator pocket. (From Rodrigues P, Reis H, Lagarto V, et al. Permanent pacemaker implantation using a femoral approach. *Rev Port Cardiol.* 2014;33[11]:733. Copyright 2014 Sociedade Portuguesa de Cardiologia. Published by Elsevier España, S.L.U. All rights reserved.)

but may be the only available route or offer advantages because of concomitant conditions. Frequent indications for epicardial lead placement will be reviewed and technical considerations in lead placement discussed.

Infectious Endocarditis

Infectious endocarditis (IE) involving any heart valve may result in heart block usually due to abscess formation affecting the crux and specifically the anterior basal ventricular septum. In most cases, heart block related to endocarditis constitutes an urgent indication for operative management and, if needed, is best managed preoperatively with a temporary transvenous lead. Even if the patient is in sinus rhythm at the end of the operation, a ventricular lead in this situation should be considered for multiple reasons: The rhythm can be quite dynamic in this milieu, recurrent infection (prosthetic valve endocarditis) is always a concern (especially in cases related to intravenous drug abuse), and placement of an intravenous lead if needed in the postoperative period is unappealing from an infection standpoint. The lead may be capped and left coiled within the rectus sheath on the most convenient side or tunneled to an infraclavicular pocket on the anterior chest wall. A generator can be placed later if needed, which is a relatively minor procedure. Indeed, one general advantage of epicardial leads is their extravascular position, which changes the potential impact of a lead- or generator-related infection.

End-Stage Renal Disease

Epicardial lead placement is often most appropriate for patients with end-stage renal disease (ESRD) whether in the context of concomitant cardiac surgery or not. Because of the "fistula first" mantra of hemodialysis access, indwelling venous devices in the upper body should be avoided owing to the propensity for related venous scarring and fibrosis that can lead to intractable stenosis. Such stenoses can limit fistula effectiveness or even mitigate initial maturization. Additionally, repeated use of the fistula for hemodialysis increases the risk of introduction of bacteria into the bloodstream and subsequent infection of the transvenous lead(s). Finally, venous pressures occurring as a result of anastomosis with its arterial component significantly

increase the risk of bleeding during lead insertion. In these patients epicardial lead placement should be considered and is generally preferable to a transvenous device via femoral access.

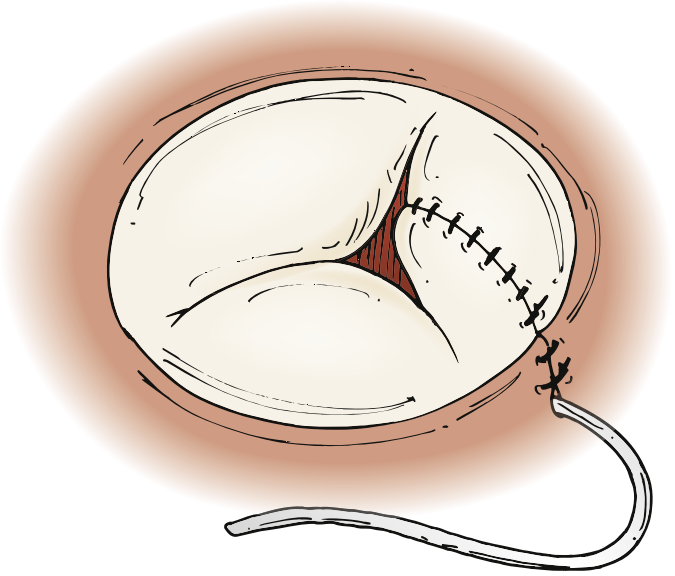
In advanced cases patients may present with exhausted venous access and have no central venous channels through which pacing leads can be advanced into the heart. These patients can have extensive thoracic venous collaterals, to which the surgeon must be attentive. A physical examination is crucial to identify an approach that avoids such collaterals, and a venous-phase computed tomography scan of the chest may also be helpful for operative planning. In these patients, epicardial lead placement via a subxiphoid window is the most reasonable solution. Atrial and right ventricular leads can usually be placed with minimal difficulty using this approach. If a left ventricular lead is required, a separate limited anterior left thoracotomy may be needed. In patients with previous sternotomy, access for epicardial lead placement may be limited by scar formation and a partial or full reoperative sternotomy may be required.

Adult Congenital Heart Disease

Patients with adult congenital heart disease (ACHD) are another group in which epicardial lead placement is often necessary. In patients with single ventricles with completed Fontan circulations, there is no connection between the cavae and the heart. In patients with "classic" atriopulmonary Fontan circulations, transvenous leads can be placed. However, if such patients have nonemergent pacing needs, the possibility of Fontan conversion should be considered before a permanent transvenous device is placed as this is often the most definitive way to deal with Fontan-related rhythm issues. Other common issues in patients with ACHD relate to bilateral superior cavae (in which case both are usually relatively small) and persistent left cavae connected to the left atrium, as intracardiac pacing leads on the left side of the heart should be avoided. Similarly, patients with unrepairable intracardiac shunts and Eisenmenger physiology are poor candidates for transvenous leads given the risk of systemic embolization. Patients with Mustard repair of dextrotransposition of the great arteries can also pose pacing challenges, especially if associated with venous baffle obstructions or leaks. Patients with congenitally corrected transposition are at especially high risk of heart block and epicardial leads should be placed in any adult with this lesion who is undergoing sternotomy.

Tricuspid Valve Surgery

It is not ideal to have pacing leads course through a repaired or replaced tricuspid valve. This becomes increasingly evident the more complicated the repair becomes, and is relatively imperative for all replacements. Pacing decisions related to tricuspid valve surgery patients must be individualized. If tricuspid annuloplasty alone is performed with leads already in place, it is simplest to bicuspidize the valve by suturing together the posterior and septal leaflets and in the process trapping the lead in the posteroseptal commissure (Fig. 9.5). This is most commonly done in patients undergoing left ventricular assist device (LVAD) implantation, a large majority of whom have cardiac implantable electronic devices (CIEDs) already in place. It is imperative that the electrophysiologist understand that this technique is used, as it may bear considerable impact on a decision to perform transvenous lead extraction. For patients with more complicated tricuspid valve repairs (i.e., cone repair for Ebstein anomaly), transvenous leads should be removed and replaced with an epicardial system.



• **Fig. 9.5** Representation of tricuspid valve bicuspidization to "trap" an intracardiac lead outside of the functional valve apparatus.

Patients With Intracardiac Shunts

There are minimal guidelines regarding transvenous versus epicardial lead placement in patients with intracardiac shunts (atrial septal defect, ventricular septal defect, patent foramen ovale). The concern in this situation is for systemic embolization of lead-related thrombus or vegetation. The 2008 American College of Cardiology/American Heart Association (ACC/AHA) Guidelines for Device-Based Therapy of Cardiac Rhythm abnormalities briefly state that epicardial leads are preferred in the presence of a right-to-left shunt. A large propensity-matched study revealed no difference in the rate of stroke among patients with patent foramen ovale with or without transvenous pacing leads and concluded that most strokes in the study were actually related to atrial fibrillation.[10] Epicardial leads should be preferred in patients with intracardiac shunts other than patent foramen ovale, although therapeutic decisions should be made on an individual basis. Many patients with intracardiac shunts that cannot be closed may also have significant risk related to general anesthesia and surgical epicardial lead placement (e.g., Eisenmenger physiology, systemic-to-pulmonary collaterals, multiple previous operations). Anticoagulation should be considered for patients with intracardiac shunts and transvenous leads.

Technical Features of Epicardial Lead Placement

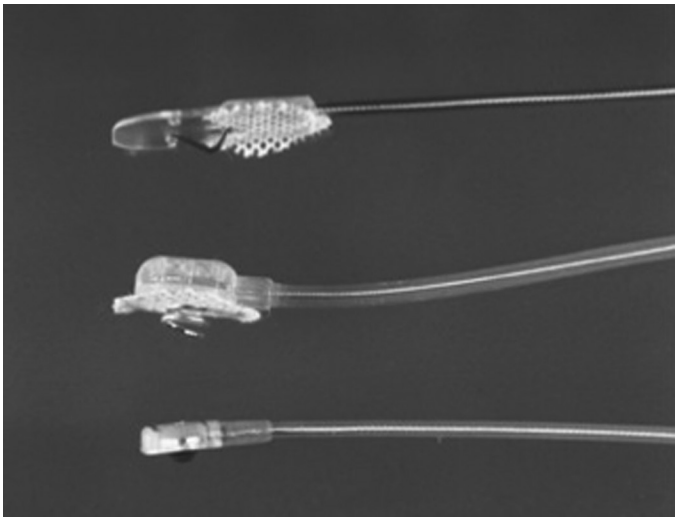
Epicardial lead placement is conceptually and technically simple. In cases of concomitant operation, the leads are placed at the end of the procedure following protamine administration to optimize sensing, lead impedance, and pacing thresholds,

and to minimize epicardial bleeding and hematoma formation. Temporary leads are placed if needed to wean from bypass and removed once permanent leads are in place and functional.

If leads are affixed to the heart in areas of bare muscle or atrium not covered with epicardial fat or scar tissue, contact and electrical conduction are usually more than adequate. Epicardial coronary arteries must be avoided. Technical challenges are faced almost exclusively in reoperative situations, in which scar tissue and loss of normal tissue planes can make it difficult to identify myocardium and place leads that have adequate contact. Often this becomes a "hunt and peck" session that can be quite tedious. In this situation suboptimal lead testing parameters often must be accepted at the expense of shorter expected battery life.

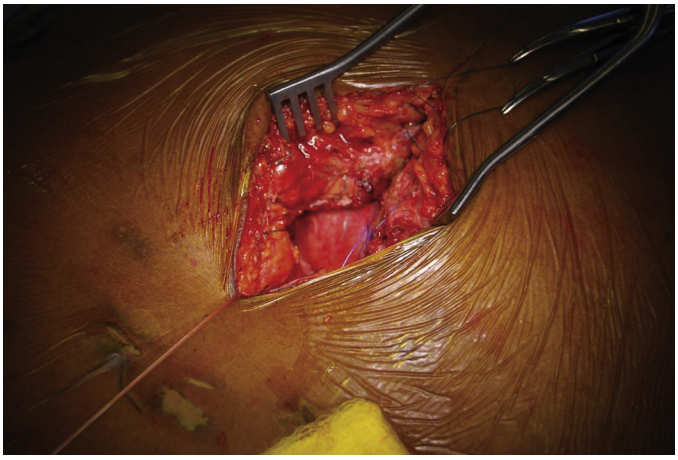
Epicardial leads come in two main types: sew-on and screw-in (Fig. 9.6). The most common configuration consists of bipolar sew-on atrial and bipolar screw-in ventricular leads in surgical patients with complete heart block. For patients with chronic atrial fibrillation, a ventricular lead is sufficient. Rarely the sinoatrial node is damaged in isolation, in which case an atrial lead alone is sufficient. The main advantage of the screw-in ventricular lead is only needing to find one area of adequate myocardium. Atrial leads can be placed on the right or left atrium, on either atrial appendage, or near the pulmonary veins if necessary. In young patients consideration should be given to placing leads on both ventricles to allow for synchronized biventricular pacing, thereby potentially minimizing the risk of right-ventricle-only pacing-induced cardiomyopathy. Data from the transvenous pacing literature in patients with atrioventricular block support this approach.[11,12] Placement of the left ventricular lead should be targeted to the lateral or posterolateral basal left ventricle (e.g., between the first and second obtuse marginal coronary artery branches), as apical and anterior sites generally result in poor interlead pacing vectors.[13] Figs. 9.7 through 9.11 show placement of an epicardial pacing lead.

Once affixed, leads are individually tested for sensing (P waves, R waves), lead impedance, and pacing thresholds. Ideally, ventricular leads should display an R-wave amplitude of at least 5 mV and atrial P-wave amplitude of 2 mV. Pacing thresholds less than 2 V at a pulse width of 0.5 ms are desirable in each position.[14] Parameters generally improve once leads are connected to the generator and placed intracorporeally.

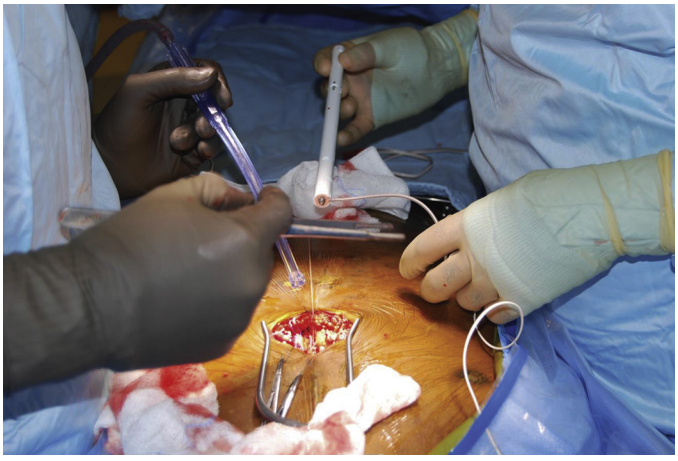


• **Fig. 9.6** Epicardial pacing leads: stab-on *(top),* screw-on *(middle),* and suture-on *(bottom)* buttons.

• **Fig. 9.7** Myocardial contact surface of bipolar epicardial screw-on pacemaker lead (Greatbatch Medical).



• **Fig. 9.8** Subxiphoid incision through recent median sternotomy incision, inferior wall of right ventricle exposed.



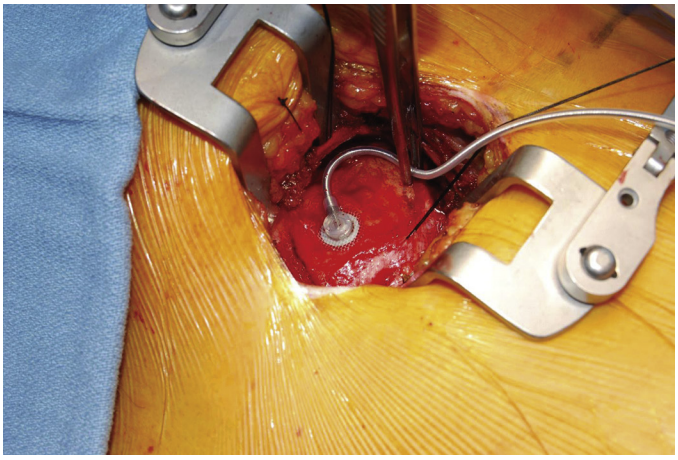
• **Fig. 9.9** Epicardial screw-on lead loaded on delivery tool.

Generator placement is most easily accomplished in the upper rectus sheath either anterior or posterior to the muscle. A generator pocket created between the muscle belly and the posterior sheath provides excellent soft tissue coverage and allows good exclusion from the mediastinal wound and chest drains. Generator replacement is a bit more difficult compared to placement in the anterior rectus sheath or in subcutaneous tissue, but not prohibitively so. Alternatively, the lead may be tunneled laterally and anteriorly through the chest wall into a pocket formed in the more typical subclavicular location. Regardless of pocket location, once the generator is attached to the lead(s), the lead redundancy is coiled and positioned posterior to the generator to protect the leads.

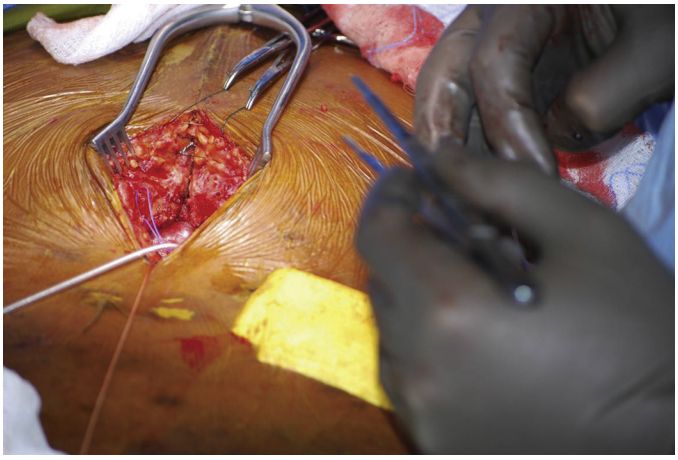
Leadless Pacing

Advancements in technology and delivery systems have brought us the option of leadless pacing, where the entire pacing system is fitted in a miniaturized capsule that is delivered and secured directly onto the heart. Leadless pacing therefore avoids all pocket-related complications including infections, erosion, pain, and cosmetic considerations. In addition, it circumvents lead-related complications including insulation and conductor fractures and iatrogenic tricuspid valve regurgitation. Leadless pacemakers also offer a great solution for patients with upper extremity vascular access limitations such as vein occlusions and atrioventricular fistulas for dialysis.

Currently, two options are available for leadless pacing: the St. Jude Nanostim leadless cardiac pacemaker (LCP)[15] and the Medtronic Micra transcatheter pacing system (TPS).[16,17] These devices had not yet received approval from the U.S. Food and Drug Administration at the time of writing this chapter, although it is soon anticipated (see Figs. 9.8 and 9.9). Both of these systems are implanted using a delivery system inserted through the femoral vein. They offer VVI pacing with a programmable rate response mode. The Nanostim utilizes a temperature sensor, whereas the Micra uses an accelerometer rate response sensor.[18,19] Sensing and capture thresholds and battery longevity were similar in leadless systems and conventional pacemakers in initial published reports. The key differences between the currently available leadless pacing options are summarized in Table 9.1.



• **Fig. 9.10** Epicardial screw-on lead in place on myocardium.



• **Fig. 9.11** Epicardial screw-on lead in place with lead exiting the subxiphoid incision.

TABLE 9.1 Summary of Specifications and Key Differences Between the Nanostim and Micra Leadless Pacemaker Options

	St. Jude Medical Nanostim	Medtronic Micra
Volume (mL)	1.0	0.8
Length (mm)	41.4	25.9
Weight (g)	2	2
Sheath size (Fr)	18	23
Fixation	Screw-in helix + secondary tines	Self-expanding tines
Battery longevity in years[a]	9.8	4.7
Pacing mode	VVI(R)	VVI(R)
Rate response mechanism	Blood temperature	Accelerometer
Wireless communication	Conductive	Radiofrequency

[a]International Organization for Standardization, 60 beats/min, 100% pacing, 2.5 V at 0.4 ms, impedance 600 ohms.

Patients with mechanical tricuspid valves, severe pulmonary hypertension, preexisting endocardial pacing, or defibrillator leads and inferior vena cava filters were excluded from the initial studies on leadless pacing. Initial experience demonstrated that these devices could be retrieved and repositioned. Future generations will likely allow for dual-chamber pacing.

As the expertise with these device implants grows, complication rates associated with the initial experience likely will be diminished. These complications include vascular injury and bleeding, cardiac perforation, and tamponade, which may lead to death or severe disability, especially in frail elderly patients.[17,18]

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10

Implanting the Subcutaneous Implantable Cardioverter-Defibrillator

KRISTEN K. PATTON

Introduction

Implantable cardioverter-defibrillator (ICD) therapy reduces the risk of arrhythmic mortality[1]; however, transvenous leads are associated with complications that include vascular obstruction, pneumothorax, lead dislodgement, tricuspid valve injury, infection, cardiac perforation, and long-term electrical and mechanical failure.[2] The development of the subcutaneous ICD (S-ICD) was driven by the desire to provide life-saving ICD therapy and avoid lead-related complications. The S-ICD allows for detection and shock therapy of ventricular arrhythmias without requiring an intravascular component, thereby reducing the substantial procedural and long-term risk of endocardial transvenous lead placement. Implant techniques for the S-ICD date back to the initial days of defibrillator placement and require knowledge of chest anatomy and tunneling methods beyond those typically necessary for implanting transvenous systems.

The S-ICD system consists of a subcutaneous lead that is tunneled from the generator pocket to a paraxiphoid incision and then superiorly along the sterum. The generator is positioned between the left lateral anterior and midaxillary lines (Figs. 10.1 and 10.2). The lead has two sensing electrodes: one near the tip and the second just proximal to the 8-cm shock coil. Sensing occurs between either of the sensing electrodes on the lead to the can or between the two sensing electrodes, allowing for three choices of a sensing vector (Fig. 10.3): primary (proximal electrode to can), secondary (distal electrode to can), and alternate (distal electrode to proximal electrode). The S-ICD automatically selects the sensing vector to avoid inappropriate detection of T waves, myopotentials, and noise. Sensing algorithms have been continually refined to further reduce rates of inappropriate shocks.[3] Shocks of up to 80 J are delivered between the parasternal coil and the generator, and shock polarity can be reversed.

Indications

S-ICD indications mirror those of the conventional transvenous defibrillator[4] with some exceptions. While the S-ICD can provide temporary transcutaneous pacing immediately following a shock, it cannot provide bradycardia pacing therapy or antitachycardia pacing, so patients with indications for either of these modalities are currently not candidates for this device. The S-ICD system is compatible with bipolar pacing. In patients with unipolar pacemakers in whom the S-ICD is the preferred choice, screening must be carefully performed to be certain the unipolar pacing artifact is not sensed by the S-ICD.

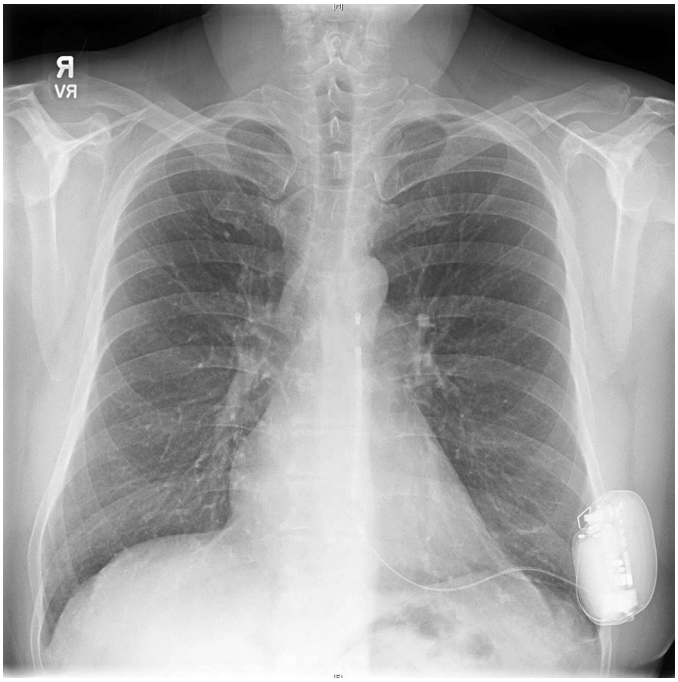
Preparation: Electrocardiographic Screening

Unlike the transvenous ICD detection of intracardiac electrograms, the cardiac far-field signal detected by the S-ICD is similar to a surface electrocardiogram (ECG) lead. Patients being considered for S-ICD implantation must be screened by a three-channel surface electrocardiogram that mimics the placement of the S-ICD generator and lead to ensure that the R-wave/T-wave ratio for appropriate sensing is satisfactory in at least one sensing vector. The S-ICD lead can be positioned on either side of the sternum, although the left parasternal position is preferred. If the patient fails screening in this position, then screening can be performed in a right parasternal lead position. The patient must pass the screening in the same lead performed both supine and standing. Some implanters also routinely screen using exercise testing to assess for changes in T-wave amplitude, which may occur during exercise, especially in patients with hypertrophic cardiomyopathy or rate-related bundle branch block.

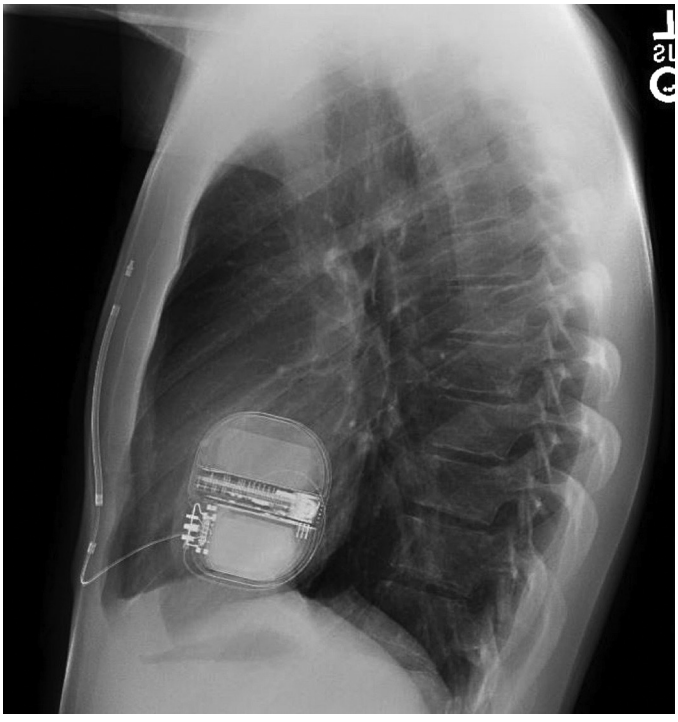
Operative Strategy

Anesthesia

Unlike standard transvenous device implants commonly performed with local anesthetic and conscious sedation, many centers use monitored anesthesia with deep sedation or general anesthesia for S-ICD implantations due to the uncomfortable positioning necessary for the procedure, discomfort of lateral chest wall pocket formation, lead tunneling, and testing defibrillation efficacy.



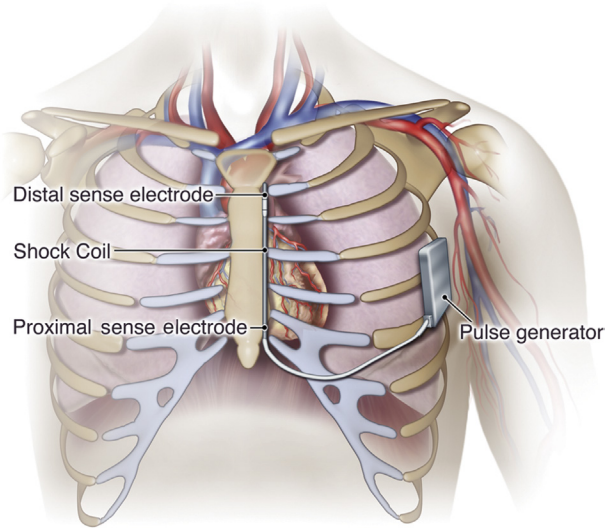
• **Fig. 10.1** Posterior-anterior chest radiograph of S-ICD generator and lead.



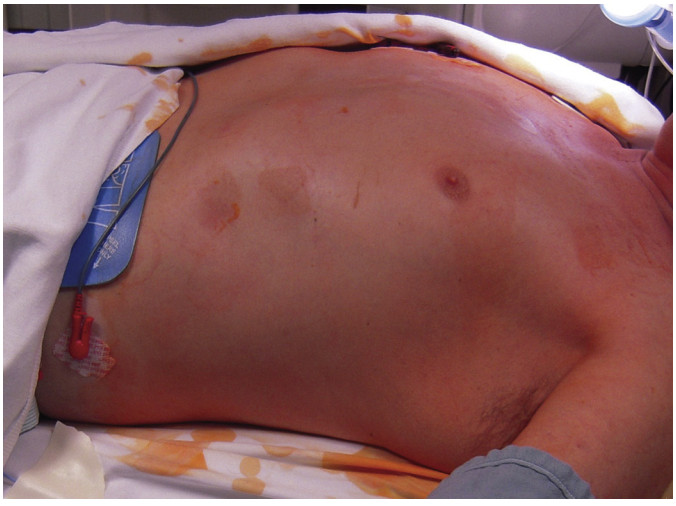
• **Fig. 10.2** Lateral chest radiograph showing S-ICD generator and lead in place.

Patient Preparation

Positioning monitoring electrodes and two sets of defibrillator pads is more challenging with the extensive exposure required for S-ICD implantation (Fig. 10.4). Chest electrodes should not be incorporated within the surgical field. Positioning one set of defibrillation pads on the low left-lateral chest and right scapular areas and the second set from the left scapular area to the right-lateral chest has provided adequate defibrillation success when required.

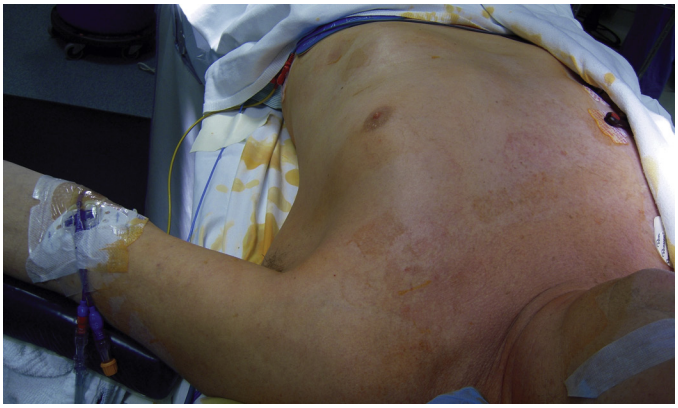


• **Fig. 10.3** Illustration of S-ICD system showing distal sensing electrode, proximal sensing electrode, shock coil, and pulse generator.

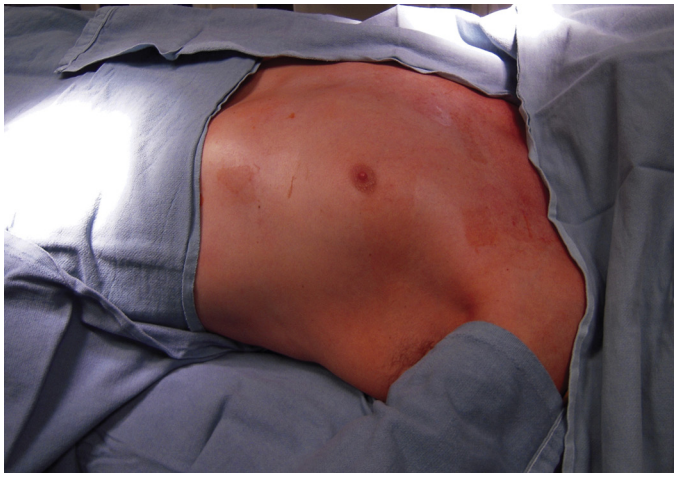


• **Fig. 10.4** External defibrillation pad placed lower than traditional device implantation location to avoid surgical field.

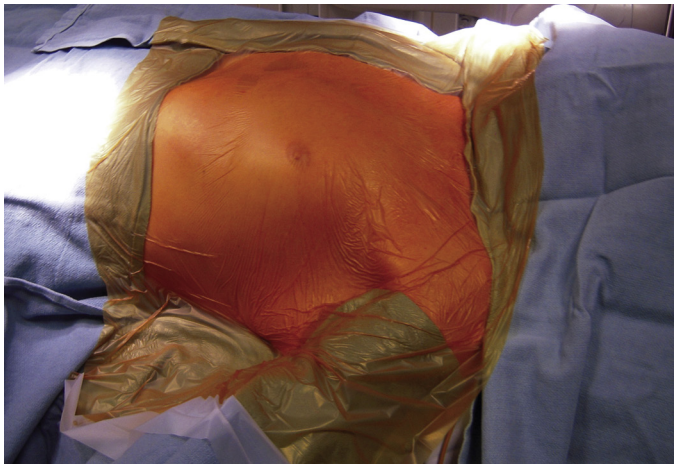
Suitable patient positioning on the procedural table is an important determination of success, particularly for avoiding postoperative infections. The patient is placed supine with the left arm adducted between 45 and 90 degrees to provide access to the lateral chest wall. Adduction beyond 90 degrees may place undue traction on the brachial plexus, resulting in brachial plexus injury, and should not be done. The area of skin preparation is wider than for standard transvenous device implantations, encompassing both sides of the sternum from the suprasternal notch to 2 to 3 cm below the xiphoid process and extending from the contralateral nipple line on the right chest to the posterior axillary line on the left (Figs. 10.5 to 10.8).



• **Fig. 10.5** Surgical field prep. Note arm on protective gel and abducted at 45 degrees.



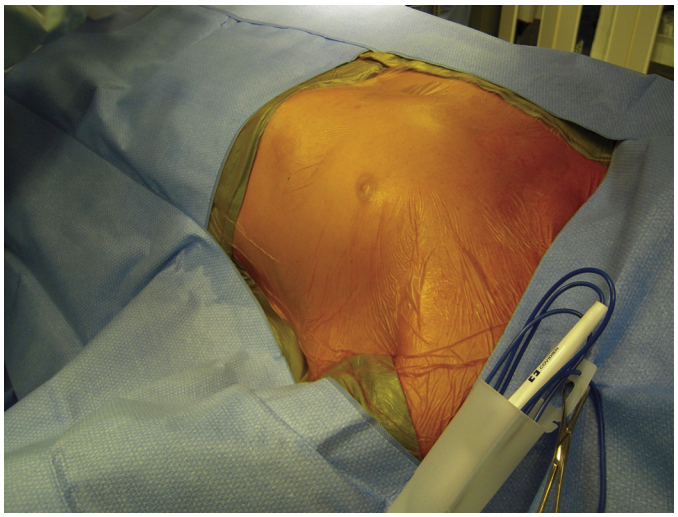
• **Fig. 10.6** Surgical field prepped with chlorhexidine and sterile towels.



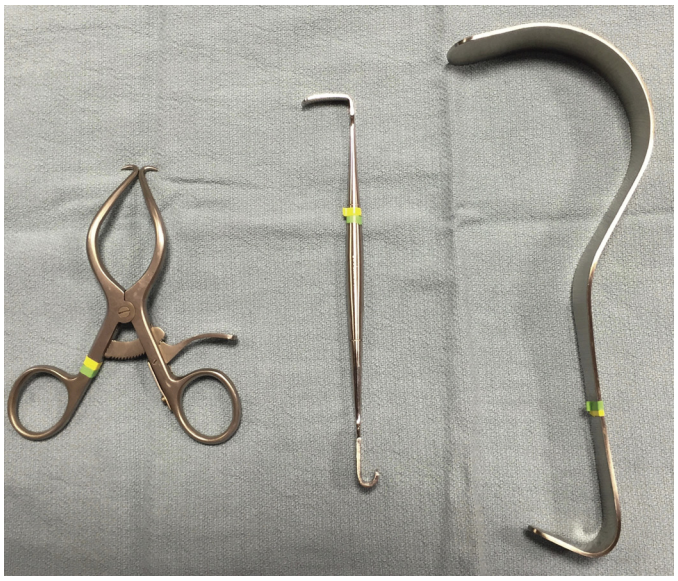
• **Fig. 10.7** Iodine-impregnated occlusive barrier placed over sterile drapes.

Surgical Instruments

The S-ICD requires surgical instruments similar to those for transvenous device implantation; however, a few additions to the surgical table can improve exposure and efficiency (Fig. 10.9). For the generator pocket, a small Deaver retractor can provide



• **Fig. 10.8** Universal drape placed over iodine-impregnated occlusive barrier to complete preparation of surgical field.



• **Fig. 10.9** Retractors useful for S-ICD implantation. From left to right, Gelpi, Senn, and small "baby" Deaver.

better exposure for dissection than a conventional Army-Navy retractor, particularly in the obese patient. Cerebellar retractors (small or large) may also be beneficial for exposure in the obese patient or in cases where breast tissue obstructs the surgical field. For the small paraxiphoid incision, a Gelpi retractor can be useful as the incision is often too small for a Weitlaner retractor, and a Senn retractor can provide additional exposure if an assistant is present to help with retraction and exposure.

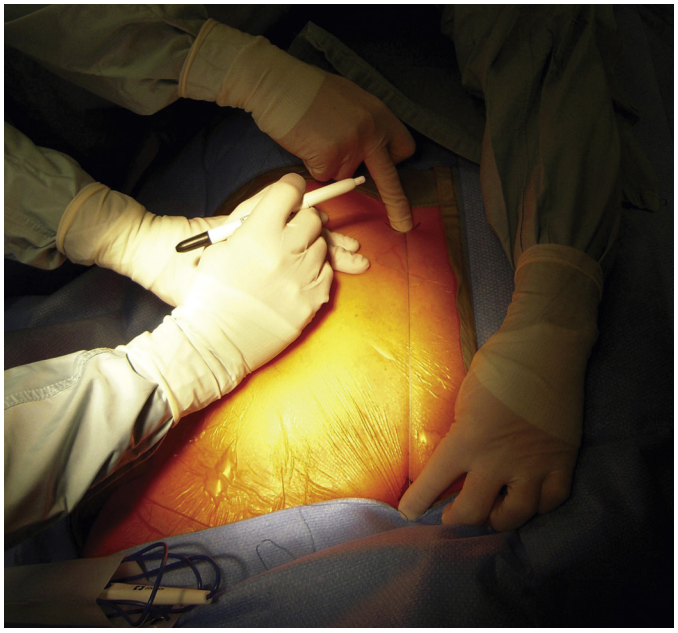
Operative Technique

The S-ICD system is designed to be correctly positioned using anatomical landmarks; however, fluoroscopy may be beneficial in cases of unusual anatomy or extreme obesity or when sternal wires are present. The sensing electrodes in particular should not be in contact with sternal wires. A good history and physical examination should be performed to identify chest

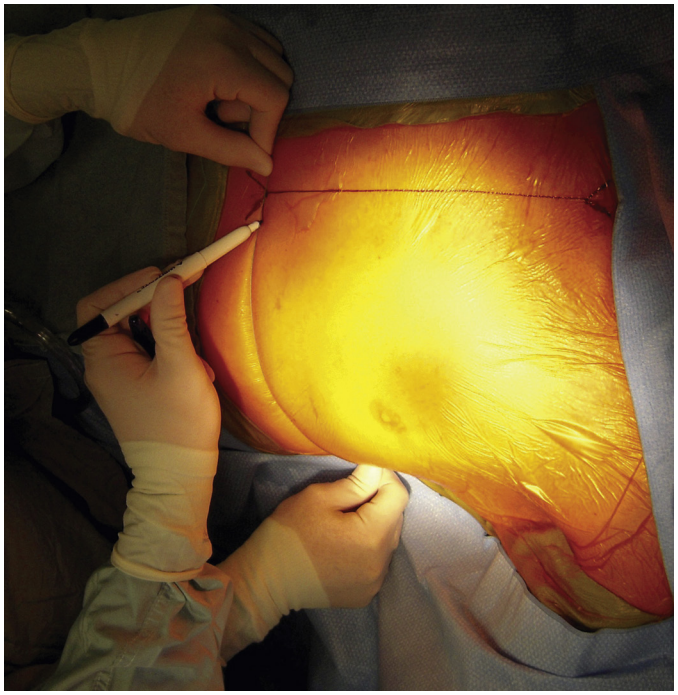
wall anomalies as noted earlier, or for the presence of breast implants or surgically treated breast cancer. A review of a pre-implantation chest radiograph is important to ensure that there are no unexpected anatomical features.

Once the patient is appropriately positioned, prepped, and draped, the chest should be marked for anatomical landmarks and proposed incisions using a sterile indelible marking pen on the incisional drape. A line drawn in the midline of the sternum from the suprasternal notch to the xiphoid process is useful as a parallel guide when tunneling the lead. Similarly, a perpendicular line extending from the xiphoid process to the lateral chest wall is useful to identify the inferior margin for the S-ICD generator pocket (Figs. 10.10 and 10.11).

The xiphoid process is a reliable, central landmark for S-ICD implantation. For patients who have had a previous median sternotomy, it is important to note that the xiphoid may be absent and the junction of the costophrenic angle should be identified as the most caudal aspect of the sternal border.



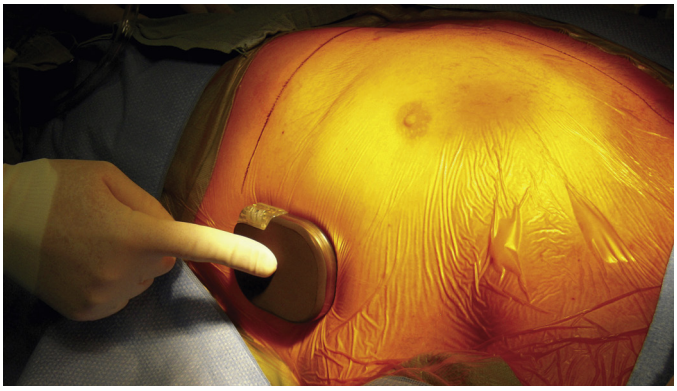
• **Fig. 10.10** Using suture ligature to guide marking of the center of the sternum from xiphoid process to suprasternal notch.



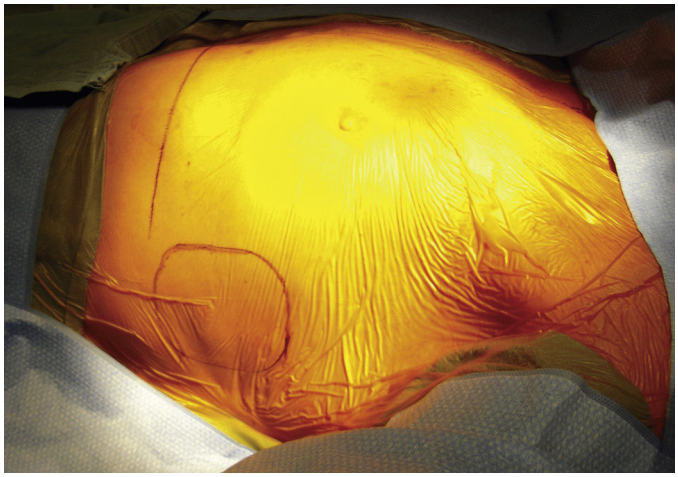
• **Fig. 10.11** Perpendicular line using suture ligature from xiphoid process to posterior axillary line to demarcate inferior border of S-ICD generator.

Generator Placement

Once the anatomical lines are drawn, the generator is placed on the patient's chest in the midaxillary line with the inferior border of the generator at the perpendicular line drawn from the xiphoid. This position generally coincides with the fifth and sixth intercostal space. A spot fluoroscopic image with the generator held in place can help confirm its position relative to the heart. The generator should be positioned over the cardiac apical shadow (Fig. 10.12) such that the shock vector is maximized. The generator can then be traced with a marking pen to provide a reference for the pocket size and location



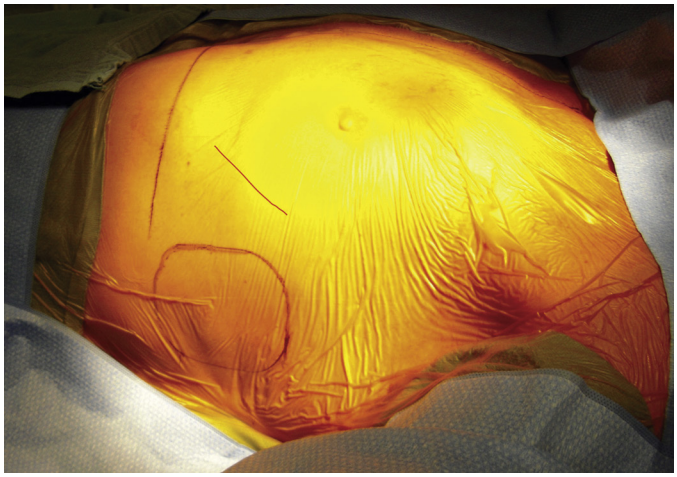
• **Fig. 10.12** Placement of generator on chest wall to determine final location using previously drawn line to mark inferior border of generator.



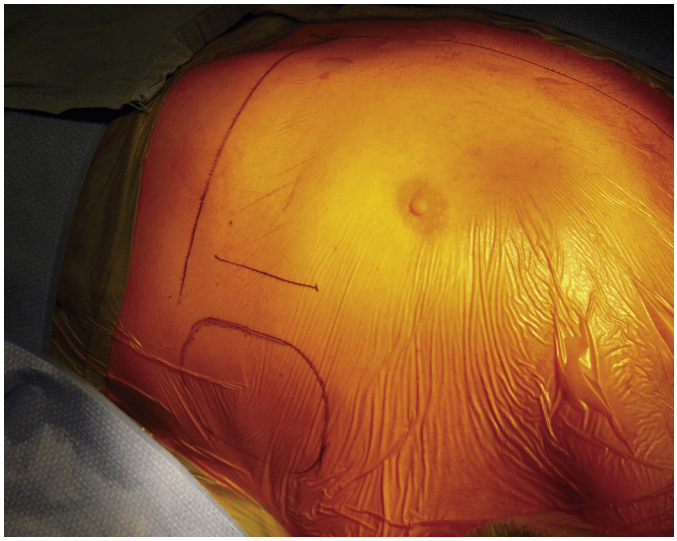
• **Fig. 10.13** Outline of generator traced to illustrate size and location of generator pocket.

(Fig. 10.13). The incision for generator placement may be made either in a curvilinear fashion inferior to the breast along the inframammary crease or horizontally (supine patient) at the anterior axillary line (Figs. 10.14 and 10.15). A horizontal incision provides excellent visualization of the chest wall when making the pocket, provides easier access to the generator and lead during generator replacement, and has an advantage in women by avoiding contact with an underwire bra, which can irritate the inframammary incision in the early stages of healing. The inframammary incision may have a preferred, cosmetic appeal.

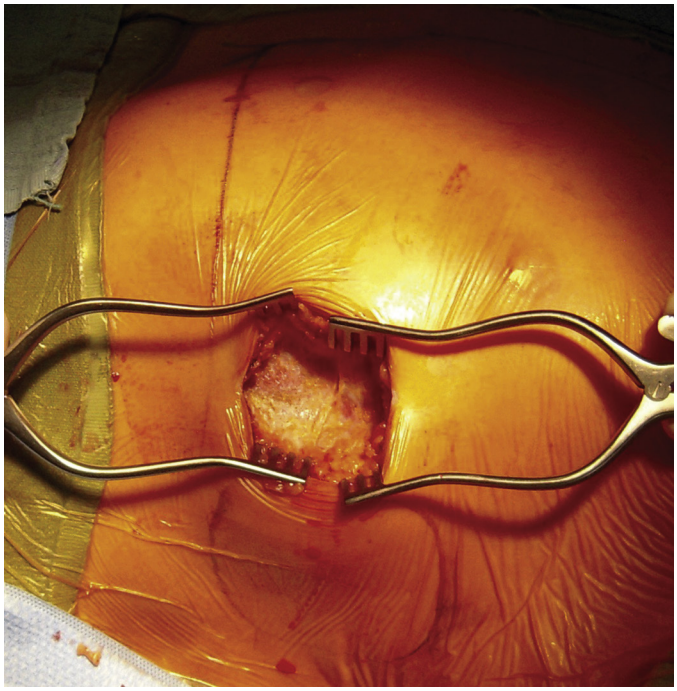
Local anesthetic is injected along the marked incision lines, an incision is created, and dissection is carried down to the fascial layer using electrocautery or a plasma knife. The pocket is created immediately upon the fascial plane and not within subcutaneous fat (Figs. 10.16 and 10.17). It is critical that dissection along the chest wall posteriorly remains on this fascial plane as the chest curves and the implanter could inadvertently dissect too superficially into the subcutaneous tissue. It is not unusual to encounter large dermal or perforating veins in this area; if encountered, they should be ligated with sutures to reduce the risk of bleeding and subsequent pocket hematoma formation. Hemostasis is otherwise achieved using electrocautery or a plasma knife comparable to pectoral device placement. The previously marked outline of the generator on the incisional drape is used as a guide to create an appropriately sized pocket (see Fig. 10.13). Care should be taken to avoid making the pocket too large, which would allow the generator to rotate parallel or perpendicular to the chest wall or become



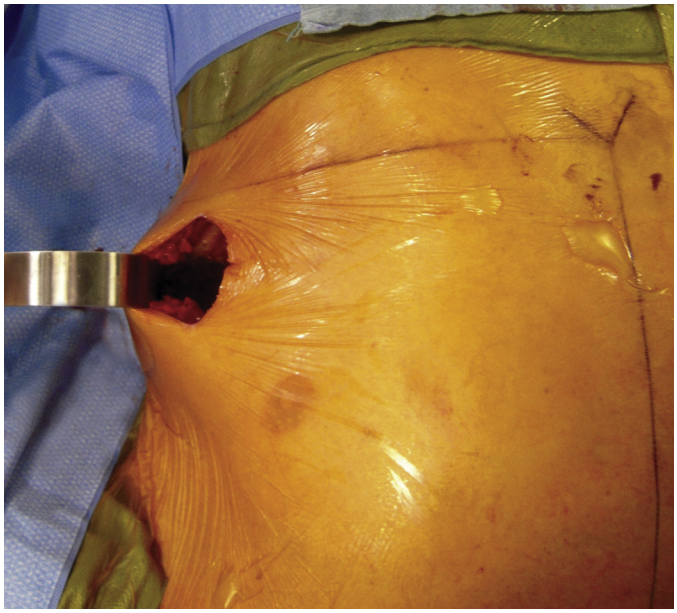
• **Fig. 10.14** Curvilinear inframammary incision location.



• **Fig. 10.15** Anterior axillary line incision location.



• **Fig. 10.16** Generator pocket formed on fascial plane.



• **Fig. 10.17** Generator pocket formed on fascial plane.

displaced caudally or posteriorly. Migration of the generator can result in pain and reduced defibrillation efficacy. A bupivacaine-soaked gauze placed within the pocket while lead placement is performed may facilitate improved pain control. Some operators use liposomal bupivacaine in the generator pocket to extend local analgesia out to 48 to 72 hours.

Lead Placement

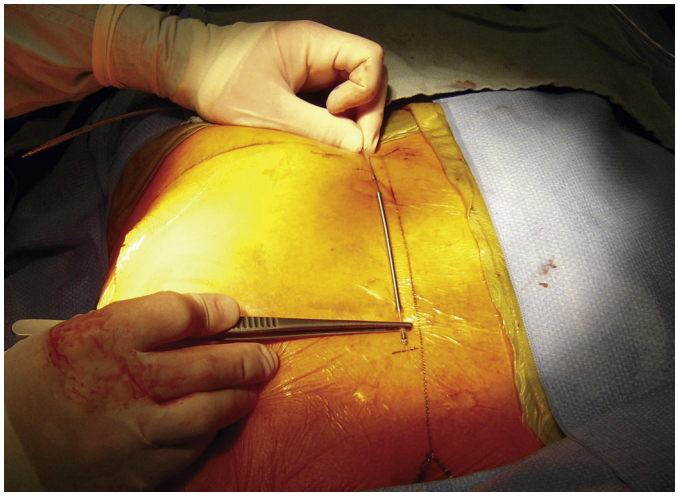
The incision site for the proximal lead anchor should be marked 1 to 2 cm lateral to the xiphoid. Care must be taken to manually confirm the location of the xiphoid or caudal border of the costophrenic angle in patients with previous median sternotomy. Fluoroscopy may be a useful aid in identifying these landmarks when the implanting physician is unsure of their exact location. The location of the incision should be over the sternum/chest wall and not over the epigastrium. Regardless of whether the lead is tunneled along the right or left side of the sternum, the coil will be positioned parallel to the sternum in the fascial plane (Fig. 10.18). Tunneling for lead positioning is accomplished with the tool provided.

There are two techniques for tunneling and securing the lead. The original implant described includes two parasternal incisions; a revised technique uses a single paraxiphoid incision.[1,5]

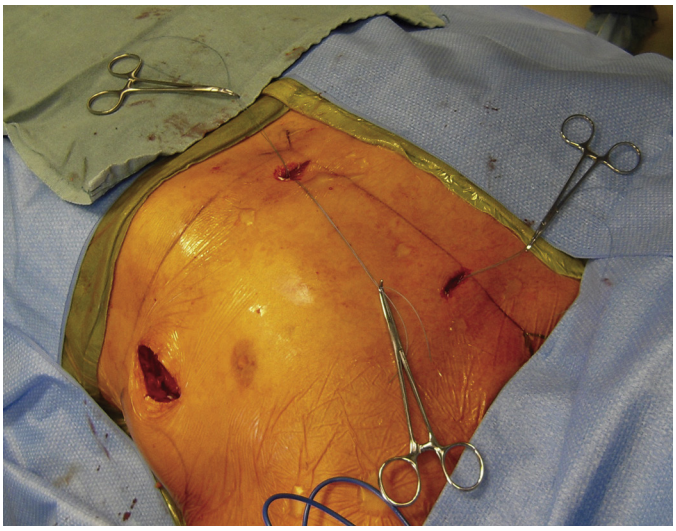
Three-Incision Technique

After injection of local anesthetic, a 2-cm incision is created 1 to 2 cm lateral to the xiphoid process, and a second 2-cm incision is made 1 to 2 cm lateral to the sternal angle, approximately 14 cm cranial to the xiphoid incision. These incisions are typically horizontal, but in particularly tall patients vertical incisions may allow for adjustments to the position of the lead's coil in relation to the heart. Dissection is carried down to the fascia with electrocautery for both incisions. A Gelpi retractor is used for exposure. To secure the anchoring sleeve, two nonabsorbable sutures are placed in the fascial layer in the paraxiphoid incision (Fig. 10.19). One or two nonabsorbable sutures are also placed in the deep fascial layer of the upper incision to secure the lead tip downward and prevent erosion. 0-TiCron sutures (Covidien) or similar sutures on a GS-22 needle will help facilitate suturing these small incisions. Gentle manual traction on the suture can help confirm that the suture was placed in the fascia and not subcutaneous fat.

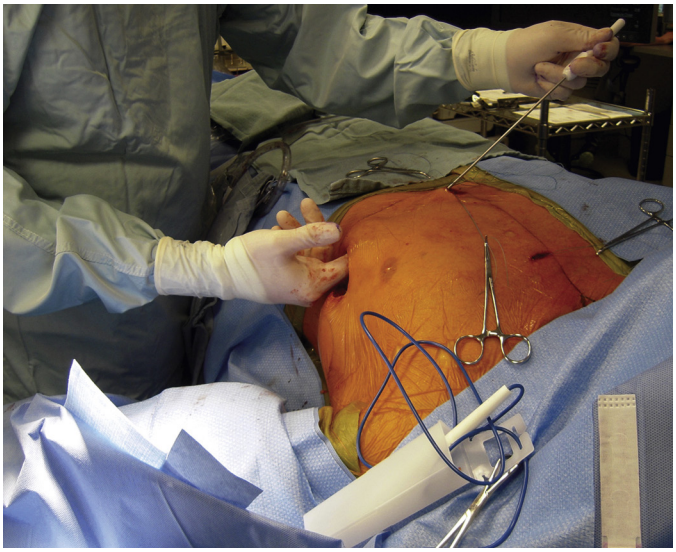
The tunneling tool is then inserted into the paraxiphoid incision with care to dissect on the fascial plane and advanced to the generator pocket (Figs. 10.20 and 10.21). A finger inserted in the generator pocket on the chest wall assists with proprioception as the tunneling tool is advanced such that the tool remains in the correct plane and enters the generator pocket. A long suture is then placed through the hole located at the tip of the tunneling tool and passed through the suture hole located at the tip of the lead. A 30-cm nonabsorbable ligature is used to ensure adequate



• **Fig. 10.18** Lead positioning to determine location of parasternal incisions.



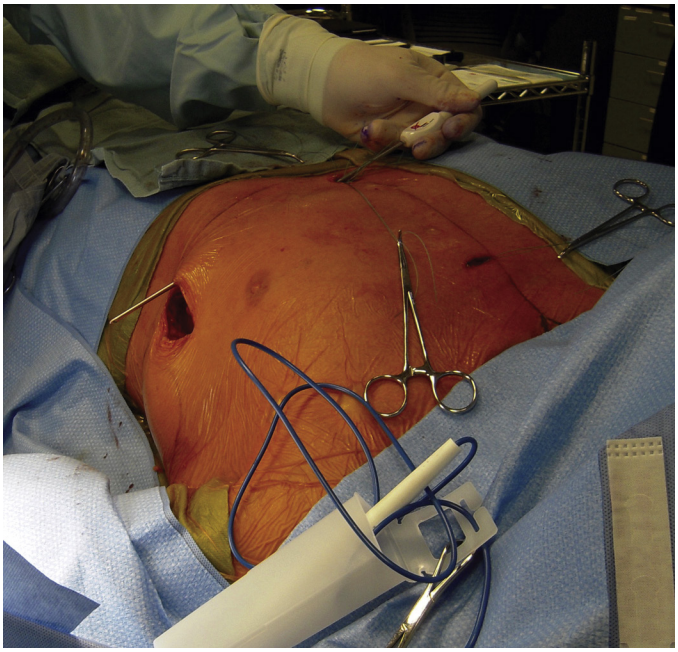
• **Fig. 10.19** Sutures on fascial plane of parasternal incision to secure lead anchoring sleeve following tunneling.



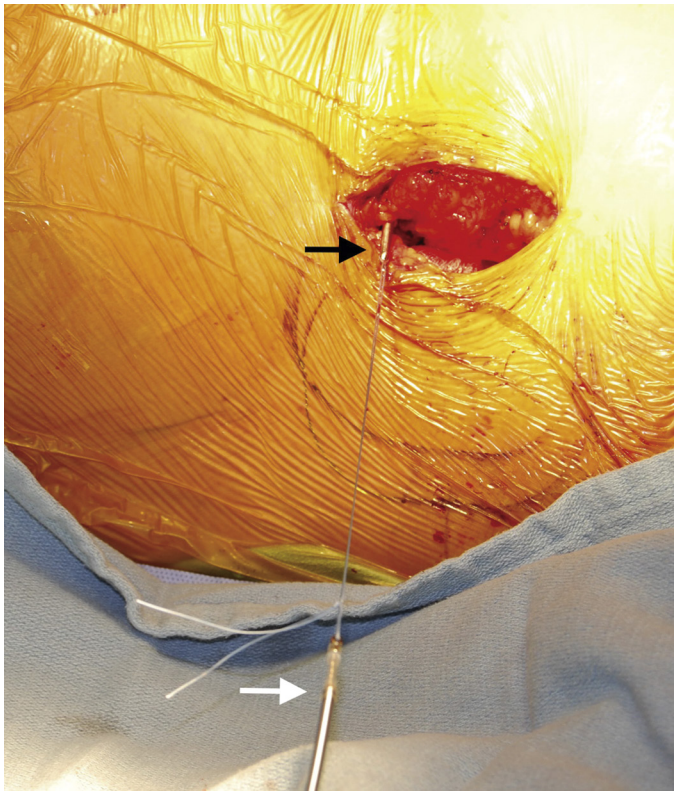
• **Fig. 10.20** Tunneling tool directed from parasternal incision to generator pocket.

suture length when tunneled. The two ends are then tied in a knot (Fig. 10.22). The tool, suture, and lead are pulled from the pocket to the paraxiphoid incision until 1 to 3 cm of lead is visible beyond the proximal sensing electrode (Figs. 10.23 and 10.24). A suture sleeve is then placed approximately 1 cm proximal to the electrode and secured with two (0-TiCron or similar) nonabsorbable sutures to prevent distal slippage of the suture sleeve, which could impair sensing (Figs. 10.25, 10.26, and 10.27). A tug test should be performed to ensure security of the suture sleeve attachment.

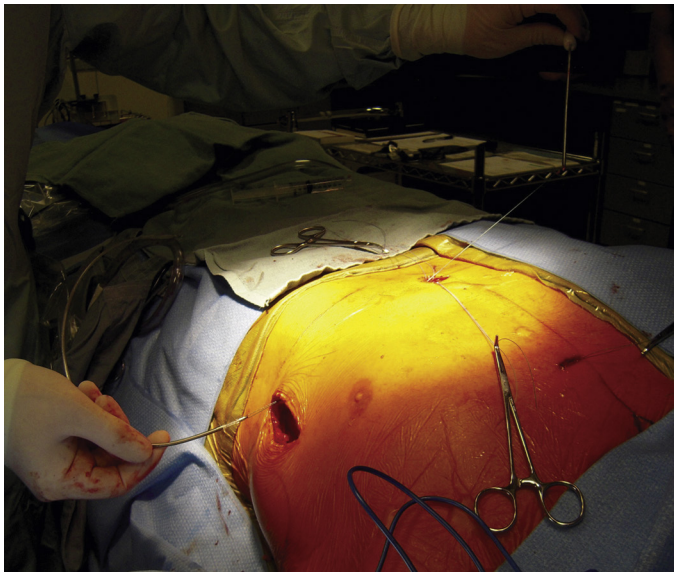
The tip of the tunneling tool is then placed in the paraxiphoid incision with the looped suture still attached. The tool is advanced with the handle held at approximately 30 degrees to the chest wall to ensure contact with sternal fascia (Fig. 10.28). When the distal tip is visible at the superior parasternal incision, the suture is grasped and cut from the tunneling tool (Fig. 10.29). The tunneling tool is removed and the suture that is still attached to the lead is used to pull the lead up through the tunnel until the tip of the lead is visible. The suture is removed



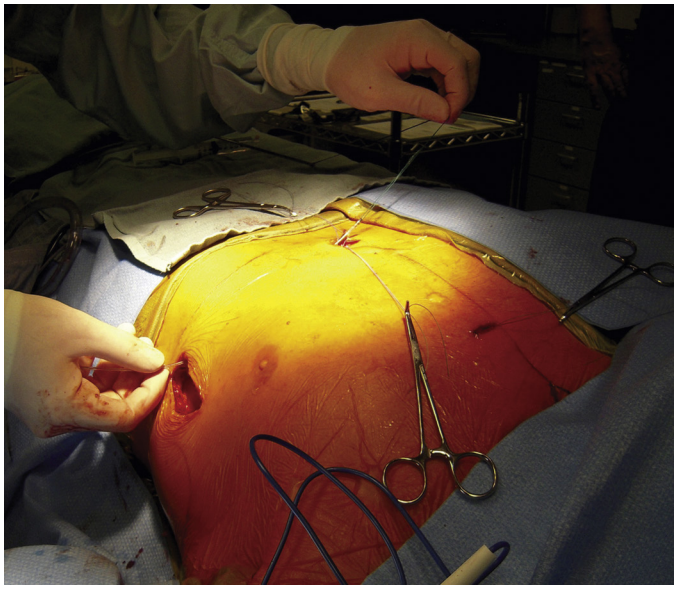
• **Fig. 10.21** Tunneling tool exiting generator pocket.



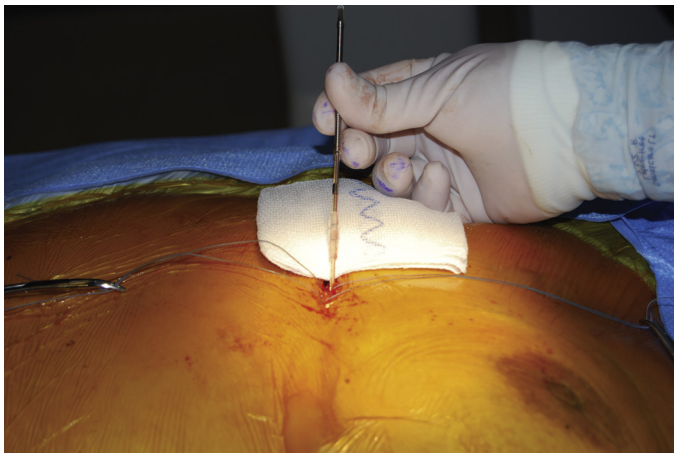
• **Fig. 10.22** Distal tip of lead *(white arrow)* connected to tunneling tool *(black arrow)* with ligature before tunneling.



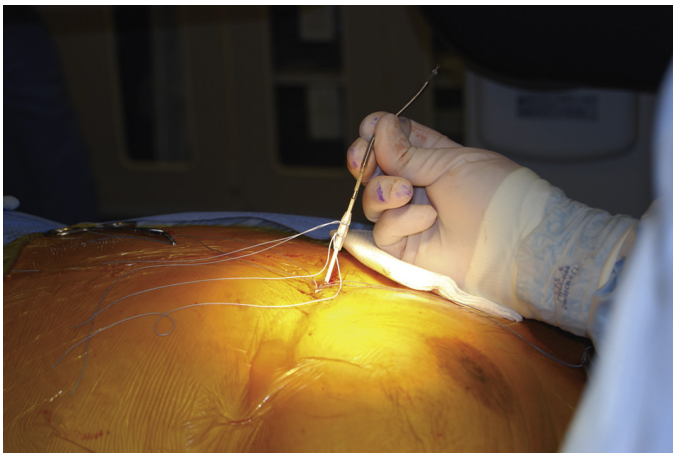
• **Fig. 10.23** Tunneling tool withdrawn from paraxiphoid incision with ligature attached.



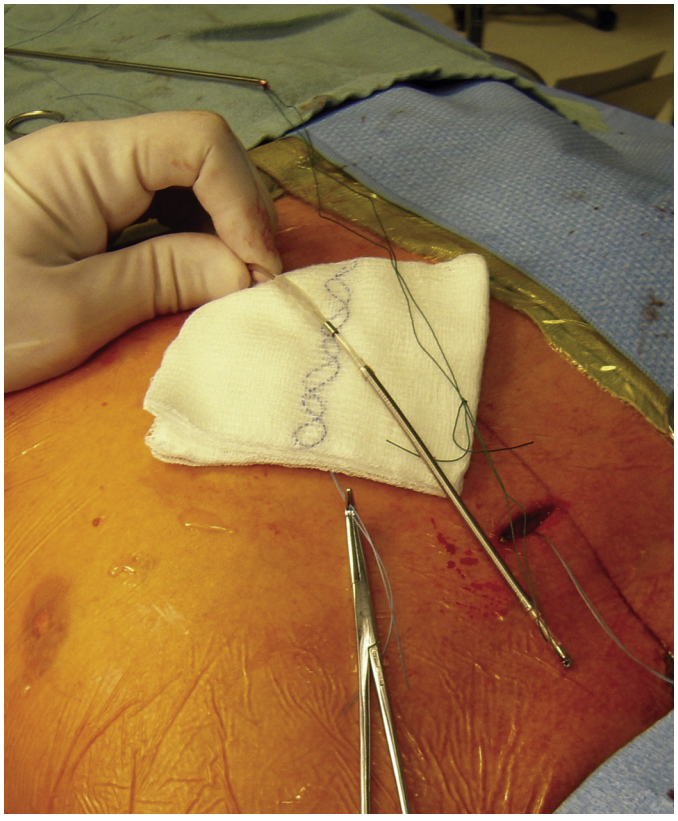
• **Fig. 10.24** Ligature used to pull lead through tunnel, exiting from paraxiphoid incision.



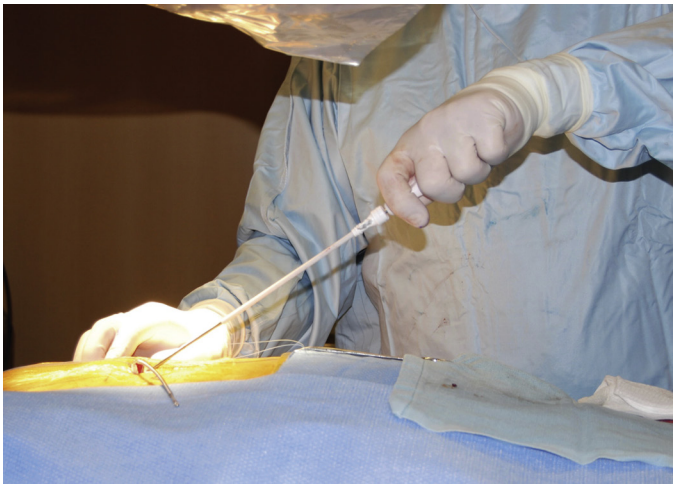
• **Fig. 10.25** Suture sleeve placed 1 cm from proximal sensing electrode. Fascial sutures placed on fascia beneath paraxiphoid incision are also illustrated.



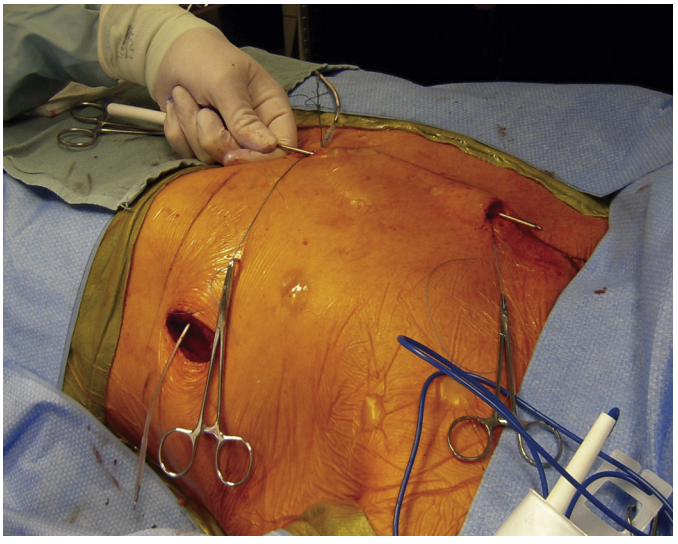
• **Fig. 10.27** Suture sleeve secured to lead with two nonabsorbable ligatures before parasternal tunneling.



• **Fig. 10.26** Suture sleeve in relation to proximal sensing electrode.



• **Fig. 10.28** Tunneling from paraxiphoid incision, seen from contralateral side of implant. Note the angle of the tunneling tool in relation to the chest wall (sheath inserted for a two-incision approach).



• **Fig. 10.29** Tunneling tool inserted through paraxiphoid incision, exiting at sternal angle incision.

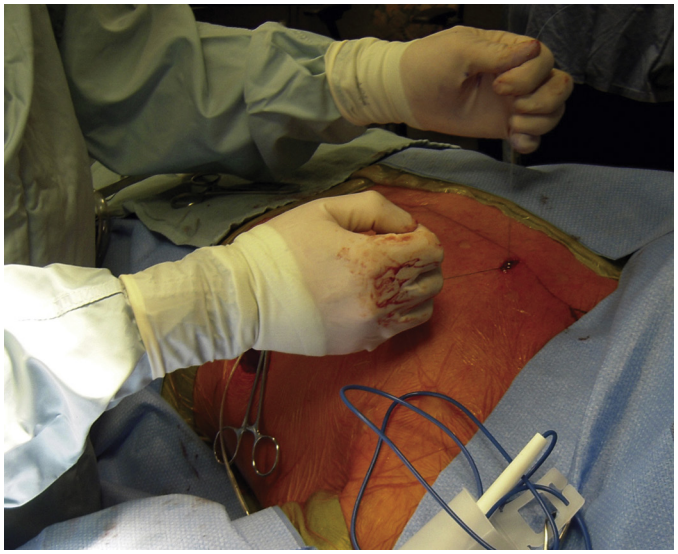
and the lead secured using the previously placed anchoring ties (Fig. 10.30).

The parasternal incisions and pocket are flushed with antibiotic solution. Firm pressure is applied over the length of the tunneled lead to express residual air that can otherwise result in sensing malfunction (Fig. 10.31). Bupivacaine can be injected into the tunnel from either or both parasternal incisions before closure. Note that the lead tip should not extend beyond the sternal angle. In pediatric and small adult patients with a sternum length less than 14 cm, redundancy must be

added to the lead using a C- or S-shaped tunnel to accomplish this (Fig. 10.32).

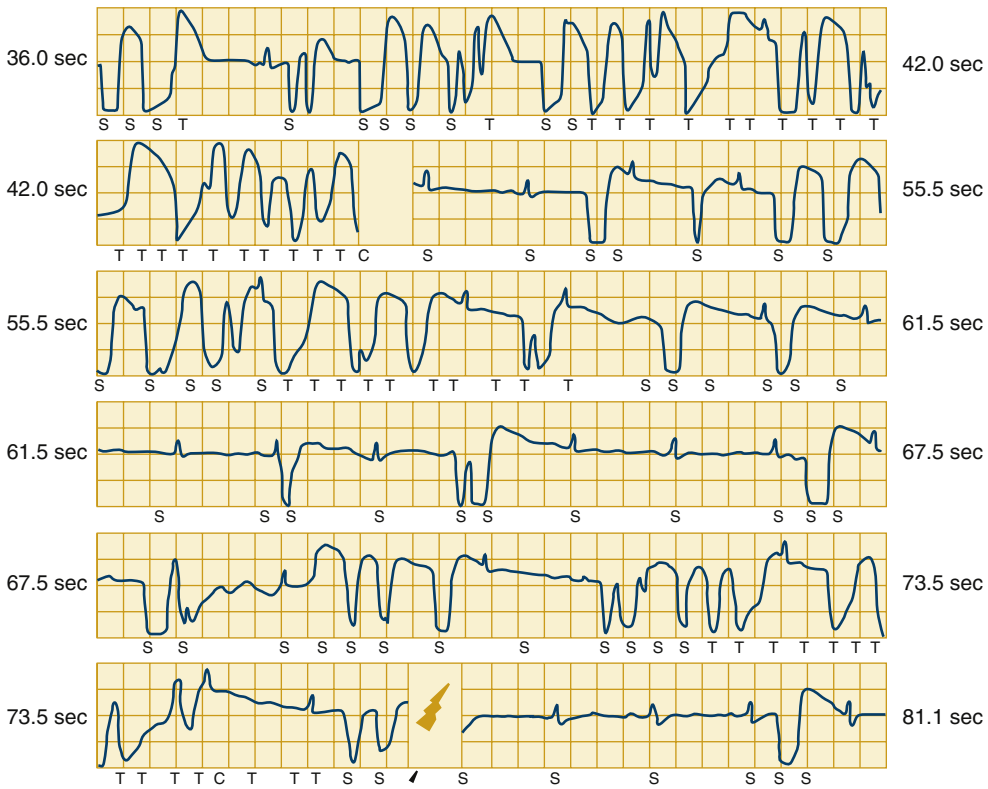
Two-Incision Technique

The superior parasternal incision has been noted to be a source of potential infection and discomfort, and can be cosmetically unappealing.[6] These concerns led to the development of a two-incision technique that takes advantage of the security of the xiphoid anchoring sleeve and eliminates the superior incision.[7] Like the original technique, the two-incision approach does not require fluoroscopy and uses anatomical landmarks. The paraxiphoid incision is unchanged from the three-incision technique; the tunneling tool is used to pull the lead from the generator pocket to the paraxiphoid incision and the suture sleeve is secured as described earlier. The superior incision is not performed; however, it is helpful to measure 14 cm above the xiphoid incision to mark on the incisional drape the superior aspect of the tunnel.

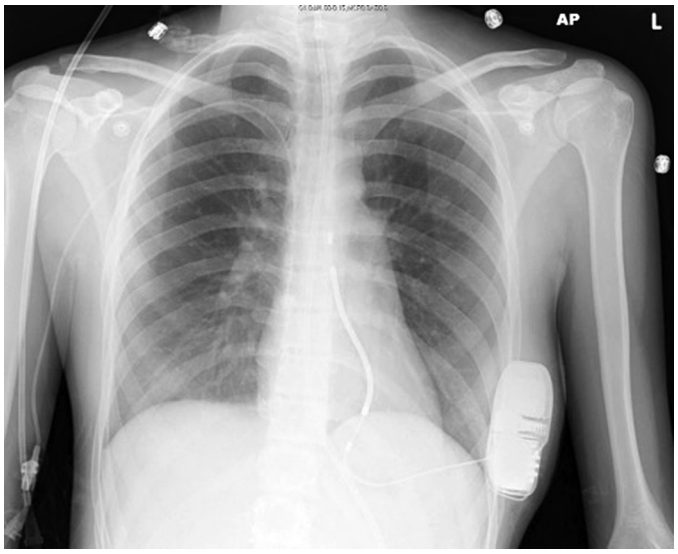


• **Fig. 10.30** Tunneling tool removed, and distal tip of lead anchored to sternal fascia.

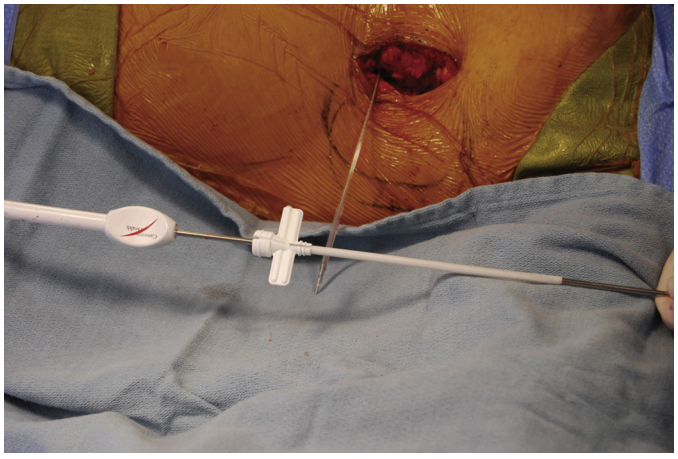
After the lead is tunneled from the generator pocket to the paraxiphoid incision, the looped suture is cut and removed from both the tunneling tool and the lead. An 11- or 12-Fr peel-away hemostatic sheath is placed over the tunneling tool (Fig. 10.33), and the tip of the tool is placed in the paraxiphoid incision and tunneled superiorly to the mark at the sternal angle (Figs. 10.34 and 10.35). As with the three-incision technique, a 30-degree angle relative to the sternum is required to ensure that the tunnel is maintained at the fascial layer. During this process, the sheath often does not advance as distally as the tool owing to tissue drag, but it can then easily be advanced over the tunneling tool until fully inserted. The tunneling tool can then be removed, leaving the sheath in place (Fig. 10.36). The lead is inserted into the sheath and advanced until the lead's suture sleeve reaches the hemostatic valve of the sheath. The sheath is peeled away, leaving the lead in the correct position. If an assistant is present, it is helpful to have the lead held with forceps where the lead exits the



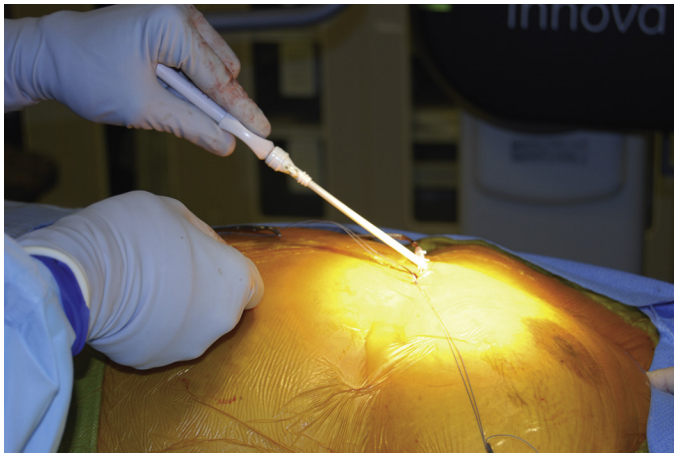
• **Fig. 10.31** Device interrogation revealing inappropriate shock from air within the S-ICD generator pocket.



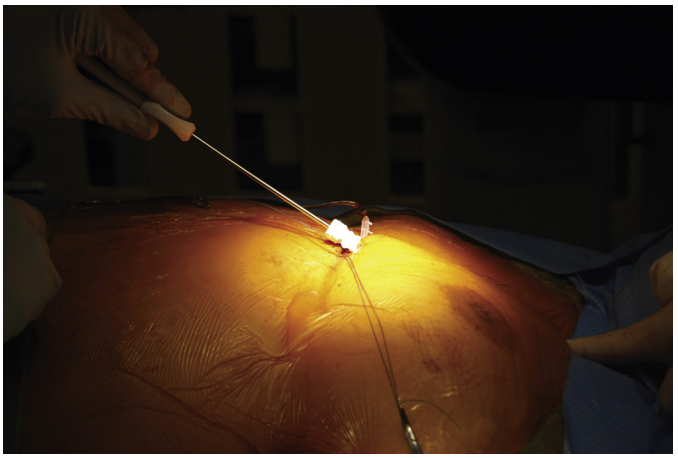
• **Fig. 10.32** Adapting lead length in short-statured patient via an S- or C-shaped configuration when tunneling.



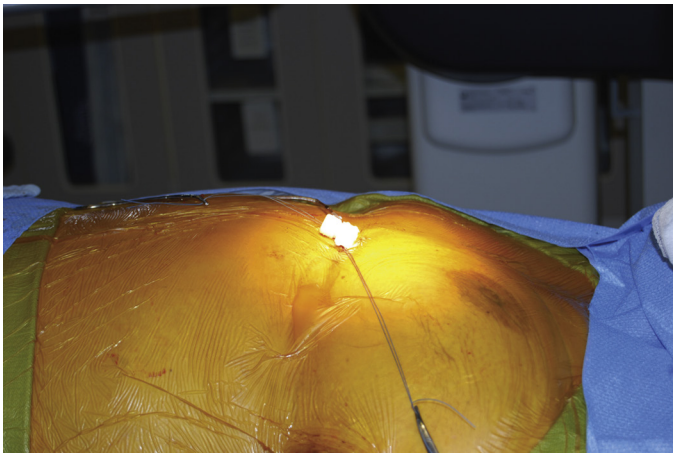
• **Fig. 10.33** Hemostatic tear-away sheath placed over tunneling tool for two-incision technique.



• **Fig. 10.34** Tunneling tool with hemostatic sheath inserted into paraxiphoid incision for parasternal tunneling in two-incision technique.



• **Fig. 10.35** Hemostatic sheath advanced over tunneling tool following parasternal tunneling.



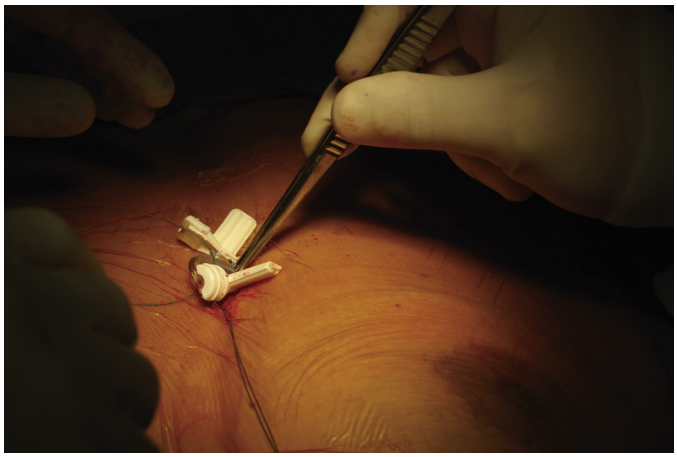
• **Fig. 10.36** Hemostatic sheath in place with tunneling tool removed.

hemostatic sheath, with forward pressure held during sheath removal to avoid dislodgement (Fig. 10.37). The suture sleeve is secured to the paraxiphoid fascia with the previously placed nonabsorbable sutures. The tunnel and pocket are flushed as previously described.

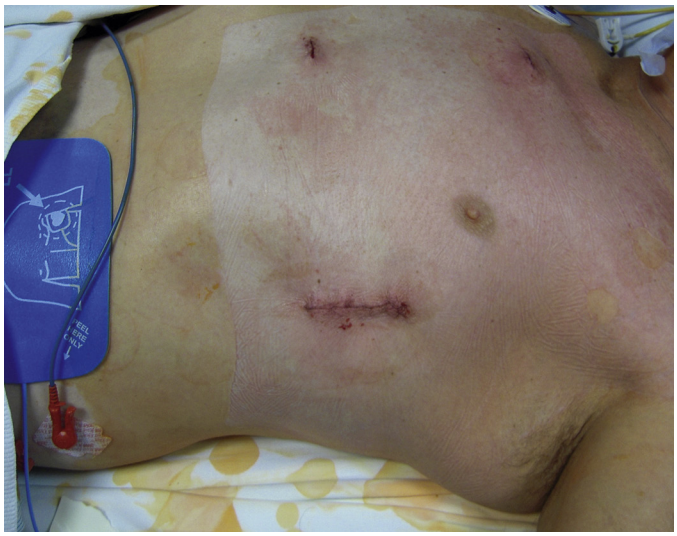
Physicians may want to perform the original three-incision technique when first learning to implant the S-ICD until they achieve comfort with S-ICD implantation. The three-incision technique can help maximize the success in appropriate lead placement and positioning.

Closure

The proximal tip of the lead is inserted into the S-ICD header and the set screw is deployed. A manual tug confirms appropriate connection. The bupivacaine-soaked gauze is removed if it had been previously placed, before the pocket is flushed. The generator is placed in the pocket with any excess lead curled gently between the serratus anterior muscle and the device. The generator is secured with nonabsorbable sutures using the two suture holes present in the header block to maximize fixation of the device to the chest wall.



• **Fig. 10.37** Lead inserted into hemostatic sheath, with DeBakey forceps used to hold lead in place as sheath is split and removed.



• **Fig. 10.38** Wound closure following three-incision technique.

The sternal incisions are closed with two layers (interrupted or running) of absorbable suture and a subcuticular layer. The generator incision is closed with sufficient layers to eliminate dead space and tension on the surrounding tissue (Fig. 10.38).

Additional Considerations

Potential sources of inappropriate shocks related to the surgical implant include the following:

Subcutaneous air: It is important to flush the generator pocket and tunneling tract carefully and evacuate any trapped air. Trapped air around the proximal and distal sensing electrodes has been reported to cause inappropriate shocks in the immediate postoperative period.[8,9] Air can sometimes be visualized on postoperative radiograph.

Prior cardiac surgery: Electrical interference due to contact between sternal wires and the sensing electrode has been reported as a cause of inappropriate shock.[10] Intraoperative fluoroscopy is helpful to ensure lead placement distant from the sternal wires. If the lead position is located on the right parasternal border, removal of the inferiormost sternal wire may be considered.

Unusual Anatomic Presentations

Many patients exhibit anatomical complexity that creates challenges in optimal S-ICD system placement. Care must be taken in patients with pectus excavatum or carinatum when tunneling the lead to ensure the tunneling plane remains on the fascia and does not drift into subcutaneous tissue, particularly at the superior aspect of the tunnel, which slopes downward in most patients.

Morbid obesity and poor tissue turgor from age or chronic disease can result in disruption of the generator anchoring sutures. This resultant displacement of the generator can cause chronic pain and alter the shocking vector. In patients with very poor tissue strength, the generator can be placed within an antibiotic pouch to allow for a more secure attachment to the underlying muscle by suturing the pouch and the generator header to the chest wall. Patients with congenital heart disease are best implanted with fluoroscopic guidance as the cardiac silhouette may vary.

Patients who have had extensive chest wall surgery and/or reconstruction may not be suitable candidates for S-ICD implantation and should be considered on a case-by-case basis.

Periprocedural Evaluation and Defibrillation Testing

Most implanters of the S-ICD perform defibrillation testing at implantation unless there are contraindications to induction of ventricular fibrillation or shock. Ventricular fibrillation is induced with 50-Hz stimulation, and the first shock is programmed at 65 J. In the event of defibrillation failure, polarity is reversed and a second shock is delivered at 80 J. Failure of defibrillation can be remedied by ensuring positioning of the lead and the generator directly on the fascial layer, with the generator at the midaxillary line or even more posterior.[11] Emerging data suggest an anterior generator location and sub-coil fat are associated with increased defibrillation threshold.[12] If a shock impedance is noted to be more than 100 ohms, the lead or generator may be incorrectly positioned in the subcutaneous tissue and not on the fascia. Fluoroscopy should be used at this point to look at the location of the generator and shock coil in relation to the heart to discern if an appropriate defibrillation vector could be improved by generator or lead repositioning.

Programming

The S-ICD was developed to be simple to program. Detection of ventricular arrhythmias is programmable using either a single- or a dual-rate zone. In single-zone programming, only a rate threshold is used to determine shock delivery. In the dual-zone configuration, the S-ICD uses a stepwise algorithm in the lower of the two zones (the "conditional zone") to classify the rhythm. The discrimination algorithm has been very reliable,[13] and it is strongly recommended that patients be programmed with dual zones to reduce inappropriate shocks.[14] Programming is usually set with a conditional zone between 200 and 250 beats/min and a shock zone of 250 beats/min or greater. The S-ICD does not have a monitor-only zone.

Conclusion

The S-ICD is an entirely novel defibrillation system that allows therapy of life-threatening ventricular arrhythmias without requiring a transvenous or intracardiac component. Implant complication risks are lower compared with the standard transvenous ICD, but early experience revealed excess infection risk, lead dislodgement, and inappropriate shocks due to T-wave oversensing. These early difficulties have largely been overcome by careful attention to surgical skin preparation, adding an anchoring sleeve at the xiphoid incision, and refining the sensing algorithm. Although many of the surgical techniques used at S-ICD implantation overlap with those of transvenous devices, the procedure requires additional skills in tunneling and knowledge of anatomical landmarks.

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11

Device Implantation in Pediatrics and Congenital Heart Disease

FRANK CECCHIN

Introduction

Cardiac implantable electrical device (CIED) implantation can be a challenge in pediatrics and in those with congenital heart disease. It is technically possible, even in the smallest child. For those with complex congenital heart disease, special considerations and techniques are required. Challenges that distinguish children from adults include small body size, need for long-term therapy, differences in venous capacitance and venous access, and unusual anatomic variations.

Loop Recorders

Loop recorders are a valuable diagnostic tool for the evaluation and management of rhythm disorders in children.[1] It can be difficult to secure an accurate history regarding syncope and arrhythmia-related symptoms in the young. Cognitive development may be such that accurate reporting of prodrome or associated symptoms, such as palpitations, is not possible. In infants, it is not possible to obtain a sound history; therefore episodes of altered consciousness are often considered an acute life-threatening event (ALTE). For symptomatic children or infants with an ALTE, and those without symptoms but at high risk of an arrhythmia (e.g., long QT syndrome type 3 with a QTc >500 ms), loop recorders have been used as an important monitoring tool.[2] Loop recorders can be implanted in the smallest infant; however, general anesthesia may be required. Most are implanted along the left side of the sternum, but in those with small body size, good signals can be obtained in an infraclavicular or axillary position.

Pacemaker and Implantable Cardioverter-Defibrillator Implant in Pediatrics (Normal Anatomy)

Pacemaker Implantation

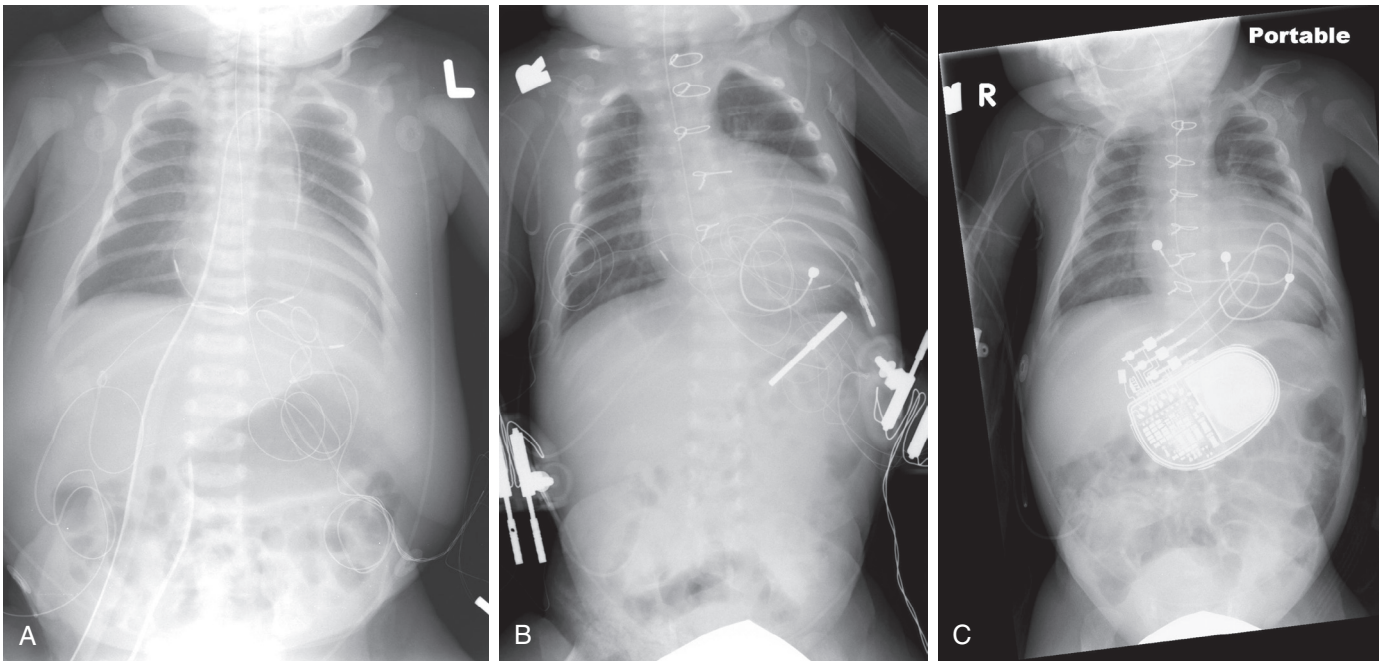
For a child with normal cardiac anatomy requiring permanent pacing, it is important to remember that this individual may require lead implants for more than 70 years. Technically, transvenous pacing systems can be performed in newborns, but the effect on vascular capacitance and the tricuspid valve need to be considered.[3] Therefore, the first implant in a young child is often an epicardial system with transition to a transvenous system after he or she has grown. Even though epicardial lead implantation requires sternotomy, thoracotomy, or a subxiphoid approach and is associated with higher chronic stimulation thresholds, higher lead failures and fractures, and shorter battery life, venous access is preserved for future use.[4–6]

In the premature newborn, temporary epicardial wires can be placed, and the child can be paced with an external pacemaker for weeks or months until he or she is large enough for a permanent system (>2 kg in body weight; Fig. 11.1).[7] Epicardial pacing with temporary wires is preferred over transcutaneous pacing since the latter can cause serious skin burns in infants.[8] The first system implanted should be epicardial with a left ventricular apical lead placed via left thoracotomy.[9–11]

Unless ventricular function is severely impaired, a single unipolar ventricular lead is all that is needed in a newborn. It is best to use suture-on steroid-eluting leads. Limiting the implanted hardware in the pocket and chest is important since it may never be removed. The shortest lead length should be used. In a newborn, a 15-cm unipolar lead often suffices (see Fig. 11.1). Excess lead should be placed in the pocket under the device.

If excess lead remains in the pericardial space, it should not wrap around the heart since cardiac strangulation can occur as the child grows.[12,13] The pulse generator is preferably placed in an abdominal subrectus pocket. A median incision is made along the linea alba with lateral dissection to the rectus sheath (Fig. 11.2). Careful dissection of the rectus sheath from the rectus muscle helps maintain the integrity of the peritoneum.

The pacemaker pocket is made below the rectus muscle but above the sheath. Preserving the rectus muscle protects the device and maintains abdominal musculature architecture and function. If the rectus sheath is compromised, then the device is at risk for migration into the peritoneal cavity. The device should always be palpated during follow-up and, if peritoneal migration is suspected, an abdominal lateral radiograph can confirm that the device remains within the rectus sheath. If the



• **Fig. 11.1** Staged cardiac resynchronization therapy in a newborn with complex congenital heart disease. Chest radiographs of a newborn with tricuspid atresia, D-transposition of the great arteries, congenital complete heart block, aortic arch hypoplasia, and severe left ventricular dysfunction who underwent staged repair and pacing owing to acute kidney and hepatic injury at presentation. (A) On the second day of life the newborn underwent bilateral pulmonary artery banding and temporary multisite ventricular pacing. (B) Two weeks later that was followed by removal of the branch pulmonary artery bands, dilation of the branch pulmonary arteries, enlargement of the ventricular septal defect, atrial septectomy, internal pulmonary artery banding, and aortic arch repair and then permanent posterior lead placement on cardiopulmonary bypass. (C) Ten weeks later temporary wires were removed and a permanent multisite pacemaker was implanted with a unipolar anterior lead and an atrial lead added to the previously implanted posterior lead.

pacemaker has migrated to an intraperitoneal position, then revision is needed since serious damage to abdominal organs can occur.[14] If a subrectus pocket is not possible, the pacemaker can be placed in an extrapleural intrathoracic pocket.[15] If a unipolar system is used, a pneumothorax will result in loss of capture.

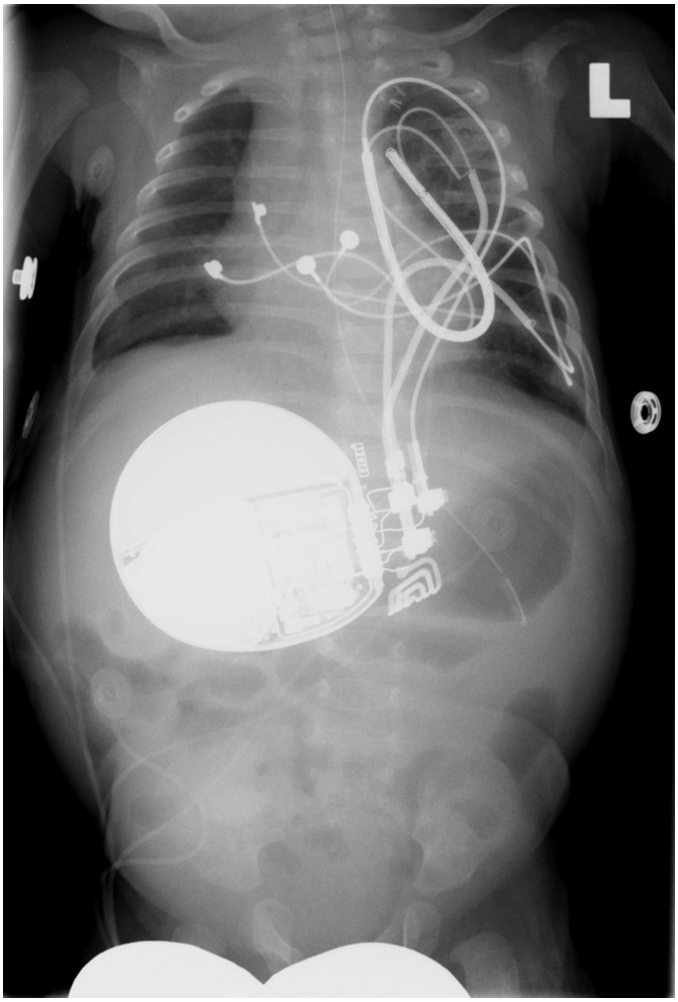
As the child grows, the lead will develop tension and can fracture.[4,5] Before that occurs, a decision needs to be made regarding whether the epicardial system should be revised or switched to a transvenous system. If the epicardial system is abandoned, then the pacemaker should be removed and the leads capped.

There is no exact age regarding when a child is ready for a transvenous system. However, a minimum body weight of 10 to 15 kg for a single-chamber system and 20 to 25 kg for a dual-chamber system is a general recommendation followed by most pediatric implanters. The implanter should use leads with the smallest possible diameter. Lumenless active fixation leads are the leads of choice since they take up the least amount of space and are easier to extract versus conventional leads.[16] This lead is a bipolar, steroid-eluting (lipid-soluble beclomethasone), 4.1 Fr lead that offers better atrioventricular valve integrity and venous development than a larger lead.[17] It lacks a hollow core, has an exposed helix, and uses composite polyurethane/silicone insulation.

At implantation, a separate, slice-away, 8.4 Fr diameter steerable catheter system can be used. However, in a small child, implants can be placed via a shaped delivery sheath provided by the manufacturer or any soft-tipped sheath with a 5 Fr or greater inner diameter that can be split.[18] The lead can be placed either in the right ventricular apex or, preferably, at the midventricular septum. The right ventricular outflow tract should be avoided.

Vascular access can be obtained via the subclavian, axillary, or cephalic veins. There are no data in children to support one approach over another. The implanter should use the technique with which he or she has the most experience and success. Subclavian crush is not common in children since they have more compliant ligamentous attachments compared with adults. In a growing child, a loop of excess lead should remain in the right atrium to allow for growth and prevent future lead stretch-induced dislodgement. Placing the lead on the ventricular septum is another way to prevent lead stretch, since the heart grows from the apex down.

The incision can be either infraclavicular or axillary. Some implanters use a double-incision technique with a small infraclavicular incision for lead placement and a larger axillary incision for device placement.[19] The device can be placed in a subpectoral (under the pectoralis muscle) or prepectoral (between the pectoralis sheath and muscle) or axillary pocket. In general, prepectoral location is best since a submuscular location will put more compression on the leads and device. However, some very small and/or thin children are at risk for



• **Fig. 11.2** Epicardial implantable cardioverter-defibrillator (ICD) placement in a newborn. Chest radiograph of a nontransvenous ICD system in a small infant with malignant long QT syndrome. This infant was 2 months old and 3.5 kg when she underwent epicardial placement of atrial and ventricular bipolar suture-on steroid-eluting pace/sense leads in conjunction with a superior vena cava (SVC) defibrillation lead in the epicardial space via a midline sternotomy. The SVC lead had a 5-cm electrode. The SVC lead was anchored distally to the posterior pericardium and seated in the lateral edge of the pericardial space. It is important not to wrap the lead around the heart since this can cause cardiac strangulation as the child grows. Excess lead was placed in the left pleural space.

erosion with a prepectoral location. Some children are very concerned about the cosmetic appearance and want the device concealed completely. In that situation, a subpectoral implant with a single axillary incision is the best approach.

In those with complete heart block, a single ventricular lead with capability of atrial sensing via an electrode embedded in the lead and ventricular pacing can be used in the VDD mode. The lead has a large diameter (8.1 Fr), so its use is limited to older children.

Implantable Cardioverter-Defibrillator Implantation

Despite advances in implantable cardioverter-defibrillator (ICD) technology, generators and leads remain too large for small children, so implantation using a standard endovascular technique is only possible in older children and adolescents. Depending on indications, patient preference, and body habitus, either a transvenous system or subcutaneous system can be used. Children heavier than 30 kg are candidates for the subcutaneous system.[20,21] ICD implantation in older children is similar technically to an implant in adults (see Chapter 8).

The subcutaneous ICD offers distinct advantages including simple and low-risk implantation, reliable lead longevity, low-risk extractions, and no risk of endocarditis. The disadvantages with the current-generation device, compared with transvenous ICDs, are no bradycardia or antitachycardia pacing, potential for T-wave oversensing, additional risk of erosion, shorter battery life, and greater delay in the detection of arrhythmias.[20] Many of these obstacles are likely to be addressed with future generations of this defibrillator system.

If a transvenous system is chosen, a single-coil pace/sense/shocking lead is preferable since they are easier to extract.[22–24] Standard implantation techniques are used for the older child (see Chapter 8). For a thin child or if there is heightened concern regarding cosmetics, the device can be placed in a subpectoral pocket with an axillary incision.

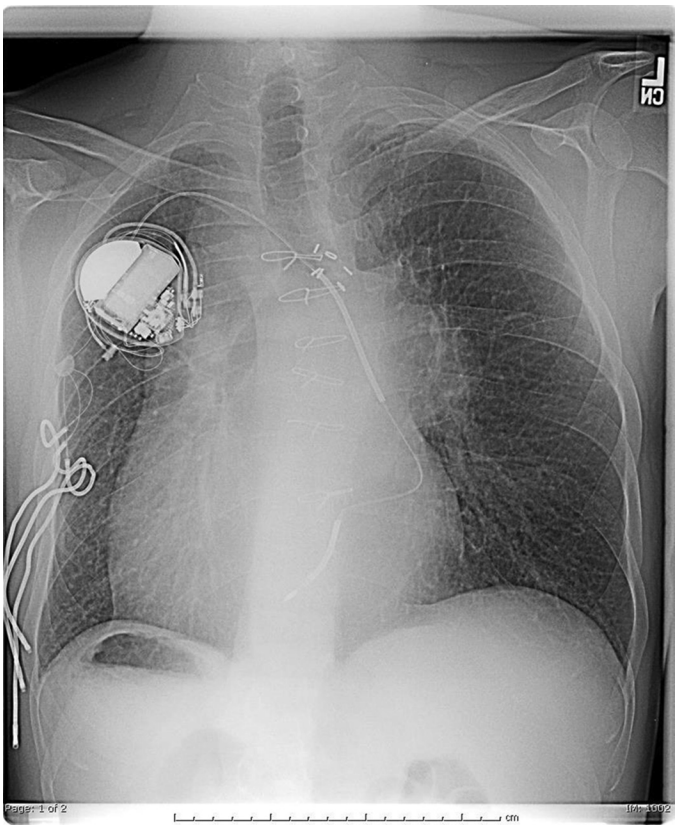
ICD implantation in small children (body weight <20 to 25 kg) is more challenging. They are too small for a transvenous or subcutaneous system, so an epicardial system is needed. Various techniques have been utilized, but for an infant weighing less than 5 kg, the most reliable system consists of a bipolar epicardial lead placed on the right ventricle to be used for pacing and sensing, in conjunction with a coil for defibrillation.[25–27] A coil (typically in the superior vena cava [SVC]) with 5 cm of electrode can be placed in the posterior pericardial space, creating a shock vector between the anteriorly positioned generator and posterior coil. The tip of the coil can be sutured to the pericardium just above the left ventricle and then anchored again near the apex of the heart (Fig. 11.3).

Alternative approaches have been devised that include a subxiphoid incision and pericardial placement of a transvenous ICD lead under fluoroscopic guidance in the pericardial space. A downside to this approach is that the leads are too long and take up valuable pocket space. Other options include pleural or posterior subcutaneous lead placement.[28–30]

The disadvantage of these nontransvenous types of systems is that there are more components that can fail. When compared to standard transvenous systems, they have a higher failure rate (3-year survival of 49% vs. 76%).[31] For larger infants and children, see the technique described in the next section for those with congenital heart disease.

Pacemaker and Implantable Cardioverter-Defibrillator Implantation in Congenital Heart Disease

Congenital heart disease adds complexity to device implantation. There are issues regarding venous access to chambers, venous obstruction, venous anomalies, cardiac position, cardiomegaly, comorbidities, high capture thresholds, and poor sensing owing to fibrosis and valve issues.



• **Fig. 11.3** Implantable cardioverter-defibrillator (ICD) placement in an adult with complex congenital heart disease. Chest radiograph of a hybrid ICD system in a 30-year-old with L-transposition of the great arteries/dextrocardia who presented with ventricular tachycardia and second-degree atrioventricular block. A dual-coil ICD lead was placed via the right subclavian vein into the left superior vena cava (SVC) via a bridging vein and anchored into a posterior right ventricle. The defibrillation threshold was high, so a right-sided three-finger subcutaneous array was placed and used in substitution for the SVC coil.

Pacemaker Implantation

For those with congenital heart disease, pacing can be required owing to congenital deficiencies in sinus or atrioventricular (AV) node dysfunction, postoperative heart block, progressive loss of sinus or AV node dysfunction from surgery, and/or poor hemodynamics or antiarrhythmic drug–induced bradycardia. In general, most individuals benefit from dual-chamber pacing, but there are individuals with pure sinus node dysfunction who only need atrial pacing. Ventricular demand pacing in those with congenital heart disease is not recommended.

Most children with congenital heart disease have had heart surgery. Therefore, epicardial systems are common and pacing must be factored into the surgical planning. Placing a posterior lead can be difficult from a midline sternotomy unless cardiopulmonary bypass is used. If pacing is anticipated, leads can be placed at the time of surgery with a pulse generator implant later (see Fig. 11.1).

Implanting a transvenous system in an individual with congenital heart disease requires a thorough understanding of the cardiac anatomy and surgical history. There may be synthetic septal patches, atrial baffles, conduits, obstructed venous channels, persistent left superior vena cava, and extensive surgical fibrosis. Computed tomography (CT) or magnetic resonance imaging (MRI) can help navigate the anatomy, potential vascular routes, and obstacles. However, old leads and other devices may cause artifact, limiting the image's diagnostic value. Venography at the time of generator implantation can help elucidate venous anatomy and is strongly recommended.

Once all background data have been reviewed and a procedural plan created, the next step is to review the venography. If the veins are partially obstructed, traversing the occluded veins should be attempted using floppy hydrophilic guidewires through stenotic areas followed by placement of sheaths. It may be be possible to perform a venoplasty to widen the tract. If the patient has SVC syndrome or arm edema, the vessel should be stented. When venous access is completely obstructed, the occlusion can be penetrated with a needle or dilator; otherwise, alternate routes such as supraclavicular access of the internal jugular vein or transhepatic, transatrial, transthoracic, transiliac, or transfemoral approaches are possible.[32–36]

Once access to the desired chamber is obtained, active fixation and lumenless leads are the leads of choice. A wide array of delivery sheaths allows for lead placement in the optimal location.[16–18] Sometimes a deflectable electrophysiology catheter can facilitate location of optimal pacing sites.

Pacing in Specific Anatomic Variants

In individuals with D-transposition of the great arteries or postoperative atrial switch, sinus node dysfunction is common. Surgical correction (Mustard, Senning procedures) involves creating an intraatrial baffle to direct venous return to the proper ventricular chamber, resulting in the left atrium becoming the systemic venous atrium. The atrial lead passes through the baffle and secures to the roof or posterior wall of the left atrium. The implanter should avoid placing the lead in the left atrial appendage, since phrenic nerve capture usually occurs in that position. In those in whom ventricular pacing is also indicated, the systemic venous ventricle is a morphologic, smooth-walled left ventricle. There is the possibility of systemic venous baffle stenosis in these patients, and there may be a need for venous stents to enlarge the atrial baffle concomitantly.[37]

Corrected transposition of the great arteries is associated with a high incidence of AV block (2% per year) from a superiorly displaced AV conduction system. The systemic venous ventricle is a smooth-walled morphologic left ventricle in which the apex is rotated rightward. The double-switch procedure is being used with increasing frequency for these children and, at the time of surgical repair, a lead can be placed on the posterior right ventricle for future pacing needs.[38]

The most complex anatomic variant to implant a pacing system into is for patients with a single ventricle and those whose status is post–Fontan operation. Of the latter group, 20% to 53% require pacing over the long term.[39,40] The type of Fontan operation performed determines which, if any, chamber can be approached by endocardial access.[41] Endocardial pacing has been performed in patients after the extracardiac conduit Fontan procedure, but it requires transhepatic puncture or atrial access via the superior vena cava to the right pulmonary

artery to the right atrium.[42] Otherwise, an epicardial approach is needed.

In the lateral tunnel Fontan procedure, atrial access can be obtained for pacing through the systemic veins. Both chambers can be accessed in the classic atriopulmonary form of the Fontan procedure. Access to the ventricle in the latter is achieved via a coronary sinus vein.[43] Dual-chamber pacing may require a hybrid approach with a transvenous atrial and epicardial ventricular lead. The sluggish venous pooling associated with this physiology increases the risk of thrombi, and anticoagulation is advised.[44] In tetralogy of Fallot, the anatomic arrangement is normal, but if severe tricuspid regurgitation is present, it may be difficult to stabilize and affix a ventricular lead across the tricuspid valve. A long peel-away sheath can help place the lead.

Anomalies of caval veins are common in those with congenital heart disease (5% to 10%). Situs anomalies are frequently associated with malformations of systemic venous return, but a left superior caval vein would be considered normal in situs inversus. SVC anomalies include connection of a right SVC to the left atrium and absence of the right caval vein with a persistent left superior caval vein (PLSCV) that connects to the right atrium, coronary sinus, azygos vein, or left atrium.

Right and left SVCs ("bilateral SVCs") represent 80% to 90% of these anomalies. About 90% drain into the coronary sinus. Alternative sites include the inferior vena cava, hepatic vein, or left atrium. Of those with bilateral SVCs, 30% have a bridging vein.[45,46] Thus, a left-sided implant is mandatory in at least one-third of patients for anatomic reasons. A left-sided implant using a persistent left SVC to the coronary sinus requires a longer lead or a long peel-away sheath since the leads tend to bunch up in the atrium[47] (Fig. 11.4).

Implantable Cardioverter-Defibrillator Implantation

The indications for ICD implantation in adult congenital heart disease were published in a 2014 consensus statement.[48] The same indications apply to children with congenital heart disease. The type of system used can be either transvenous, epicardial, subcutaneous, or hybrid. The choice depends on cardiac position, ventricular mass, pacing indications, body habitus, previous hardware, vascular access, intracardiac shunt, valvar competency, and whether concomitant heart surgery is needed. Before ICD implantation, venography, echocardiography, and either cardiac CT or MRI is needed to assess these factors.

Transvenous ICD implantation is straightforward in most patients with two septated ventricles and normal AV relationships. This includes tetralogy of Fallot, atrial septal defect, ventricular septal defect, and left-sided valvular disease. Standard implantation techniques used in adults with normal cardiac anatomy can be used (see Chapter 8). Right ventricular angiography before ICD lead placement can ensure lead placement in the true right ventricular apex.

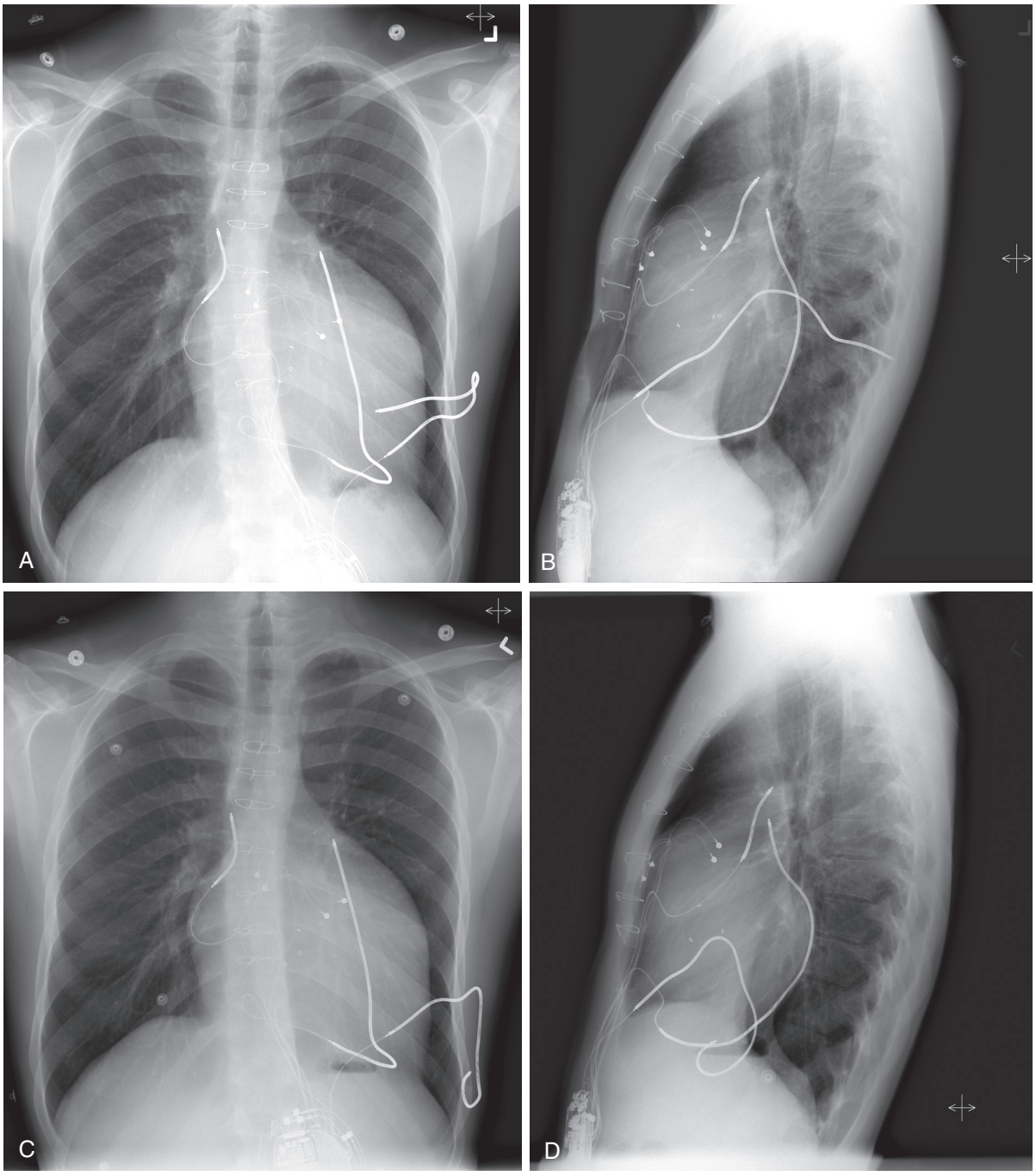
A subcutaneous ICD (S-ICD) is an excellent choice for many older children and adults with congenital heart disease who are not excluded owing to being pacemaker dependent, requiring biventricular resynchronization, or having sustained ventricular tachycardia episodes treated with antitachycardia pacing. The main advantage of the S-ICD is that it eliminates complications related to endocardial leads. The physical stresses exerted on the lead are fewer than for an endocardial lead because the lead is not subject to the repetitive motion from cardiac contraction.

As noted earlier, the primary limitation of this approach is that some patients will not benefit, including young children (<30 to 40 kg) and patients requiring antibradycardia or antitachycardia pacing. As with all patients receiving an S-ICD (see Chapter 10), a screening test is performed before implantation to confirm the existence of at least one vector obtaining an acceptable R-wave/T-wave amplitude ratio, both supine and upright at rest. I further recommend that all candidates also pass the screening test during exercise. If the patient fails the screening test, it is not advisable to implant the S-ICD because it is not possible to ensure proper functioning of the device. In a study specifically addressing this issue involving 30 patients with congenital heart disease, eligibility was 87% versus 100% in those with normal cardiac anatomy.[49]

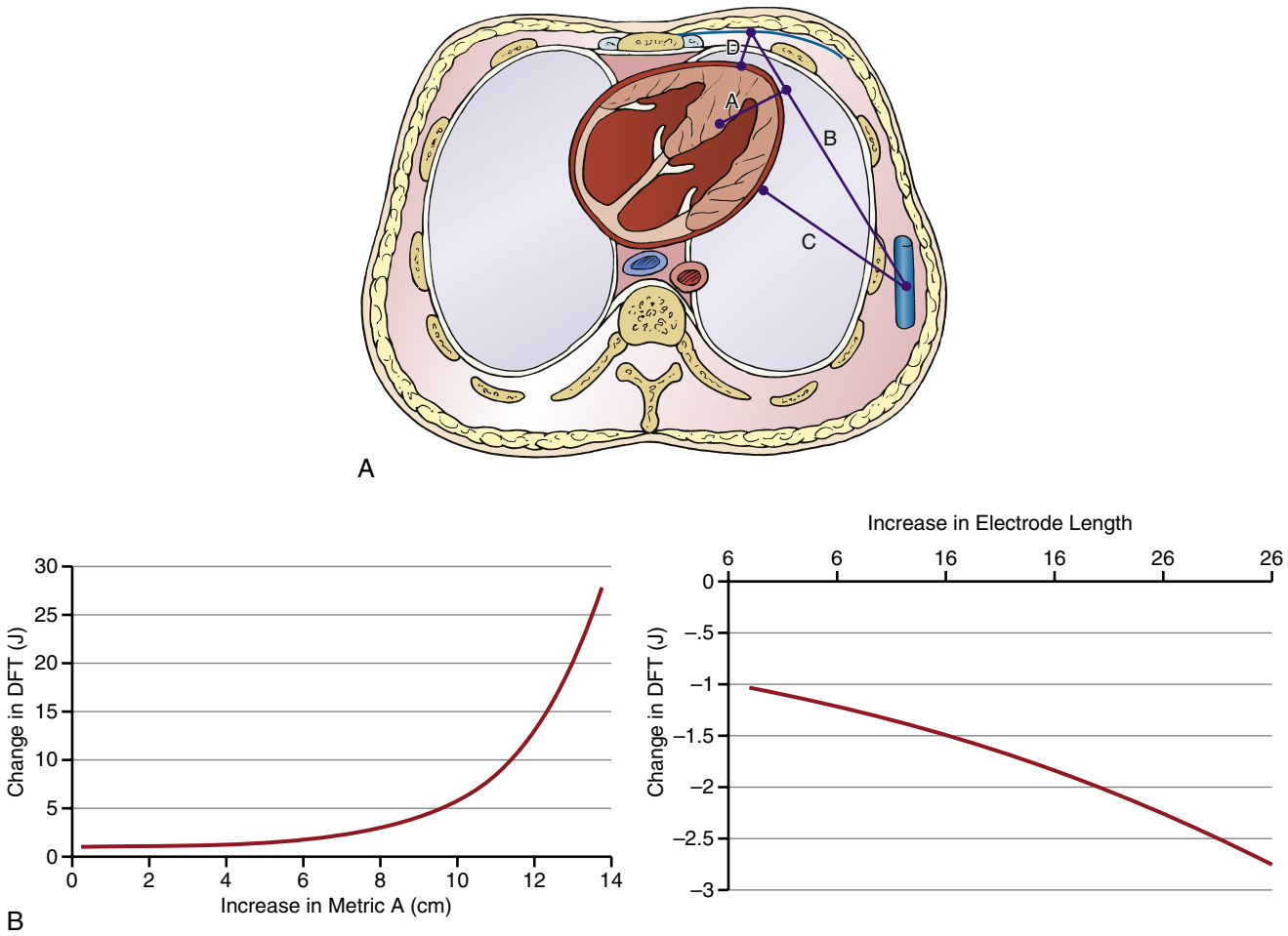
For individuals who are not candidates for a transvenous or subcutaneous ICD, the only option is a nontransvenous system composed of a rate-sensing lead attached directly to the epicardium and coils placed in the subcutaneous tissue and/or pericardial space.[50] Various configurations have been used beyond the newborn period for nontransvenous ICD systems.

Jolley et al.[52] used a finite-element model to predict defibrillation threshold (DFT) efficacy and risk of myocardial injury for different single- and two-electrode subcutaneous configurations using epicardial electrodes. The best configurations involved contralateral placement of electrodes. With their model, the two most important principles in lowering DFTs were placement of electrodes to align the interelectrode shock vector as closely as possible to the center of mass of the ventricular myocardium, and use of longer electrode coil lengths (Fig. 11.5). The length of the electrode can be either 5 cm (superior vena cava coil) or 25 cm (subcutaneous coil). However, it is possible to cut the 25 cm to any smaller length and cap the cut tip. According to the Jolley et al. finite model, placing the coil in the pericardial space will lower the DFT but may increase the risk of electroporation-induced myocardial injury from voltage gradients greater than 30 V/cm. This can be mitigated by using a coil length of 10 to 25 cm, depending on the size of heart.

When subcutaneous coils are used during transthoracic defibrillation in humans, only approximately 4% of the total current traverses the heart. For transthoracic defibrillation, only current traversing the myocardium has physiologic importance.[52] Because current flow through the thoracic volume conductor is determined by geometric factors as well as by the relative resistivity of the thoracic tissues, it is not unexpected that the DFT could change significantly when thoracic resistances are altered by clinical conditions such as pleural effusion, pneumothorax, or change in lung volume (Fig. 11.6). The lung volume could change if there is a change in heart size after significant ventricular remodeling in response to cardiac surgery or cardiac resynchronization therapy (CRT). Also, as



• **Fig. 11.4** Hybrid implantable cardioverter-defibrillator (ICD) placement in an adult with complex congenital heart disease. This 22-year-old with severe Ebstein anomaly presented with cardiac arrest, severe tricuspid regurgitation, and massive right ventricle enlargement. The patient underwent tricuspid valve replacement, bidirectional Glenn procedure, and implantation of an epicardial ICD. (A [anteroposterior], B [lateral]) In the immediate postoperative period he had a defibrillation threshold (DFT) of 35 J with the following configuration: a 5-cm electrode lateral to the superior vena cava, and a 25-cm electrode in the posterior pericardial space wired to a subcutaneous 25-cm electrode going from the lateral to posterior chest. (C [anteroposterior], D [lateral]) Due to the narrow safety margin, follow-up DFT testing was done and the DFT was greater than 40 J. It was evident from the chest radiograph and a chest computed tomography scan that the right ventricle had undergone such severe remodeling that the distance from the electrode and heart had increased significantly, which would increase the amount of current that is lost in the thorax and change the DFT. To change that distance, half of the lead was moved from the posterior thorax to a lateral position. This was done via a 1-cm incision adjacent to the middle of the coil and the pocket was never opened. The chest radiograph shows the new lead position. The DFT was then ≤30 J.



• **Fig. 11.5** Variables that affect nontransvenous implantable cardioverter-defibrillator placement. Jolley et al. used a finite-element model of defibrillation to test the effect of multiple variables on the human defibrillation threshold via subcutaneous electrodes. (A) Diagram of variable distances measured for each electrode and generator configuration. Metric A: Alignment of thoracic field with myocardium, measured as the distance of the center of mass of the heart from the line between generator and lead. Metric B: Minimum distance between generator and lead. Metric C: Minimum distance between generator and surface of heart. Metric D: Minimum distance between lead and surface of heart. (B) The variables and simulated defibrillation threshold (DFT) were used to create a multivariate linear regression model, which showed that variability in the predicted DFT was highly attributable to alignment of the electrode generator shock vector with the ventricular myocardium (metric A squared) and electrode length together. The graph on the left shows that the DFT increases as the distance of the center of mass of the heart to the leads and generator grows beyond 10 cm. The graph on the right shows that the DFT decreases as the electrode length increases. (From Jolley M, Stinstra J, Tate J, et al. Finite element modeling of subcutaneous implantable defibrillator electrodes in an adult torso. *Heart Rhythm.* 2010;7[5]:692–698.)

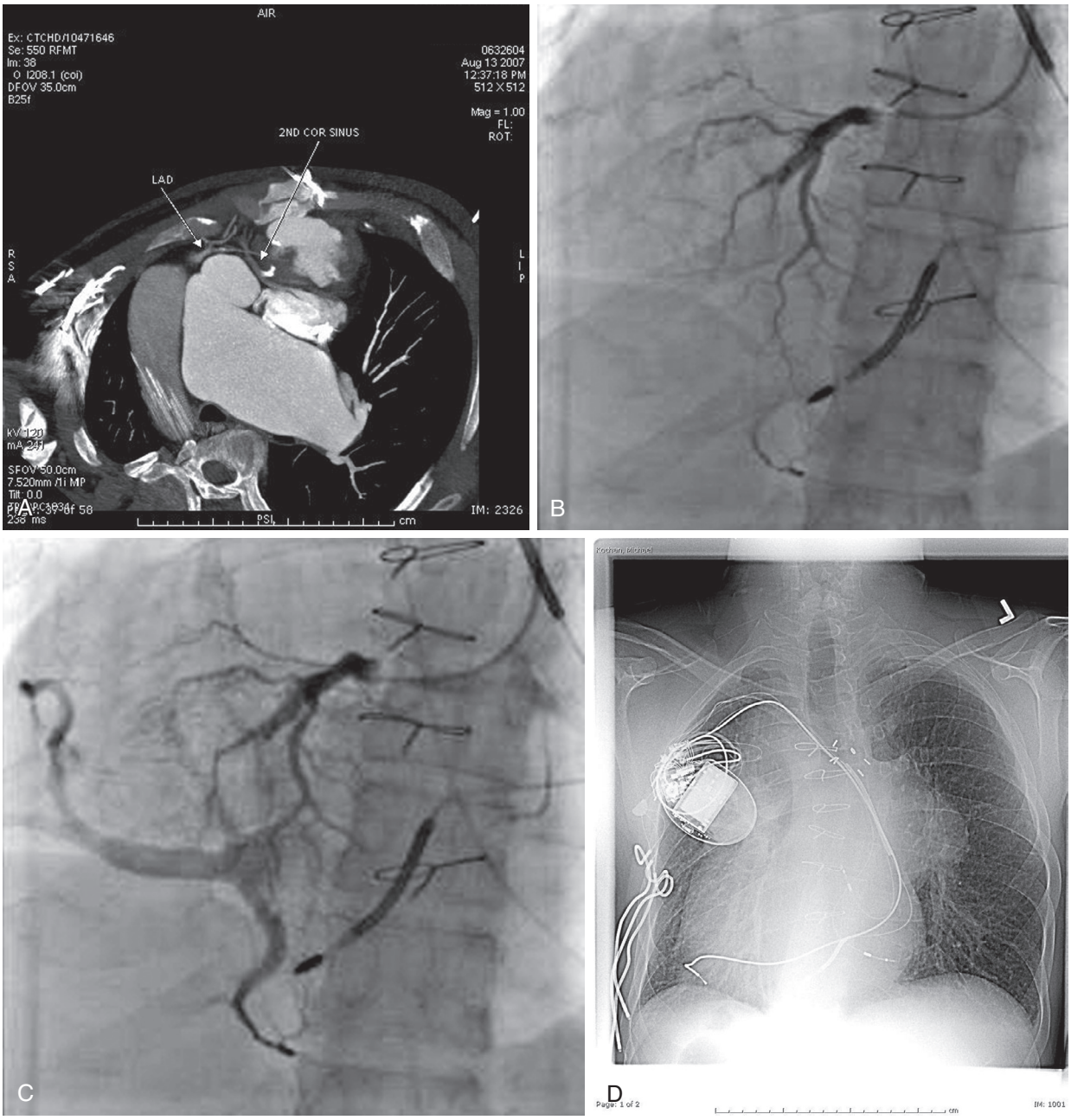
children grow, lung growth will increase the transthoracic current fraction, which, in combination with increasing heart size, can significantly alter the DFT.

Cardiac Resynchronization Therapy via Biventricular or Multisite Pacing

CRT has been studied and shown to be a treatment for adults with left ventricular failure, but no prospective randomized controlled trial evaluates CRT in pediatric patients or in those with adult congenital heart disease. What is known has been extracted from single-center case series and multicenter registries in those who have undergone CRT.[53–56] In those series, more than 70% of CRT implants in the pediatric age group have been in those with congenital heart disease.

The current CRT studies in patients with congenital heart disease demonstrate that CRT can benefit certain subsets of patients with congenital heart disease with a lower nonresponder rate (11% to 23%) than the 30% nonresponse rate seen in the adult CRT literature. CRT can be used safely in patients with congenital heart disease with similar complication rates as in the adult population (10% to 29%). The single most common major complication was coronary sinus lead issues, which were found in 5% to 18% of all transvenous pacemakers.

This may be related to anatomic issues found in the congenital heart disease population. The types of patients requiring CRT



• **Fig. 11.6** Transvenous cardiac resynchronization therapy in adult with complex congenital heart disease. The individual illustrated in Fig. 11.4 developed severe right ventricular dysfunction and was a candidate for upgrade to biventricular pacing. (A) Owing to the dextrocardia and high possibility of coronary sinus anomalies with L-transposition of the great arteries (L-TGA), a chest computed tomography scan was done for procedural planning. The coronal section shows a large thebesian vein (2nd cor sinus) coming off the anterior right atrium. This is common in those with L-TGA. (B, C) The thebesian vein was easily cannulated and a venogram provided a roadmap for the main coronary sinus. (D) A pacing lead was placed in the coronary sinus to pace the lateral right ventricle.

can be divided into four groups based on the type of ventricular arrangement: systemic left ventricle, subpulmonary right ventricle, systemic right ventricle, and single ventricle. Coronary sinus anomalies may be present in those with congenital heart disease.

Another potential use of CRT is to provide "backup" pacing for the individual who is pacemaker dependent. Lead fractures are more common in the young and those with epicardial systems. Having a second lead has been shown to reduce the incidence of cardiovascular events (syncope and hypotension) in association with lead fracture in pacemaker-dependent children with biventricular pacing versus those with single-ventricular pacing.[57]

Cardiac Resynchronization Therapy for the Systemic Left Ventricle

The failing systemic left ventricle represents approximately 50% of pediatric and congenital heart disease patients in CRT studies. Classically, antibradycardia pacing is easily achieved by right

ventricular apical pacing, given the procedural ease of accessing the venous ventricle and the proven long-term lead stability; however, chronic right ventricular apical pacing results in both interventricular and intraventricular dyssynchrony and may in fact be the least favorable place to pace.[58,59] These patients develop ventricular dyssynchrony and ventricular dysfunction related to conventional right ventricular pacing or dilated cardiomyopathy associated with left bundle branch block.

Presence of a systemic left ventricle was the strongest multivariable predictor of improvement in cardiac function with CRT. CRT in this patient subgroup resulted in major clinical improvement, left ventricular reverse remodeling, and a significant decrease in QRS duration. The best response to CRT in patients with a systemic left ventricle occurred in those with pacing-related dyssynchrony who were upgraded to biventricular pacing. Thus, at the first signs of left ventricular dilation and dysfunction, the goal should be to move to either left ventricular pacing or biventricular pacing.

Neonates presenting with congenital complete atrioventricular block are at higher risk for developing a dilated cardiomyopathy and should have prosynchronization (optimal single-site pacing) with epicardial left ventricular pacing as the first system. In a large group of children requiring long-term pacing, left ventricular apical and left ventricular lateral wall pacing were associated with the best preservation of left ventricular function, which appears to be related to preserved mechanical synchrony and contraction efficiency.[9–11] Infants and children (<15 to 20 kg) require an epicardial approach for placement of a left ventricular pacing lead. Conventional techniques of placing a left ventricular lead in the coronary sinus in an adult can be used for older children with cardiomyopathy and congenital heart disease with normal coronary sinus anatomy (see Chapter 8).

Cardiac Resynchronization Therapy for the Subpulmonary Right Ventricle

Right ventricular heart failure is an important cause of late morbidity in congenital heart disease, with 30% to 40% of resynchronization therapy involving the right ventricle. Several pacing strategies exist for CRT in patients with right ventricular failure, which has been most extensively studied in patients with tetralogy of Fallot, where the right ventricle is chronically damaged by a combination of pressure and volume overload resulting in myocardial scarring. These individuals also have surgically induced right bundle branch block resulting in electrical dyssynchrony.[60–62]

Similar to left ventricular pacing for left bundle branch block, it is possible to perform single-site or even dual-site right ventricular pacing or use an appropriately timed atrioventricular interval to allow merging of the native conduction through the left bundle with that of the paced right ventricle.[63–65] Although technically feasible, it may be difficult to achieve consistent electrical fusion because of variations in intrinsic AV conduction over a wide range of activities and heart rates. Another option is His bundle pacing, which has been reported to be effective in some patients with left bundle branch block and complete heart block but has not been tried in those with right bundle branch block.[66,67] Thus, CRT with biventricular pacing may be necessary to resynchronize the subpulmonary right ventricle.

Some of these individuals also have left ventricular dysfunction (5% to 10% of patients after repair of tetralogy of Fallot).[68,69] CRT for primary right ventricular failure has demonstrated improved ventricular synchrony, increased myocardial function, and increased exercise performance. Despite promising results in the adult congenital heart disease population with a systemic left ventricle, there remain little data on the effectiveness of noninvasive imaging to locate the optimal pacing site. One approach is to target the latest site of right ventricular activation or place the left ventricular lead first and then look for the location that results in the shortest QRS duration.

Cardiac Resynchronization Therapy for the Systemic Right Ventricle

Patients with systemic right ventricles (such as L-transposition of the great arteries [L-TGA] and D-transposition of the great arteries [D-TGA] status after an atrial switch) represent another congenital heart disease population at risk for developing right ventricular dysfunction. This is a complex group of patients, with some individuals having a rudimentary left ventricle. Results of CRT in this patient population have been mixed. Some of the discrepancy in response may relate to older age at the time of CRT, with less favorable responses seen in older patients. The smaller benefit of CRT in the systemic right ventricular population may be attributed to suboptimal myocardial fiber arrangement and abnormal ventricular contraction patterns when compared with both subpulmonary right ventricles and systemic left ventricles.[70,71]

Mechanical dyssynchrony is more important than electrical dyssynchrony for those with systemic right ventricular dysfunction.[72,73] The right ventricular morphology is distinguished from the left ventricle (LV), with the right ventricle (RV) being wrapped around the LV along its short axis. Thus, right ventricular contraction is dependent on left ventricular mechanical movement, with approximately 30% of the contractile energy of the RV being generated by that of the LV. When the RV contracts, it is more wavelike, in which the free wall begins to move at the base and apex and ends at the outflow tract.[74] Thus, RV shortening is greater longitudinally than radially. The higher-pressure LV contributes to right ventricular contraction via "transseptal" mechanical push. When the RV is systemic, the whole process is off kilter. Normally, the right ventricular outflow tract begins to contract later (25 to 70 ms) and remains contracted longer than the duration of the contraction of the remaining body of the RV.

If there is a single right ventricle and rudimentary hypoplastic LV, then the heart activates via a unique interventricular dyssynchrony, which resembles a swinging biventricular motion. There is a contraction delay between the LV and the RV, but the blood flow moves from the LV to the RV during

the left ventricular contraction and from the RV to the LV during the right ventricular contraction through a ventricular septal defect.[75]

With such complex mechanical interactions, it is important to take into consideration separate electrical and mechanical activation of the right ventricle. One approach that shows promise is use of three-dimensional (3D) imaging to determine if the mechanical delay is longitudinal or short axis. Leads should be placed at the farthest sites along a longitudinal direction in those with longitudinal delay; for those with short-axis delay, leads should be placed laterally on opposite sides of both ventricles.[72]

In L-TGA, if the left ventricular lead is placed via the coronary sinus, it is essential that preprocedural imaging of the coronary sinus be performed, since coronary sinus atresia occurs in up to 20% of patients.[76] The coronary sinus ostium is otherwise normally located in these patients, but the ventricular veins that drain the morphologic right ventricle tend to be small and short. These may not be adequate for lead implantation. However, large thebesian veins and profuse interventricular collateral vessels offer alternative access routes for lead implantation. Angiography of these vessels can provide a roadmap for the entire coronary venous system.[77,78]

In a patient whose status is D-TGA after an atrial switch, three approaches are possible: a complete epicardial system, a hybrid system with transvenous atrial and left ventricular leads, and an epicardial or transbaffle right ventricular lead. The last configuration has not been studied rigorously and chronic anticoagulation is needed.[79,80]

Cardiac Resynchronization Therapy for the Single Ventricle

Patients with single-ventricle physiology, by definition, do not have two separate ventricles; thus, resynchronization must be achieved by pacing two sites of the functional single ventricle (multisite pacing). This strategy of multisite pacing was first evaluated in the acute postoperative setting in which temporary multisite epicardial pacing resulted in an improvement in systolic blood pressure, cardiac index, indices of dyssynchrony by echocardiography, and QRS duration.[81]

Three studies of chronic CRT in patients with congenital heart disease that have included small numbers of patients with single-ventricle physiology have shown mixed results.[53–55] Although the small number of patients in these studies does not allow one to draw firm conclusions about the effect of CRT in patients with single-ventricle physiology, the inconsistent responses seen may reflect the complex and heterogeneous structural abnormalities in this population and the nonstandardized techniques utilized. There have been well-described case reports of a strong response to CRT in this population.[82]

The first step to achieve effective resynchronization therapy in a single-ventricle patient is to assemble a team committed to intensive review of the clinical data. The team includes a congenital heart disease specialist, an electrophysiologist, a cardiac imager with congenital heart disease experience, and a cardiac surgeon. Three-dimensional imaging via CT or MRI scan is then obtained and a 3D virtual model is created with musculoskeletal structures in place to plan the surgical approach.

Next, an evaluation of mechanical dyssynchrony is needed to find the area of latest mechanical activation. To succeed in this endeavor, the electrophysiologist needs to be in the operating room with the cardiac surgeon, and both need to have a thorough understanding of the anatomy and ventricular position. The posterior or apical lead is implanted first with the target being the area of latest ventricular activation or mitral apparatus.

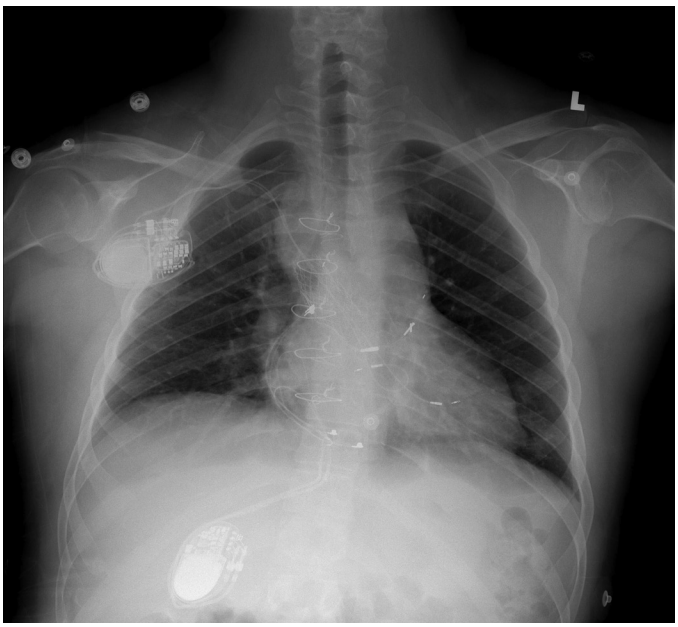
Then, a second, diametrically opposed, lead is placed (see Fig. 11.1). Another option is to pace from the first lead and target the area of latest activation or greatest shortening in QRS duration. This may require two surgical approaches (e.g., a thoracotomy and then a sternotomy). Significant pericardial adhesions are usually present, increasing the surgical risk for bleeding and generally leadings to a lengthy and tedious procedure. Thus, it is important to choose precisely where to make the thoracotomy incision for placement of the posterior lead.

Lead Extraction, Venous Occlusions, and Intracardiac Shunts in Pediatrics and Congenital Heart Disease

As children with transvenous leads age, they inevitably will require lead extraction to maintain vascular patency.[83–85] There are some basic principles that should be followed:

1. Lead extraction should not be attempted unless the operator is committed to completing the procedure. The operator and/or assistant should have experience in taking care of children or adults with congenital heart disease. The 2009 Heart Rhythm Society/American Heart Association guidelines provide a consensus for training in, indications for, and patient management for transvenous lead extraction. In adherence to the principles of those guidelines, the surgical backup team needs to have training and experience with children and congenital heart disease.
2. It is preferable to extract leads if at all possible rather than abandoning nonfunctional leads.
3. Since children require lifelong pacing, a nonfunctional lead should never be cut short if it is going to be abandoned, as the extractionist will not have sufficient lead length to secure the lead proximally.
4. A lead should not be jailed with a stent or a valve, since late infection would then require surgical extraction.
5. Venous access should always be maintained when a lead is being extracted. In the situation of venous occlusion, we recommend extraction and reimplantation on the ipsilateral side rather than implanting on the contralateral side. Sites for venous access should be preserved as much as possible. If one side is occluded, it is better to extract than to abandon an occluded side.

Venous occlusion is a known long-term complication of permanent transvenous pacing leads.[86] Risk factors for venous thrombosis after lead placement have been described. They include the absence of anticoagulant therapy, a history of prior



• **Fig. 11.7** Complexity of device placement in adult with D-transposition of the great arteries (D-TGA), after a Mustard operation. Chest radiograph of a 27-year-old with D-TGA, status postoperative Mustard procedure, and sinus node dysfunction who had multiple pacing systems and revisions. He started with an epicardial system at age 5 years, and the lead fractured when he was 8 years old. A transvenous system was implanted; he developed superior baffle stenosis, and a stent was placed jailing an atrial lead. That lead failed at age 17 years and a new system was implanted, but he developed superior vena cava (SVC) syndrome after 10 years and more stents were placed after lead extraction. SVC stenosis was present, so thin lumenless leads were used. The atrial lead is in the inferior baffle because the body of the left atrium is blocked by the original stent that was placed beyond the end of the superior baffle. The ventricular lead is in the left ventricular apex.

venous thrombosis, use of female hormone therapy, and the presence of multiple pacing leads.[87]

If venous access is needed in the setting of a complete venous occlusion, a variety of techniques for recanalization and venous dilation are now available.[88,89] Baffle obstruction D-TGA after an atrial switch is fairly common and may require endovascular stent placement before lead placement[90] (Fig. 11.7).

Embolic stroke in those with implanted leads can occur from an intracardiac right-to-left shunt or inadvertent lead placement in the systemic circulation.[91,92] These shunts can be located at the atrial or ventricular level, ranging from a patent foramen ovale to large residual septal defects and patch leaks. Fontan patients with high central venous pressure can develop direct connections between the supracardiac veins and pulmonary venous atrium through venovenous collaterals.[93]

Trivial shunts that are predominately left to right are probably not absolute contraindications to transvenous leads, but larger shunts, particularly if right to left, need to be evaluated carefully by angiography or echocardiography before a final decision is made on the route for lead implantation. If transvenous leads are strongly preferred in such cases, shunt closure can be attempted beforehand with interventional techniques such as septal occluders, covered stents, or even surgery.[94–96] If intracardiac shunting cannot be eliminated satisfactorily, epicardial lead placement is probably the wisest alternative. If there are still compelling indications for a transvenous system, anticoagulation is used after lead placement in patients with residual shunting.

Conclusions

Device implantation in pediatrics and patients with congenital heart disease requires specialized techniques, experience, and training to meet the unique needs of these patients. For the young patient it is necessary to think about the future and plan for a life of pacing. In those with congenital heart disease, the anatomic and physiologic issues are not equivalent to the typical older adult with acquired heart disease. Implant techniques and lead placement must be individualized to the patient's specific anatomic diagnosis.

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12

Special Circumstances and Obstacles

ROGER CARRILLO, CHRIS HEALY

Introduction

Implantation rates for cardiac implantable electronic devices (CIEDs) have continued to rise.[1,2] These devices have proven to be invaluable tools in the modern practice of cardiology. As indications for device implantation increase and thus the patient population eligible for these procedures continues to broaden, the surgical challenges faced by implanting physicians also continue to expand.[3] Although the knowledge and techniques associated with CIED implantation have advanced significantly, a focus on proper surgical technique is key to successful implantation and the avoidance of complications. This chapter focuses on some special circumstances faced by implanting physicians and the techniques available to manage them.

Prior Median Sternotomy

Prior median sternotomy is neither an absolute nor a relative contraindication to the implantation of CIEDs. Many patients who have previously undergone open heart surgery require and benefit from device therapy. Special consideration must be given to patients with central venous stenosis, prior right atrial appendage ligation, and a requirement for lead tunneling. Central venous stenosis can result from the placement of multiple indwelling central catheters or ligation of the left brachiocephalic vein (for optimal exposure of the aortic arch vessels) during cardiac surgery. Right atrial appendage ligation is common in open heart surgery requiring cardiopulmonary bypass (a common site for placement of the venous cannula) and thus requires placement of a right atrial lead in an alternate location, typically the lateral right atrium (Fig. 12.1). Although tunneling leads from the right infraclavicular area to the left infraclavicular area (or vice versa) is not recommended, it should particularly be avoided in patients with prior median sternotomy. The sternotomy wires can potentially abrade the CIED leads and result in impaired durability and function.

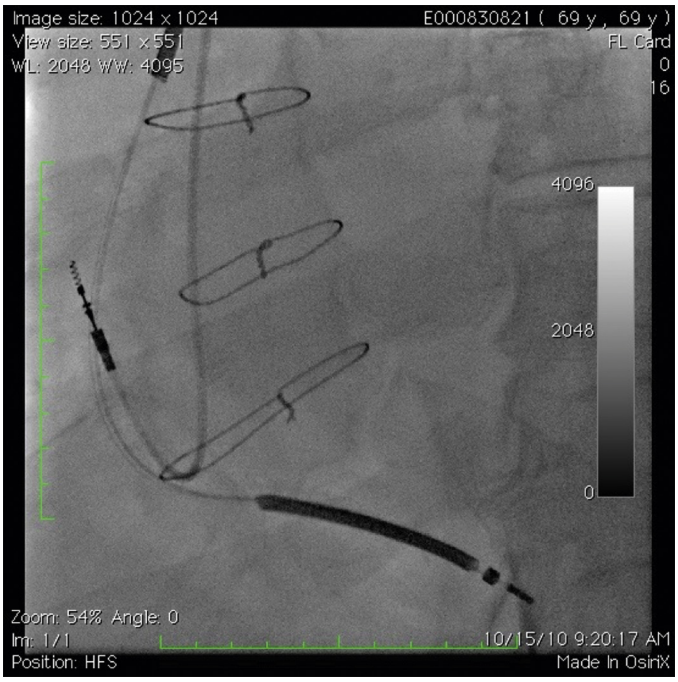
Obesity

Performing any cardiac procedure in a morbidly obese patient carries significant risk. This is further complicated by weight restrictions on procedural tables (both in the operating room and in the catheterization or electrophysiology laboratory). However, in patients with an indication for a CIED, obesity should not be a contraindication to the procedure. In fact, pacemaker implantation has been described in patients weighing over 600 pounds.[4] Proper preoperative planning can help prevent the occurrence of complications. Aspects to consider include the availability of a capable anesthesia provider, the availability of specialized surgical equipment to facilitate completing the procedure quickly and safely, and the use of appropriate-length device leads.

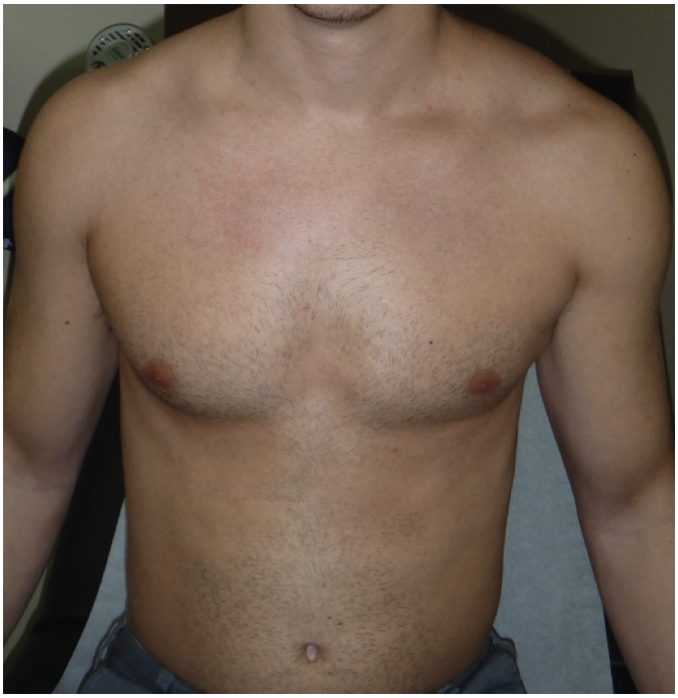
Obesity is a major risk factor for sleep apnea, which can predispose patients to airway compromise. This is particularly an issue if sedation is used. In patients with a significantly elevated body mass index (BMI), short neck, or proven/suspected sleep apnea, an anesthesia provider who has experience working with obese patients should be used. The abundance of subcutaneous tissue encountered in obese patients typically requires the use of special retractors to allow for adequate visualization of the surgical field. Furthermore, when an axillary or subclavian puncture is performed, standard needles may not be long enough. In these circumstances, a pericardiocentesis needle may be necessary to access the vein. In fact, a BMI greater than 30 kg/m^2 has been associated with an increased incidence of failure of subclavian vein catheterization.[5] For this reason, a cephalic cut-down may be preferred in these patients. Significant displacement of the heart in the thoracic cavity after extreme weight loss, with resulting dislodgement of intracardiac leads, has been described.[6] The potential for this should be recognized, and adequate slack should be provided in an attempt to prevent dislodgement.

Anorexia/Cachexia

Patients who are anorexic or cachectic tend to have very thin subcutaneous tissue. Attempts to place the pocket in the subcutaneous position poses a problem because of the lack of a significant barrier between the device and the patient's skin. In such patients, submuscular implantation is a viable alternative to mitigate the risk of the device eroding through the skin.[7] Wound healing is delayed in the majority of these patients.



• **Fig. 12.1** Lateral implantation of the right atrial lead in a patient with prior median sternotomy and ligation of the right atrial appendage.

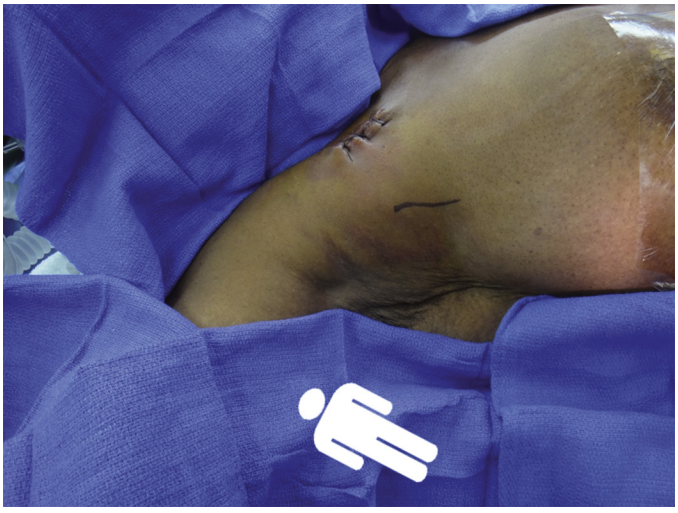


• **Fig. 12.2** Postoperative image of a patient with a muscular chest who underwent implantation of a device via an incision in the left anterior axillary line.

Tip: Interrupted closure is preferred over a running suture technique, and staples are typically used. This will reduce tissue tension and may help prevent wound dehiscence.

Muscular Chest

It can be difficult to attain venous access in a patient with a muscular chest simply because of the presence of additional tissue. Venography and the use of real-time ultrasound visualization are valuable in overcoming these issues. Cosmetic concerns may be more prevalent in patients with muscular chests. As a result, alternative implantation techniques such as an axillary approach can be considered.[8] Indications for axillary implantation include bilateral infraclavicular infections, unilateral infraclavicular infections with a contraindication to using the contralateral side (e.g., hemodialysis access on that side), and cosmetic concerns.[9] In this approach, an incision is made through the pectoralis muscle in the axilla. Once both the pectoralis major and minor have been retracted, the axillary vein can be exposed and cannulated directly. The device can then be placed in the subpectoral space with closure of the axillary fascia to avoid system migration (Figs. 12.2 and 12.3).



• **Fig. 12.3** Planned location of axillary incision in a preoperative image of a patient undergoing axillary implantation of a device. She had recent infection of infraclavicular devices on both sides. Nylon sutures from the right-sided incision from the extraction procedure are visible in the upper portion of the operative field.

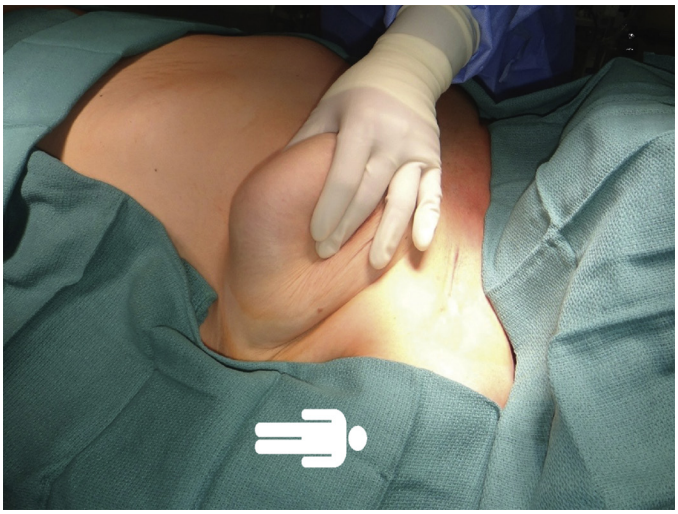
Alterations in Breast Tissue

In 2013, there were 400,000 to 500,000 cosmetic breast surgeries performed in the United States alone.[10] Furthermore, there were over 235,000 new cases of breast cancer diagnosed in the United States in 2014.[11] Mastectomies, lymph node dissections, breast implants, and breast reductions all result in clinically significant changes to the anatomy encountered during CIED implantation. Proper planning can help prevent surprises and complications in patients with atypical breast anatomy.

Pendulous Breasts

Large, pendulous breasts can create numerous issues with respect to device implantation. As in obese patients, there can

be an abundance of subcutaneous tissue between the skin and pectoralis fascia. This can make the procedure more challenging and calls for special retractors to allow for adequate visualization and exposure of the surgical field. Furthermore, there can be significant shifting of tissue when the patient moves from the supine to the upright position. This should be accounted for when the pocket is made and when the device is positioned. It is important to suture the device to the pectoralis fascia with nonabsorbable suture in these patients because clinically relevant shifting of the device can occur (Fig. 12.4).



• **Fig. 12.4** Migration of an implantable cardioverter-defibrillator in a patient with pendulous breasts (the device is in the surgeon's hand).

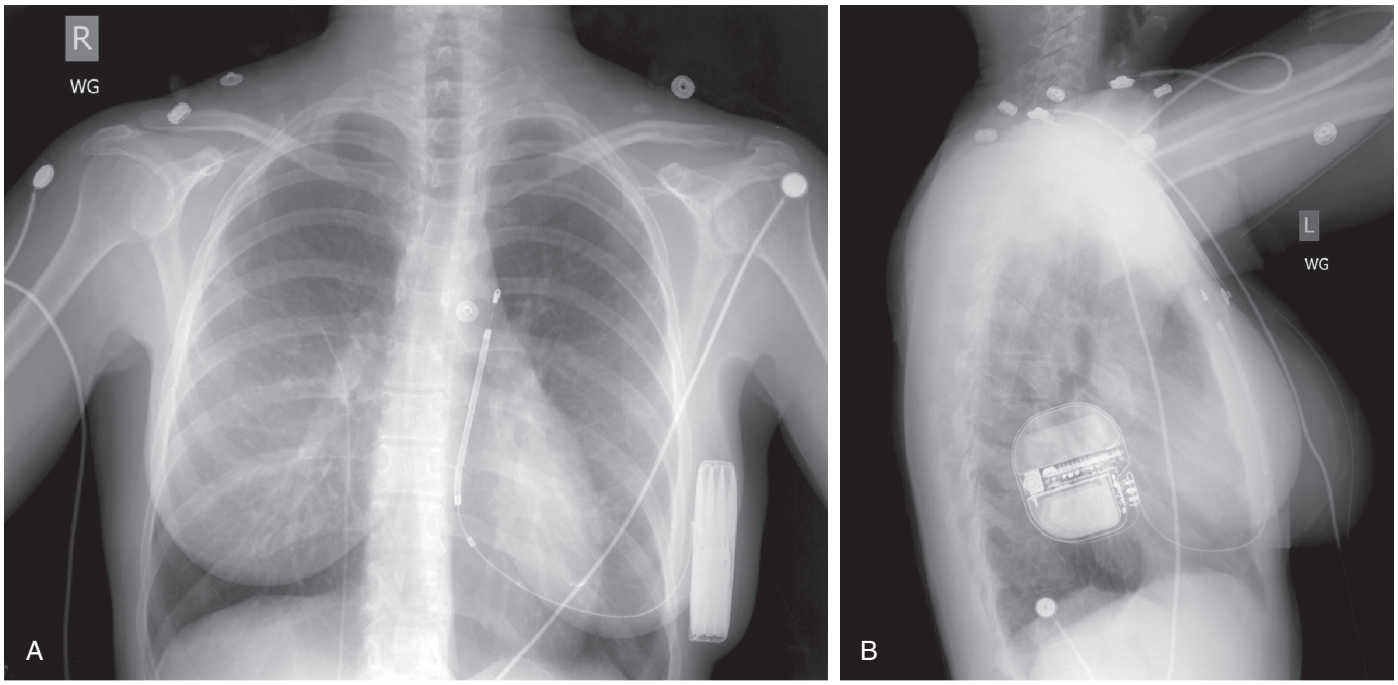
Breast Augmentation

In most circumstances, CIEDs can be implanted, extracted, or replaced in patients with breast implants without disrupting the cosmetic implant. In fact, even subcutaneous defibrillators, which require considerably more manipulation/instrumentation of the chest wall, can be placed safely in these patients without disturbing the implant (Fig. 12.5). In certain circumstances, the breast implants may need to be moved or explanted and reimplanted to allow for performance of the procedure. One such circumstance is patients who require implantation of a transatrial device, either owing to a history of superior vena cava stenosis or other contraindications to standard transvenous implantation (Fig. 12.6). When this is the case, consideration should be given to having a plastic surgeon assist when the implanter is not familiar with these types of procedures.

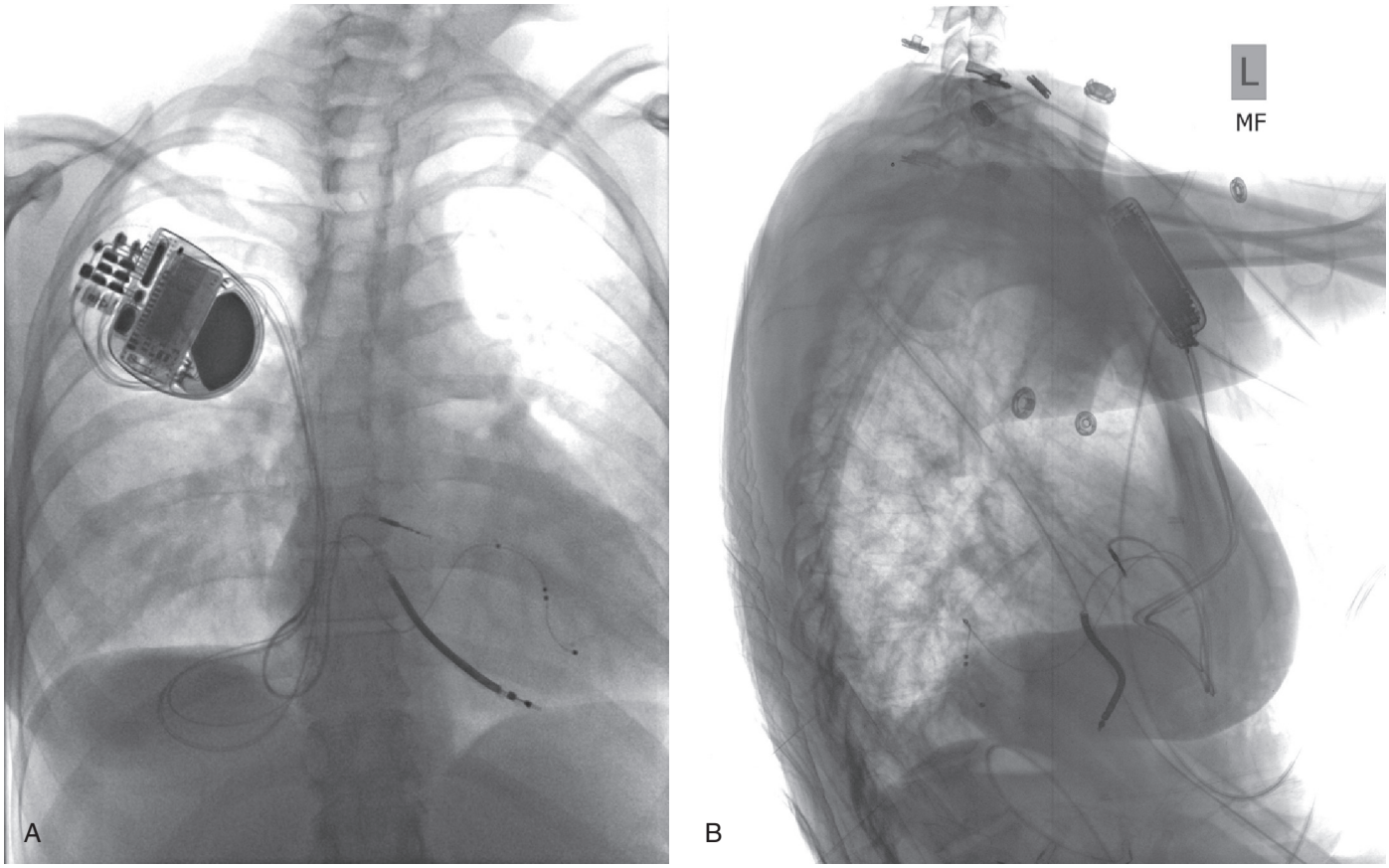
The importance of sterile technique and not manipulating the cosmetic implant unless absolutely necessary cannot be overemphasized. This will help decrease the risk of breast implant infections. Breast implant infections can be costly and challenging to treat. They commonly require removal of the implant to clear the infection.[12] This can be particularly dangerous in patients with CIEDs because the infection can seed the device, either from local extension or bacteremia (Fig. 12.7).

Breast Reduction

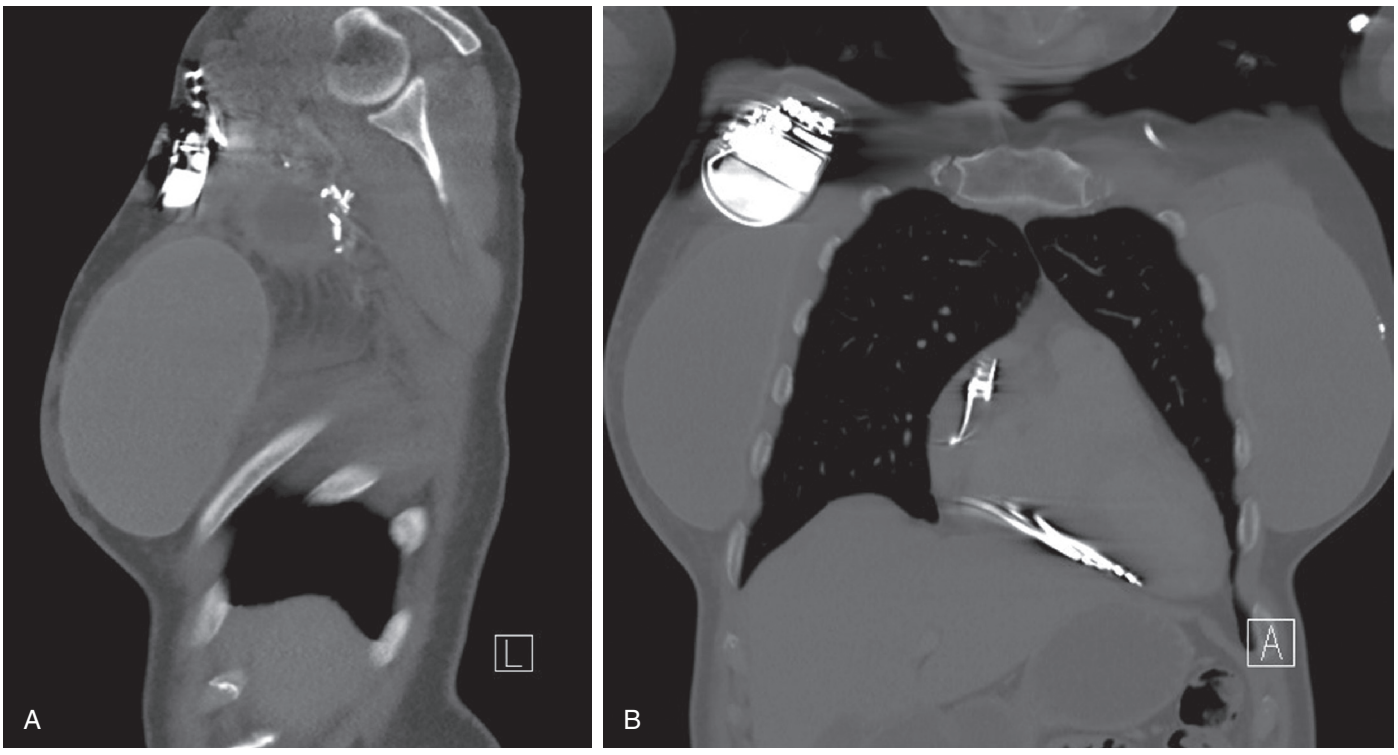
Most breast reduction procedures are performed through an inframammary or nipple incision. These incisions are typically far away from CIED implantation sites (infraclavicular or



• **Fig. 12.5** Implantation of a subcutaneous defibrillator in a patient with breast implants. (A) Posteroanterior view. (B) Lateral view. This patient with previous breast augmentation developed symptomatic superior vena cava (SVC) stenosis from a transvenous defibrillator. She underwent implantation of a subcutaneous defibrillator following extraction of her transvenous device and venoplasty of her SVC stenosis. Attention was given to not disrupting the cosmetic implants.



• **Fig. 12.6** Transatrial implantation of a biventricular defibrillator in a patient with breast implants. (A) Anteroposterior view. (B) Lateral view. This patient had a history of symptomatic superior vena cava syndrome and required implantation of a biventricular defibrillator. The breast implant was removed and the right hemithorax was entered in the fourth intercostal space. After the cardiac implantable electronic device was implanted, the implant was replaced in the same position.



• **Fig. 12.7** Sagittal and coronal images of a patient with an implanted defibrillator, bilateral breast implants, and a localized infection near both the cosmetic implant and the cardiac implantable electronic device. (A) Sagittal computed tomography (CT) image. (B) Coronal CT image. In (A), a fluid collection concerning for infection can be seen very near both the implantable cardioverter-defibrillator (ICD) and the breast implant. The fluid collection is posterior and inferior to the ICD and posterior and superior to the breast implant. In (B), it can be better appreciated just how close the ICD and the breast implant are to each other (essentially touching).

axillary). Furthermore, lymphatic drainage is usually not disturbed during the procedure. Thus, a history of breast reduction surgery ordinarily does not pose any significant issues for routine infraclavicular device implantation. One exception is implantation of a subcutaneous defibrillator in a patient who has previously undergone breast reduction surgery via an inframammary incision, as both procedures are performed in the same area. In this circumstance, care should be taken to avoid areas of previous scarring when possible to avoid problems with wound healing.

Mastectomy

Surgical mastectomy hinders lymphatic drainage on the affected side. When possible, ipsilateral implantation should be avoided because it can lead to impaired wound healing. This is often not possible (i.e., bilateral mastectomy). Bilateral surgical mastectomy is not a contraindication to CIED implantation, but the implanter must recognize that the patient is at risk for compromised wound healing. Implantation of the pacemaker pulse generator in a neck pocket has been described.[13] However, this is not recommended.

Indwelling Central Lines

Myriad indwelling central venous lines are available in clinical practice. These include peripherally inserted central venous catheters (PICC lines); temporary (nontunneled) central venous catheters used for measurement of central venous pressures, infusion of vasopressors, or temporary hemodialysis; tunneled central venous catheters for long-term hemodialysis; and subcutaneous intravascular ports commonly used for the administration of chemotherapy. These catheters are an important tool in clinical practice but present a number of problems in patients with CIEDs, including increased risk of central venous stenosis, CIED infection, and lead dislodgement.

In general, all central venous catheters should be inserted only when necessary and should remain in place for the shortest period possible. This may help prevent the development of central venous stenosis, which is addressed further under "Vascular Stenosis and Obstruction." This same approach may help prevent CIED-related infection, as indwelling central venous catheters have been associated with an increased incidence of device infection.[14] In addition, attention should be paid when inserting central venous catheters in patients with CIEDs to avoid lead dislodgement. The risk of lead dislodgement is highest immediately following CIED implantation and decreases as the time from implantation increases.

Dialysis Fistulas

Among patients with end-stage renal disease (ESRD), hemodialysis is a life-prolonging therapy that cannot be interrupted for any significant period without significant consequences. As such, maintenance of central venous patency is very important in these patients. Dialysis fistulas are the preferred hemodialysis access option whenever feasible as they are associated with the lowest risk of infection and the highest long-term patency rates.[15]

In patients who have or will require a dialysis fistula, completely subcutaneous or epicardial systems are an often underutilized option. In fact, patients at very high risk of infection, especially those with CIED-related infection secondary to hemodialysis-related bacteremia, have been successfully managed with epicardial systems.[16]

Tip: In patients who have or will require a dialysis fistula, CIEDs should not be placed on the ipsilateral side of the body.

Prior Clavicular Fracture

Common fluoroscopic landmarks may be misleading in the presence of prior clavicular fractures because of altered anatomy. A lateral axillary stick should be supplemented by concomitant venography and/or the use of real-time intraprocedural ultrasound.

Vein thrombosis in association with uncomplicated clavicular fractures is rare but not unheard of; this must be considered because it can affect procedural success.[17] If the patient has had repair of the fracture through internal fixation with the introduction of hardware (plates or screws), the system should be placed on the contralateral side to avoid abrasion and long-term damage to the leads.[18]

Tracheostomy

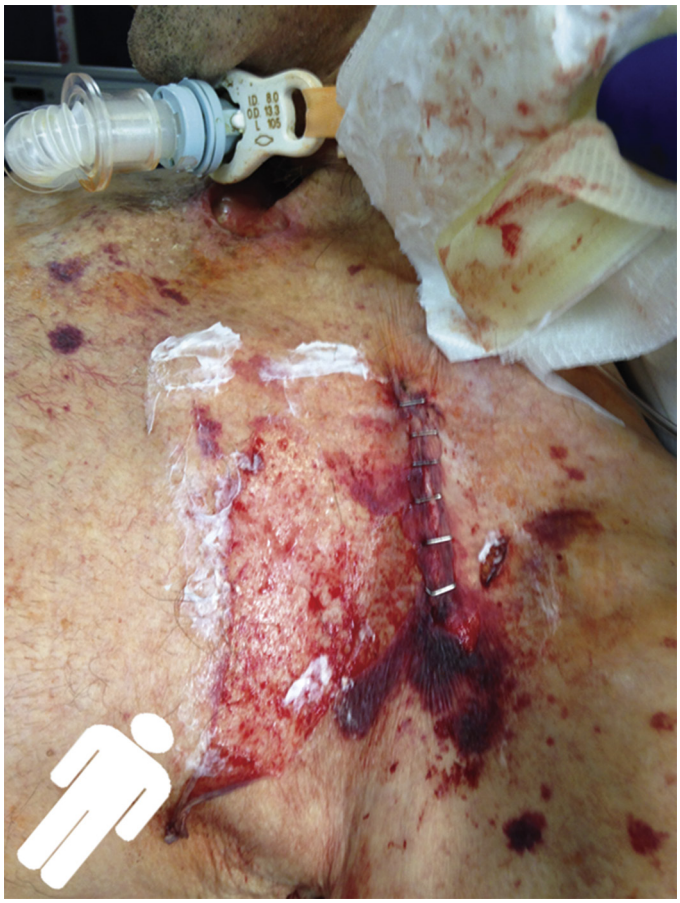
The presence of a tracheostomy poses risks for device infection in the perioperative and postoperative period until the surgical wound has healed. This phenomenon results from the close proximity of the tracheostomy to the operative field (Fig. 12.8). Secretions from the tracheostomy can migrate to the infraclavicular area and must be isolated from the sterile field at the time of implantation. Plastic surgical drapes and bandages are helpful in preventing contamination from secretions. Assuming the aforementioned precautions have been taken, an otherwise standard implantation technique will suffice for patients with a tracheostomy.

Chest Irradiation

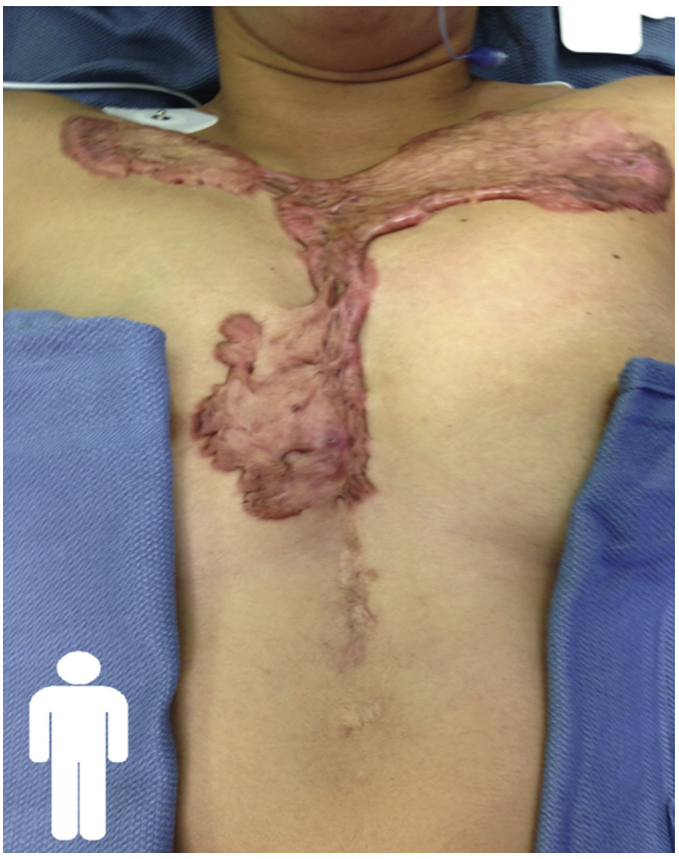
Prior chest irradiation can result in impaired lymphatic drainage and increased tissue friability.[19] As such, postsurgical wound healing will be delayed, and it is best to avoid tissue that has previously been subject to radiation. If no viable alternative exists, consider interrupted rather than running suture closure to decrease tissue tension and the risk of postoperative wound dehiscence.

Vascular Stenosis and Obstruction

Significant progress has been made in the treatment of chronic diseases, including malignancy, ESRD, and heart failure. Subsequently, an increasing number of patients are receiving central venous catheters, intravascular ports, and CIEDs. Although these interventions can be life prolonging, they predispose patients to the development of central venous stenosis and obstruction. The management of central venous stenosis can be



• **Fig. 12.8** Postoperative image of a patient with a tracheostomy who underwent implantation of a pacemaker.



• **Fig. 12.9** Extensive keloid formation encountered at the time of device generator change. This patient with corrected congenital heart disease and congenital complete heart block had previously undergone one median sternotomy, multiple cardiac implantable electronic device implantations, and two device extractions. She had developed extensive keloid related to these procedures.

an issue at the time of either the initial CIED implantation or an upgrade. In patients with ESRD requiring hemodialysis, in particular, the management of central venous stenosis can be particularly challenging. The patency of the central venous circulation is necessary for these patients to receive dialysis, a therapy that cannot be delayed for any significant period. As such, central venous stenosis must be managed aggressively in these patients.

Previous reports of the incidence of venous stenosis in patients with CIEDs have varied widely.[20–22] In a prospective analysis of patients with CIEDs, almost two-thirds had some degree of venous stenosis at 6 months, with severe or occlusive stenosis in over 20%.[22] Several treatment modalities exist for the management of central venous stenosis and obstruction, including venoplasty, stenting, and lead extraction. Venoplasty is typically the first-line therapy, particularly in asymptomatic patients where the goal of the procedure is achieving a vascular channel to accommodate device implantation or upgrade. Stenting may improve long-term patency rates, which can be clinically important if the stenosis is symptomatic, as in superior vena cava syndrome.[23]

It is worth mentioning that trapping a transvenous lead against the vessel wall when stenting open a vein should be avoided whenever possible because it eliminates the option of future transvenous extraction if necessary.[24]

Lead extraction is not a first-line therapy but certainly has a role in the management of these patients. In patients with complete occlusion of a vessel that already contains a transvenous lead, laser lead extraction may be the only option for the addition of transvenous leads when necessary.[24]

All the available therapies for central venous stenosis are plagued by high recurrence rates.[23] The use of a nontransvenous system should be considered in patients with or at elevated risk (e.g., receiving hemodialysis) of central venous stenosis. Potential nontransvenous approaches include epicardial lead placement, transatrial lead placement (see Fig. 12.6), and subcutaneous defibrillators (see Fig. 12.5). Subcutaneous defibrillators are not an option in patients who require pacing. Although not currently approved in the United States, leadless pacemakers represent a potentially useful tool for avoiding central venous stenosis.[25]

Specifically in regard to the transatrial approach, this novel technique provides the advantage of bringing state-of-the-art technology to patients with complete venous obstruction in which an endocardial system may be a better option than an epicardial system. For example, the placement of an epicardial biventricular implantable cardioverter-defibrillator requires a greater number of incisions and is a more technically difficult procedure than the transatrial approach. In this procedure, an incision is made through a right minithoracotomy at the level of the fourth intercostal space. Utilizing an endoscopic technique, wires are placed directly into the right atrium under fluoroscopic guidance and the generator is subsequently placed in a subpectoral position.

Keloids

Patients with large keloids and/or hypertrophic scars can be extremely challenging to manage. Proper surgical technique and avoidance of excess tension at the incision site can help alleviate this problem to some degree. However, some patients develop extensive keloids in spite of every effort to the contrary (Fig. 12.9). In these challenging patients, a cosmetic surgeon should be asked to assist in management.

Summary

As the number of patients with indications for implantation of a CIED continues to grow, the diversity and complexity of these patients will also continue to grow. This will invariably result in a broader array of anatomic challenges faced by implanting physicians. To successfully navigate these challenges, attention must be paid to adequate preprocedure planning and maintenance of proper surgical technique. Consultation with other subspecialists should be employed when appropriate. Through open collaboration between electrophysiologists and cardiac surgeons, a greater number of patients can receive life-improving and/or life-saving therapy with CIEDs.

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13

Pitfalls and Complications

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Introduction

Complications are inherent to cardiac implantable electronic devices (CIEDs). Foreign bodies placed into and remaining in vascular structures such as veins and cardiac chambers can result in various forms of trauma. Adverse events such as infection and dislodgement have been recognized since the early reports of transvenous pacemaker implantation.[1] Furthermore, adverse events can occur during the procedure, in the immediate postoperative period, and well after the implant procedure. In addition, complications tend to increase with the number of leads implanted. This has been demonstrated in the Ontario Implantable Cardioverter Defibrillator (ICD) Registry,[2] in an analysis from the National Cardiovascular Data ICD Registry,[3] and in a cohort of patients referred for lead addition or revision at the time of generator replacement.[4] The same trend was observed in both major and minor complications.[2,4] Interestingly, adverse events have not always been associated with death in large patient series of CIEDs.[5] Nevertheless, a systematic approach aimed at reducing complications is imperative to provide optimal patient care.

Recently, prospective and retrospective patient series have been published that attempt to identify and quantify complication incidence and prevalence.[2–4] Although no universally agreed upon list of CIED complications is available or mandated, many of these series endeavor to be comprehensive. Box 13.1 illustrates the complications prespecified in the Implantable Cardiac Pulse Generator Replacement (REPLACE) Registry, a prospective multicenter registry designed to quantify complications after CIED generator replacement with or without planned transvenous lead addition or revision.[4] In this registry, complications were carefully prespecified, defined, and independently adjudicated. Furthermore, these complications were further characterized as major or minor and as periprocedural to up to 6 months of follow-up. Summary analyses from multiple recent large patient series, the majority of whom were implanted with an implantable cardioverter-defibrillator (ICD), illustrate the most frequent complications.[2,4,6] Not surprisingly, these complications are lead malfunction or dislodgement, pneumothorax, hematoma, and infection, with cumulative incidences clustering around 1% (Table 13.1). The following sections address these more common complications.

Lead Malfunction or Dislodgement

Transvenous leads are designed to function for years in the hostile environment of the human body. Although placed into position and secured proximally with nonabsorbable sutures at implantation, not all leads are actively fixated into the endocardial surface of the heart. Cardiac contraction, rotation, and translocation forces can all affect lead stability. Moreover, transvenous coronary sinus leads placed into venous branches on the epicardium carry no active fixation mechanisms. Active fixation leads require deploying a screw or manually screwing a lead into the myocardium. Either technique can result in cardiac perforation of the atrium or the ventricle. At our institutions, it is believed that deploying an active fixation lead requires careful attention to avoid accumulation of torque, which may result in late perforation.[7] Careful observation using fluoroscopy during the implant procedure may also assist in evaluation of acute lead stability. At the time of generator replacement, the local lead environment is disrupted, which can unmask lead abnormalities (Fig. 13.1). Visual inspection is performed to look for fluid in the insulation, insulation tears, wear points, kinks, and fractures. Chest radiographs and cinefluoroscopy are also useful to evaluate lead appearance and identify potential issues (Fig. 13.2). A corollary to Halsted's surgical principle of gentle tissue handling can be applied to transvenous leads as "gentle lead handling."

Tip: Handle leads gently and assess for stability before closing the pocket.

Hematomas and Bleeding

Device site hematomas are problematic for both patients and providers. Hematomas can become tense and uncomfortable and, if significant, threaten the integrity of the incision. Large tense hematomas that are not evacuated can result in compression and retraction of the subcutaneous tissues such that closing the incision after hematoma evacuation is difficult. In this situation, vertical nylon mattress sutures may be required to approximate the edges of the pocket. These sutures are

• BOX 13.1 Definitions of Major and Minor Complications Prespecified in the REPLACE Registry

Major Complications

- Death within 30 days related to the procedure
- Cardiac arrest within 24 hr of the procedure
- Respiratory arrest/failure within 24 hr of the procedure requiring ventilator support or intubation
- Acute coronary syndrome directly related to the procedure
- Cardiac perforation with or without pericardial tamponade, requiring pericardiocentesis or other surgical intervention
- Pneumothorax requiring observation or chest tube placement
- Hemothorax
- Stroke within 30 days of the replacement procedure
- Hemodynamic instability during the procedure requiring unplanned intervention and/or aborting the procedure
- Infection requiring intravenous antibiotics and/or system removal/extraction
- Generator or lead malfunction requiring reoperation
- Pocket revision requiring reoperation
- Prolonged hospitalization attributable to the device replacement procedure[a]
- Hematoma requiring evacuation, drainage, blood transfusion, hospitalization, or extension of hospital stay to treat hematoma
- Hospital readmission directly related to the generator replacement procedure
- Coronary venous dissection with hemodynamic instability
- Pulmonary embolus
- Peripheral arterial embolus
- Deep vein thrombosis
- Drug reaction resulting in an aborted procedure
- Cardiac valve injury
- New atrioventricular conduction block developing as a result of the procedure
- Arteriovenous fistula related to the replacement procedure

Minor Complications

- Hematoma lasting >7 days with tenseness, drainage, or minor dehiscence managed as an outpatient
- Hematomas without tenseness but requiring additional outpatient evaluation
- Implant-related pain lasting >7 days requiring prolonged use of narcotic pain medications[b]
- Cellulitis treated as an outpatient with oral antibiotics
- Stitch abscess
- Minor surgical wound findings[c]
- Unanticipated device reprogramming due to inadequate lead performance with significant patient symptoms or status change, excluding asymptomatic threshold changes
- Reversal of sedation for respiratory compromise requiring benzodiazepine or opioid receptor antagonist
- Peripheral nerve injury
- Superficial phlebitis

[a]Unexpected prolonged hospitalization (excludes patients with preprocedure exacerbation of medical illness or those requiring routine intravenous anticoagulation postprocedure).
[b]Excluding patients taking chronic narcotic medications.
[c]Includes complaints such as noninfectious rashes, superficial incisional dehiscence, and painful blistering from tape.

TABLE 13.1 Common Complications From Recent Cardiac Implantable Electronic Device Registries

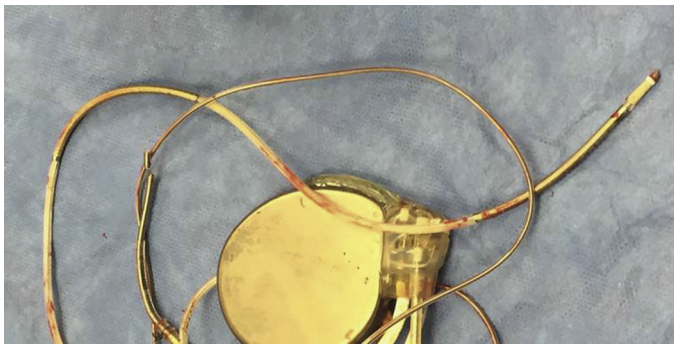
Complication	National Cardiovascular Data Registry[6] N = 268,701 (%)	Ontario ICD Registry[2] N = 3340 (%)	REPLACE Registry[4] N = 1744 (%)	
			Cohort 1	Cohort 2
Lead dislodgement or malfunction	2494 (0.93)	185 (5.5)	10 (1)	56 (7.8)
Hematoma	2509 (0.93)	33 (1)	7 (0.7)	11 (1.5)
Pneumothorax	1126 (0.42)	13 (0.4)	0	6 (0.8)
Infection	76 (0.03)	37 (1.2)	8 (0.8)	6 (0.8)
Death	1021 (0.38)			

nonabsorbable and require removal in approximately 10 days. A metaanalysis of ICD implant complications published in 2012 reported hematoma event rates ranging from 1% to 6%.[8]

The association between pocket hematoma and heparin has been recognized for over 10 years. One of the earliest randomized trials from the University of Michigan in 2000 demonstrated hematoma rates of 17% to 22% depending on when intravenous heparin was instituted after the implant procedure.[9] More recent series continue to show significant hematoma rates; a metaanalysis of 13 bleeding/hematoma trials with ICDs was published in 2012 and involved nearly 6000 patients.[10] All the expected bleeding and thromboembolic complications were recorded. Patients were categorized by anticoagulant and/or antiplatelet treatment: no therapy, anticoagulant held, continued single antiplatelet therapy, dual antiplatelet therapy, and heparin bridging strategy. The highest rate of bleeding was seen in the heparin bridging group, with an event rate of nearly 15%, followed by the dual antiplatelet group, with an event rate approaching 10%.[10]

The recent Bridge or Continue Coumadin for Device Surgery Randomized Controlled Trial (BRUISE CONTROL) confirmed these retrospective observations. This randomized prospective trial compared continued warfarin versus heparin bridging in patients undergoing initial implant, generator change, lead revision, and pocket revision from 17 centers in Canada and Brazil. The primary endpoint was clinically significant device pocket hematoma. The hematoma rate in patients continued on warfarin was 3.5% compared with 16% in those managed with a heparin bridging strategy ($P < .001$), with benefit seen in all prespecified subgroups, such as patients with mechanical heart valves and those treated with concurrent antiplatelet agents.[11]

Many patients who undergo CIED implant have concomitant cardiovascular disease and are treated with the dual antiplatelet agents aspirin and clopidogrel. One prospective series of 935 patients who underwent pacemaker or ICD implantation reported a hematoma rate of 18.3% in the subset of patients who underwent device implantation with uninterrupted clopidogrel. There were no hematomas observed in patients who had clopidogrel discontinued more than 4 days before device implantation.[12]



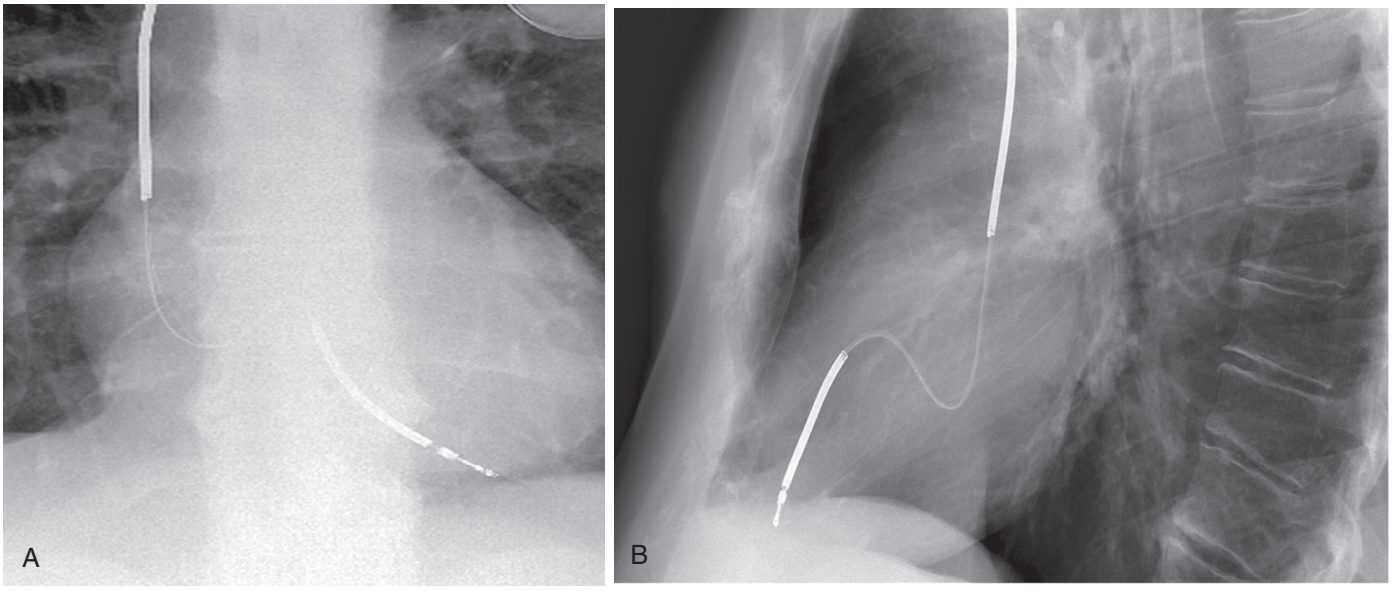
• **Fig. 13.1** Insulation defect observed at time of generator replacement and lead extraction.

In a specific retrospective analysis of 1388 patients who underwent pacemaker or ICD implantation and were treated with dual antiplatelet therapy, a hematoma rate of 7.2% was reported.[13] Thus, the existing series suggest that patients with recent or ongoing treatment with dual antiplatelet therapy or clopidogrel alone have an increased hematoma risk. In both of the prior analyses, patients treated with aspirin only had similar hematoma rates as patients who had no form of anticoagulation or antiplatelet therapy.[12,13]

There are limited data evaluating the novel oral anticoagulants (NOACs). One prospective observational series evaluated major and minor bleeding complications in patients treated with dabigatran who underwent an initial pacemaker, ICD, cardiac resynchronization therapy (CRT), or generator replacement.[14] All patients had axillary vein access and active fixation leads. No major or minor bleeding events were noted in the index hospitalization; one hematoma occurred after the index hospitalization in a patient with a $CHADS_2$ score of 6 treated with both dabigatran and dual antiplatelet therapy.

The European Heart Rhythm Association recently published consensus guidelines on antithrombotic management in patients undergoing electrophysiologic procedures and, citing the lack of prospective data, suggested interruption of NOACs without heparin bridging.[15] The period of perioperative discontinuation depends on both the creatinine clearance and pharmacokinetics of the individual agents. The periods range from 24 to 36 or more hours. Similarly, reinitiation of NOACs after device surgery is suggested to be 24 to 48 hours after the procedure.

Topical agents used in the operating room to decrease bleeding have also been used in CIED procedures. One



• **Fig. 13.2** Routine (A) posterior-anterior and (B) lateral chest radiographs of implantable cardioverter-defibrillator lead with externalized conductor.

prospective trial case-control study of 163 patients evaluated a topical agent (composed of thrombin 5000 U + collagen 200 mg + diluents) in CIED procedures.[16] The primary endpoint was the development of a hematoma requiring evacuation or a pocket infection. The rate of pocket hematoma and infection was 14.6% in patients treated with the topical thrombin agent D-Stat Flowable Haemostat versus 3.7% in the control group. The trial stopped at the interim analysis owing to an increased rate of pocket infections, and use of this particular agent was not associated with a decrease in hematomas.

Tip: Avoid heparins, pay careful attention to hemostasis, and consider topical thrombin.

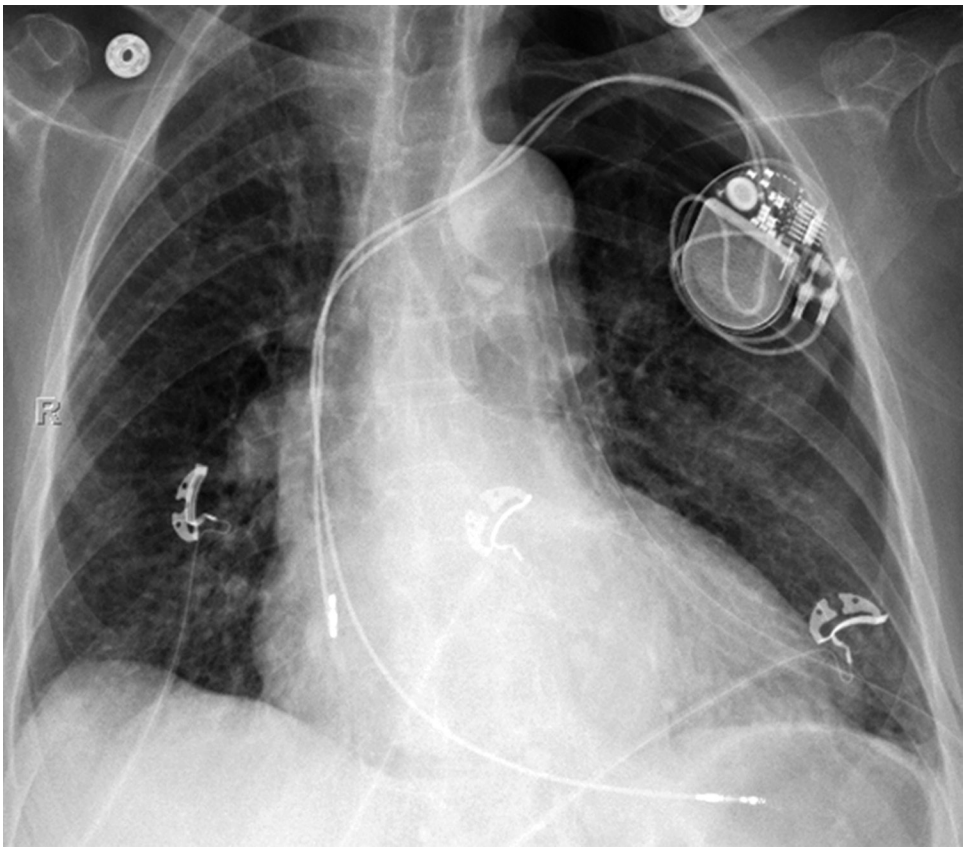
Pneumothorax and Its Variants

Pneumothorax, hemothorax, hydropneumothorax, and tension pneumothorax are infrequent but significant complications after transvenous CIED implants (Figs. 13.3 and 13.4). Occasionally, a small asymptomatic pneumothorax can be managed expectantly, but hemothorax, hydropneumothorax, and tension pneumothorax usually require intervention with a chest tube. As shown in Table 13.1, the incidence and prevalence are usually less than 1%. The potential role of a vascular access technique on pneumothorax incidence was evaluated in a small series from the Johns Hopkins Hospital, which compared the cephalic direct venotomy approach versus extrathoracic subclavian vein access.[17] Two hundred patients were randomized to lead placement by the cephalic vein or the extrathoracic subclavian or axillary vein. The rate of successful implantations was 64% with a 1% pneumothorax rate in the cephalic group, and 99% with a 3% pneumothorax rate in the extrathoracic subclavian access group. Interestingly, the pneumothorax seen in the patient with cephalic access occurred on the contralateral side. It is certainly reasonable to consider local venography before any procedure involving the addition of new leads, and it may be reasonable before de novo implantations as well.

Tip: Evaluate surface anatomy of the clavicle and first rib, perform direct venotomy, and avoid supports between the scapulae.

Venous Access and Obstruction

What makes venous access to the heart difficult or impossible? Some common reasons are prior CIED procedures, access for hemodialysis, prior malignancy, or radiation or chemotherapy.



• **Fig. 13.3** Left-sided pneumothorax on chest radiograph.

In patients with repaired congenital heart disease, venous anomalies, such as in heterotaxy syndromes, or prior surgical palliation, such as Glenn shunts, can challenge even experienced implanters.

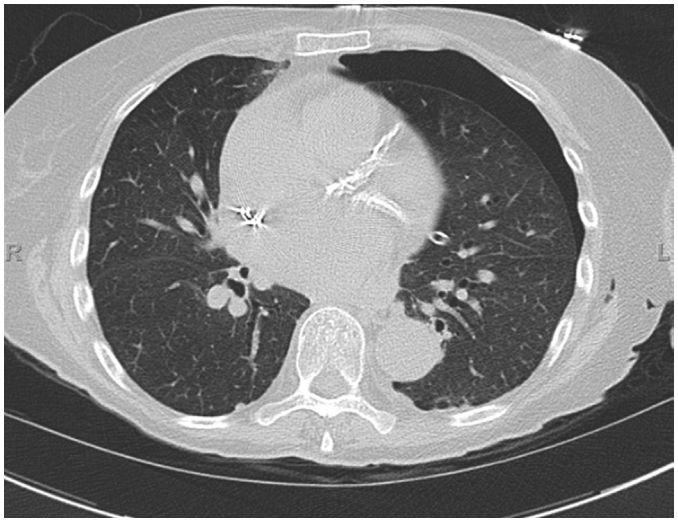
Venous obstruction can also be present, and a clue can be dilated veins across the pocket (Fig. 13.5). Identified risk factors for venous obstruction include the number of preexisting leads, dual-coil ICD leads, prior deep venous thrombosis, and hormone replacement therapy.[18,19] Three general locations of venous obstruction in the subclavian and innominate veins have been described. Peripheral, located from the mid- to distal subclavian vein, is the most common at 61%; central stenoses, involving the innominate vein, are the least common at 17%; and peripheral and central stenosis accounts for the remaining 21%.[20] An example of central subclavian stenosis is shown in Fig. 13.6. Fig. 13.7 shows the collateral veins that develop proximal to a stenosis. Venoplasty techniques have been proposed as a way to reduce the need for medial subclavian access, an approach that has been associated with increased risk of hemothorax.

A specific infrequent complication associated with CRT procedures is a coronary sinus (CS) dissection (Fig. 13.8). This can occur with wire manipulation or sheath introduction into the CS. This complication was noted once in the group of patients who underwent the addition or revision of a left ventricular lead in the REPLACE Registry.[4]

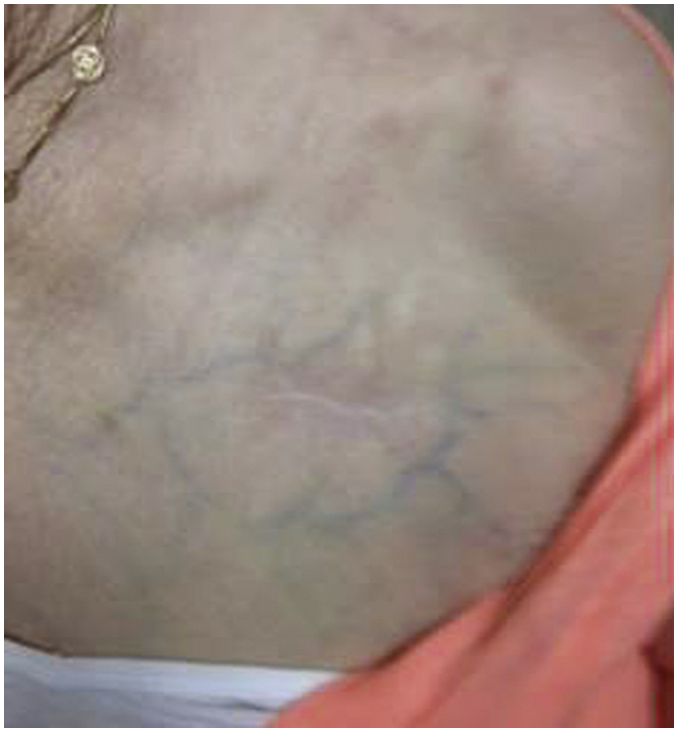
Tip: Minimize instrumentation of central veins, and use the minimum number of leads necessary.

Infections

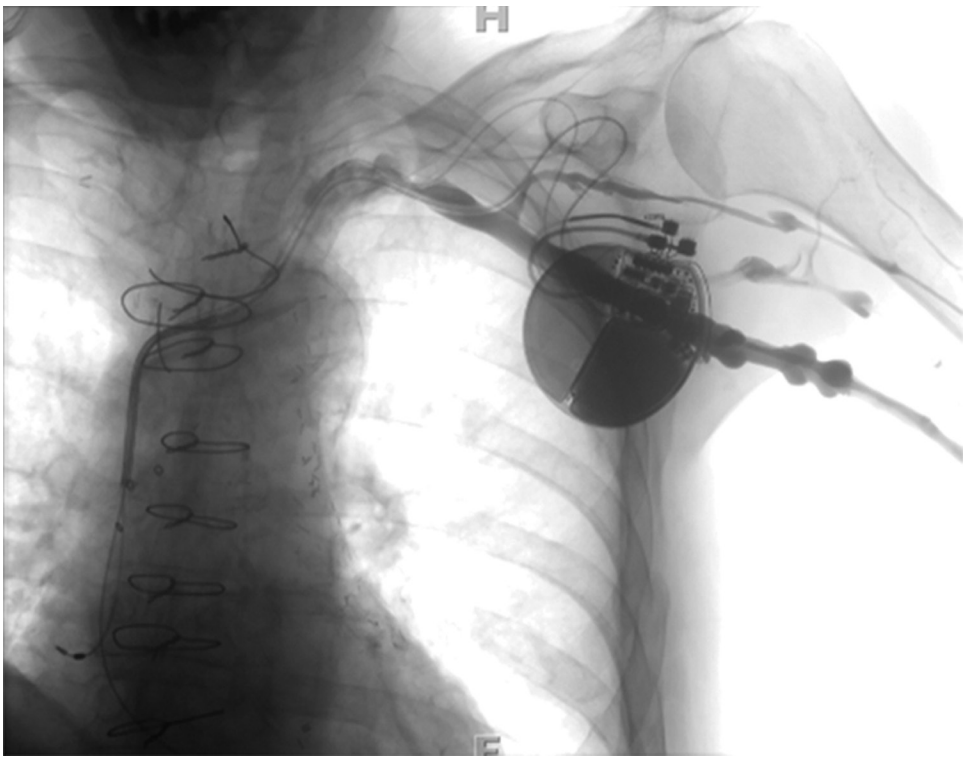
CIED infections are associated with serious morbidity and, if associated with endocarditis, death. The impact on a pacemaker-dependent patient, who may require a prolonged hospitalization with an externalized temporary device, possible extraction, and subsequent procedures, is not trivial. The spectrum of infections includes pocket cellulitis, bacteremia, endocarditis, and sepsis syndromes.[21] Lead and generator erosions are also considered infections as the integrity of the pocket has been compromised (Fig. 13.9). Multiple retrospective and prospective trials have been performed to define the incidence and risk factors for CIED infection. Comorbidities such as diabetes mellitus, congestive heart failure, chronic kidney disease, oral anticoagulant use, hematoma development, and steroid use have been identified as contributing factors. Procedural factors such as fever within 24 hours of the procedure, the use of temporary pacing leads, and early reintervention after the index procedure are also contributing factors.[22] The risk of infection was shown to be mitigated by antibiotic prophylaxis before the skin incision and now administering preoperative intravenous antibiotics before the skin incision is considered mandatory. A landmark clinical trial from Brazil evaluated cefazolin versus placebo in patients undergoing CIED implantation, with clinical follow-up to 6 months.[23] This trial stopped early because of the treatment benefit, with an infection rate in the cefazolin arm of 0.63% compared with 3.28% in the control arm (P = .016). In addition, one emphasis of the REPLACE Registry was the incidence of infection. In that registry, the infection rate was low, likely owing to all patients receiving preoperative antibiotics. Furthermore, infection was associated with the development of a hematoma. The REPLACE Registry infection analysis also evaluated infection by participating site. Sites with infection rates of greater than 5% of enrolled



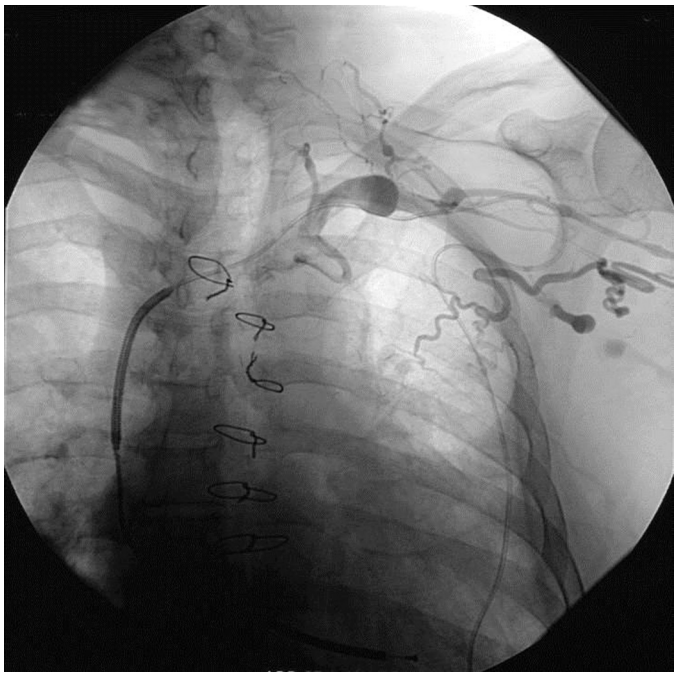
• **Fig. 13.4** Left-sided pneumothorax on chest computed tomography scan.



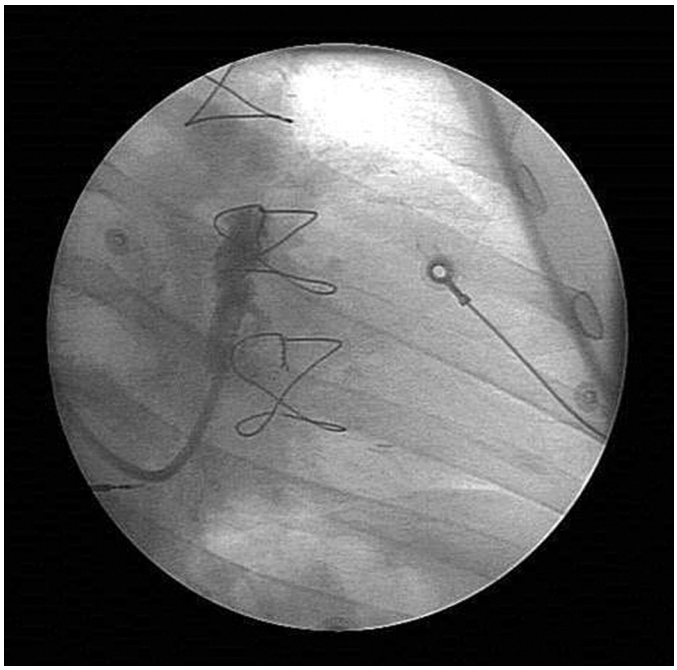
• **Fig. 13.5** Dilated surface veins over generator pocket.



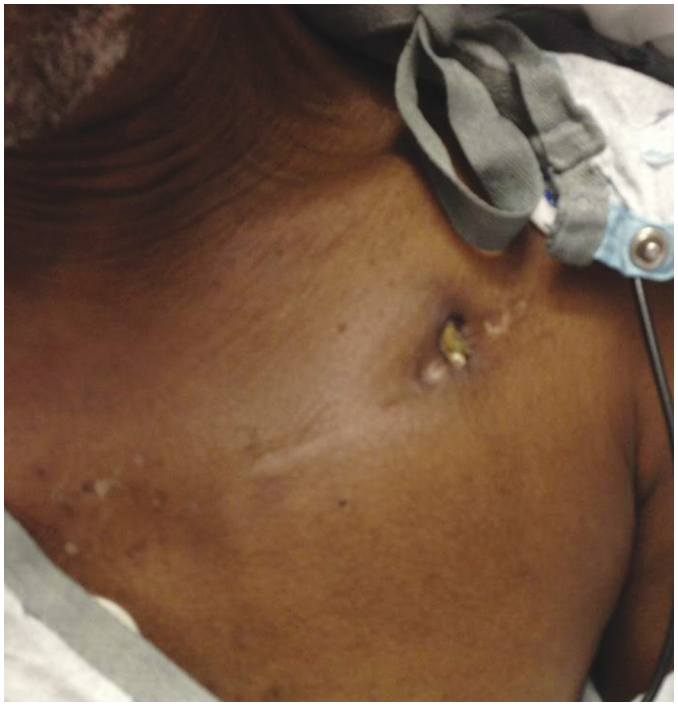
• **Fig. 13.6** Proximal subclavian vein stenosis on contrast venography.



• **Fig. 13.7** Subclavian vein occlusion with collaterals on contrast venography.



• **Fig. 13.8** Coronary sinus dissection with intramyocardial contrast staining.



• **Fig. 13.9** Lead erosion through skin.

patients were more likely to have sicker patients with greater Charlson Comorbidity Index scores, were more likely to use povidone-iodine skin preparation versus chlorhexidine gluconate, and were more likely to implant fewer than 250 CIEDs in a year.[24] A subsequent trial of surgical skin preparation demonstrated the superiority of chlorhexidine gluconate over povidone-iodine skin antisepsis; however, neither CIED procedures nor any orthopedic procedures were included in the trial.[25]

An antibacterial envelope containing minocycline and rifampin has been developed to reduce the risk of CIED infection. The pulse generator and the extravascular portion of the leads are placed into the envelope and then placed into a slightly enlarged subcutaneous pocket. One of the initial experiences with this envelope was described in a retrospective cohort trial performed at 10 centers. The primary endpoint was successful CIED implantation and CIED infection. Six hundred and twenty-four patients were enrolled, and only three patients (0.5%) developed an infection. The Worldwide Randomized Antibiotic Envelope Infection Prevention Trial (WRAP-IT, NCT02277990) is an ongoing prospective multicenter trial of an absorbable form of this envelope and is likely to provide more clinical information on efficacy.

Tip: Prep skin with chlorhexidine-gluconate, complete infusion of preoperative antibiotics, critically evaluate need for temporary pacing leads, and avoid procedures on febrile or actively infected patients.

Perforation

Cardiac perforation is one of the most feared complications after transvenous lead implantation. Two presentations are recognized: (1) acute tamponade and (2) subacute perforation with or without late development of a pericardial effusion, chest wall stimulation, pain, shortness of breath, hiccups, and/or changes in lead performance.[27,28] In one series spanning 8 years of pacemaker placement from the Mayo Clinic, a temporary pacing lead, helical screw-in leads, and oral steroid use were identified in multivariate analysis as independent risk factors for cardiac perforation. Extending the screw of an active fixation lead through a potentially thin section of atrial appendage has resulted in perforation.[29]

Tip: Handle leads gently, avoid temporary wires, discontinue steroids, consider apical septal ventricular lead position, and make appropriate lead choices by weighing clinical factors and knowledge of biomechanical properties of different pacing leads.

Death

The most significant complication to occur after any procedure is death. Because CIEDs are implanted in patients with cardiovascular disease, and because cardiovascular disease remains a significant cause of death, recent analyses have attempted to evaluate the risk of death after device procedures.[5,6] An analysis from the National Cardiovascular Data ICD Registry evaluated in-hospital mortality rates in over 250,000 patients who had ICD implants. The in-hospital mortality rate was 0.38%. Seven factors were identified in multivariable analyses as contributors to in-hospital death: New York Heart Association (NYHA) heart failure class, history of cardiac arrest, atrial fibrillation or flutter, blood urea nitrogen greater than 30 mg/dL, serum creatinine level greater than 2.0 mg/dL, systolic blood pressure less than 100 mm Hg, and reason for admission other than elective device implant.[6] A similar risk analysis for death was constructed from the REPLACE Registry.[5,30] In this analysis, risk factors for death after CIED replacement included higher NYHA class, heart failure hospitalization within the past 12 months, use of antiarrhythmic medications, cerebrovascular disease, chronic kidney disease stage, and advanced age. Interestingly, the occurrence of a complication was not identified as a contributor to mortality rates in either of those series.

Tip: Assess active medical issues and consider delaying invasive procedures in patients with acute decompensated status.

TABLE 13.2 Summary of Strategies to Reduce Complications

Complication	Strategy
Lead malfunction	Handle leads gently
Hematomas	Avoid heparin bridging
	Implant during continued oral anticoagulation
	Discontinue novel oral anticoagulants for 1–2 days preprocedure
Pneumothorax/hemothorax	Use cephalic access
	Perform venoplasty
Infection	Use chlorhexidine-gluconate skin prep
	Administer preoperative antibiotics
	Avoid temporary pacing leads
	Reduce hematoma risk
	Delay implant in presence of fever
Perforation	Avoid temporary pacing leads
	Reduce or discontinue oral steroids
	Use apical septal ventricular lead position
	Choose appropriate lead
Mortality	Assess comorbidities

Summary and Quality Improvement

This chapter reviews the major complications associated with CIED implantation and has proposed approaches for reducing complications, summarized in Table 13.2. As the clinical trial evidence continues to grow, implanting physicians are provided further information that can be used to implement quality improvement measures. Thus, the confirmation that intravenous antibiotics were administered can be added to a preprocedural briefing. Two sentinel events were identified in the REPLACE Registry: the presence of a retained surgical sponge and the implantation of a CIED generator past the use-by date. An online radiologic image library exists to help recognize these retained objects.[31] Accordingly, confirming the use-by date when devices and leads are handed off in an implant procedure and noting that the sponge and instrument counts were correct in the postprocedure debriefing may help avoid such events.

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14

Prevention, Evaluation, and Management of Cardiac Rhythm Device Infections

JORDAN M. PRUTKIN, PAUL POTTINGER

Introduction

The rate of cardiac implantable electronic device (CIED) infection has been rising rapidly,[1] and it is likely that any operator who implants enough devices will have a patient develop an infection. Infection can cause significant morbidity, mortality, and financial costs; best practices for prevention and treatment should be followed to minimize these effects. This chapter presents systematic approach to the diagnosis and treatment of suspected infections.

Epidemiology

It is difficult to accurately determine the rate of infections because of significant heterogeneity among publications on this topic. Studies have used pacemakers versus implantable cardioverter-defibrillators (ICDs), initial implants versus generator changes, transvenous versus epicardial systems, and so forth. Abdominal generators have a significantly higher rate of infection[2] but are used less frequently now, so these data may not be applicable to current patients. Most importantly, there has been a significant difference in the duration of follow-up in the literature. Although several studies have demonstrated that most patients presented within 1 year of implant,[3–5] a significant proportion present more than 1 to 2 years after their last procedure.[5–7] For instance, one study of pocket infections demonstrated that almost 50% occurred more than 12 months after the last device procedure, with one presenting more than 17 years later.[7] Those with late infections are more likely to have pocket erosion or valvular vegetations, and those with earlier infections are more likely to have inflammatory signs at the pocket.[7] Another study showed that those with epicardial leads or pocket complications were more likely to have early infections, whereas longer hospitalization at implantation and pulmonary disease increased the risk of late infections.[8] Because there is no time without risk of infection, any study with a fixed time duration must underestimate the lifetime risk of infection.

With these caveats, the best estimate of the current incidence of CIED infection is between 0.5% and 2.2% per device.[9–14] A recent metaanalysis found an overall infection rate of 1.2%, although it was 1.6% if superficial wound infections were included.[15] The largest study examining the rate of ICD infection combined data from the National Cardiovascular Data ICD Registry and Medicare claims using administrative codes.[16] Over 200,000 ICD implants over a 4-year period were followed for 6 months. A total of 3390 infections were found, for an overall rate of 1.7%. The greatest risk of infection was in the first 30 days, with a rate of 0.8% during this time, but the risk continued over time at 1.2% at 60 days, 1.4% at 90 days, and 1.6% at 120 days. The number of leads also influenced infection rate. Biventricular devices had a rate of 2.0%, dual chamber was 1.5%, and single chamber was 1.4%. In addition, generator changes or lead additions had a higher rate of infection compared with initial implants (1.9% vs. 1.1%).

That said, the number of CIED infections is increasing, and the rate seems to be rising faster than the rate of CIED implantation. Data from the 1990s showed an increase in CIED infections in Medicare patients over the course of the decade while the rate of endocarditis remained essentially flat.[17] A study of the National Hospital Discharge Survey showed an increase in CIED infections starting in 2001[18] that persisted through 2006.[19] Another study from the National Inpatient Sample demonstrated a relatively constant rate of CIED infections until 2004, when the rate significantly increased over the next 4 years.[1] This was directly correlated with an increase in patient comorbidities, including heart failure, renal failure, respiratory failure, and diabetes mellitus.

Risk Factors

The reasons for CIED infection depend on the patient, the device, and the organism.

Patient and Procedural Risk Factors

Risk factor analysis is difficult because the studies do not examine the same risk factors. The major patient-related risk factors that have been demonstrated most consistently are diabetes mellitus, end-stage renal disease or renal insufficiency, malignancy, chronic obstructive pulmonary disease, congestive heart failure, corticosteroid use, and oral anticoagulation.[15] The major procedural-related factors are longer procedure duration, noninitial implant, dual-chamber system, abdominal pocket, use of temporary pacing, postoperative hematoma, and need for lead repositioning.[15]

The previously mentioned study from the ICD Registry and Medicare data showed that the biggest risk factor for ICD infection was an adverse event at the time of implantation.[16] Those with a periprocedural adverse event had a 5.4% chance of infection. This was primarily driven by either a lead dislodgement or a hematoma that was evacuated. The common denominator with these is that both required an early reintervention into the ICD pocket. The other risk factors on multivariate analysis associated with ICD infection were prior valvular surgery, a device upgrade or generator change for malfunction or being under advisory, renal failure on dialysis, chronic lung disease, cerebrovascular disease, and warfarin use.

Hematoma and other perioperative complications have repeatedly been demonstrated as risk factors.[14,20–22] In addition, reentering a pocket for a lead addition or generator change has been associated with higher risk when compared with initial implantation,[5,9,14,16,22,23] and there may be a time-dependent phenomenon in which reentering a pocket sooner after implantation is the greatest risk factor. It has been postulated that the reason for this is the introduction of bacteria into the pocket at the time of device placement that then colonize the tissue around the device without leading to overt infection. One study cultured the preaxillary flora of patients undergoing initial implantation, the pocket just after it was made, and the pocket after generator insertion before suturing it closed.[24] Forty-eight percent of pockets after being initially made and 37% of those at the end of the procedure had positive cultures, most commonly *Staphylococcus epidermidis*, although it should be noted that perioperative antibiotics were not used in this study. Other studies have demonstrated that it is possible to culture bacteria or amplify bacterial DNA from asymptomatic pockets when reentering a pocket for generator change in up to 42% of patients.[25–27] It is thought that this disrupts the homeostasis between the bacteria and immune system, resulting in a clinical infection.

Warfarin use has shown mixed results as to whether it increases CIED infection risk.[5,14,16,21,28] The presumed mechanism for warfarin use is an increase in hematomas, although other mechanisms cannot be excluded. Hematomas may increase the risk of infection because they can provide a favorable milieu for bacterial growth or lead to impaired wound healing or dehiscence. Clopidogrel has been shown to increase the risk of hematoma,[29–32] but on multivariate analysis in one study was not associated with infection.[16]

Renal disease has repeatedly been identified as a risk factor for infection.[1,5,16,33] This is likely because of an increase in vascular access, indwelling catheters, and bacteremia as well as impaired immune function.[34,35]

Prior valve surgery is a potent risk factor, partly because of how CIED infections are defined. If a prosthetic valve has endocarditis and a CIED is present, it is presumed that the device itself is also infected, especially if the organism is *Staphylococcus aureus* or *Candida* species.[36] Prosthetic valves have a 50-fold higher rate of infection compared with native valves.[37] That said, the Mayo Cardiovascular Infections Study Group has not found prior valve surgery to be a risk factor in their cohorts.[23,38]

The ICD Registry/Medicare study, even with a very large cohort and examining over 40 perioperative factors, only had a C-statistic on the multivariate analysis of 0.676. Other studies have not published this value. This implies that the known risk factors for infection have very low predictive accuracy, and we have done a poor job defining the full spectrum of risk factors.

Organism Virulence Factors

Organisms attach either directly to the device or to host proteins such as fibrinogen, fibronectin, vitronectin, or collagen that have coated the CIED.[39–42] Some bacteria such as staphylococci ("staph") also have proteins known as microbial surface components recognizing adhesive matrix molecules (MSCRAMM) that enhance adhesion to the extracellular matrix.[42–44]

Staph species and other organisms, including gram-negative bacteria, can thrive on CIEDs after cell-device and cell-cell adhesion because of the creation of biofilm (also known as slime), which includes the organism and extracellular polymers.[42,45] Organisms within the biofilm enter a relatively inactive state, replicate slowly, and are less likely to be sensitive to antibiotic medications.[45] They are also fairly well protected from the host immune system in this location. Because of these features, antibiotics alone can rarely cure a CIED infection, and recurrence is common after antibiotics are stopped.

CIED Influence on Infection

Little is known about how properties of the CIED influence risk of infection. One reason is that the interactions between synthetic material and an organism in vitro may not reflect those interactions in vivo. Synthetic hydrophobic materials and those with greater surface area or irregular surfaces are more likely to promote bacterial adhesion.[41,43,44] As a general principle, polyethylene promotes bacterial adherence more than polyurethane, silicone more than polytetrafluoroethylene (PTFE), and stainless steel more than titanium.[44] However, no material has yet been described that is "biofilm proof."

Prevention

Antibiotics

One of the most important ways to prevent CIED infection is the use of periprocedural antibiotics. A 1998 metaanalysis

of seven studies laid the groundwork for the use of antibiotics by demonstrating their effectiveness in preventing infections, but the data were problematic because most of the studies were nonblinded.[46] Later, a randomized controlled trial demonstrated that a single dose of intravenous cefazolin (a first-generation cephalosporin) significantly reduced the rate of CIED infection from 3.3% to 0.6% compared with placebo.[20] Significantly, this finding was seen even in those considered highest risk, such as those with prosthetic heart valves, lead dislodgement, or immunosuppression. A more recent metaanalysis that included this and other studies confirmed that perioperative prophylaxis does reduce the rate of infection complications with an odds ratio of 0.32 (95% confidence interval 0.18–0.55).[15]

Cefazolin given within 1 hour of the procedure is the recommended agent for prophylaxis. If there is a high rate of methicillin-resistant *S. aureus* (MRSA) at the implanting institution, some recommend the use of vancomycin 90 to 120 minutes before the procedure in addition to, but not in place of, cefazolin. Although vancomycin's spectrum "covers" the gram-positive skin commensals of concern—methicillin-sensitive *S. aureus* (MSSA), MRSA, and coagulase-negative staphylococci—its killing power is clearly inferior to that of cefazolin for β-lactam-susceptible strains. When vancomycin was used to replace cefazolin for perioperative prophylaxis in cardiothoracic surgery, the rate of deep MSSA infections rose. Thus, our recommendation is to use cefazolin—alone or in combination with vancomycin—whenever possible.[47] If there are allergies to both agents, linezolid or daptomycin may be used. Rifampin has been shown in a mouse model to reduce the rate of colony-forming units when added to cefazolin or vancomycin, but this has not been studied in humans for this indication.[48] Rifampin should never be dosed alone, owing to high risk of resistance, and it carries a risk of significant interactions with other medications, especially warfarin.

Although 8% of the U.S. population reports a penicillin allergy[49] and 1% reports a cephalosporin allergy,[50] the rate of true immunoglobulin E–mediated β-lactam allergy in those claiming a history is low. A stated history of anaphylaxis or breathing difficulty is more reliable, but otherwise the history may be suspect and skin testing can be completed.[51,52] In those with a skin test positive for a penicillin allergy, the rate of a cephalosporin allergy is only about 2%.[53] In those with a history of "rash" as the manifestation of a penicillin allergy, cephalosporins can still be used. Vancomycin can be used in those with a true cephalosporin allergy.

The optimum dose of antibiotics depends on the adult patient's body weight: a single dose of cefazolin should be 2 g for adults weighing 120 kg, but 3 g for heavier patients. Vancomycin should also be dosed by weight, at 15 mg/kg. This can be generally rounded to the nearest 250 mg, usually with a maximum dose of 2 g.

A single dose of antibiotics immediately before incision is the standard of care for most other elective surgeries. Whether to give any postoperative intravenous antibiotics for 24 hours or to continue oral antibiotics for several days afterward has not been studied. The American Heart Association (AHA) specifically recommends against using postoperative antibiotics because of risk of antibiotic resistance, costs, and adverse events.[36]

The TYRX antibacterial envelope (Medtronic) is a pouch embedded with minocycline and rifampin that elutes over 7 days. Although the initial design was nonabsorbable, there is now a version that absorbs within about 9 weeks. Single-center studies have suggested there may be a benefit in reducing infections, although these were nonrandomized.[54–56] Preliminary results from two related prospective studies (Centurion and Citadel) that compared the envelope to historical controls demonstrated only a 0.44% rate of infection in 1129 patients over 12 months.[57] The ongoing Worldwide Randomized Antibiotic Envelope Infection Prevention Trial (WRAP-IT) is a single-blind randomized study of the envelope in those undergoing a generator change, new lead addition, or new cardiac resynchronization therapy–defibrillator (CRT-D) (clinicaltrials.gov identifier: NCT02277990). The study should be completed by December 2017.

One issue that can be difficult is the timing of implantation in those with a possible active infection who may or may not be on antibiotics. In the presence of active endocarditis or bacteremia, there is a significant risk that a new implant will become seeded with the infectious organism; in these patients it is best to delay permanent device implantation. In those with less serious infections, such as an uncomplicated urinary tract infection, it is prudent to wait as long as possible unless there is an urgency to implantation.

Skin Preparation

The night before and morning of the procedure, the patient should shower with a chlorhexidine solution to reduce bacterial counts on the skin.[58,59] Improved dosing occurs with a 5-minute dwell time on the skin, a detail often overlooked. There is a question of whether patients should receive intranasal mupirocin or other method to reduce the rate of *S. aureus* colonization. Although one study reported that mupirocin or other method may reduce cardiac and orthopedic surgical site infections,[60] there are no data in the CIED literature regarding their efficacy.

Skin should be clipped instead of shaved to prevent skin abrasion.[59] The skin should be cleaned before draping to ensure the area is without debris and to reduce the burden of skin flora at the surgical site. Antiseptic soap with chlorhexidine is preferred over povidone-iodine.[61] Although many apply the soap in concentric circles starting from the incision site, there is no evidence to suggest this is better than a horizontal back-and-forth method.

The operator should wear scrubs, a cap, and shoe covers and must perform an appropriate sterile scrub with an alcohol solution or antiseptic soap. Again, chlorhexidine gluconate may be more effective than povidone-iodine.[62–64] The operator must use a sterile gown and gloves, and there should be appropriate barrier protections to maintain a sterile field. A sterile technique should be maintained throughout the procedure.

Hematoma and Anticoagulation

As noted earlier, several studies have demonstrated that there is an increased risk of infection if there is a postoperative hematoma, although the risk is higher if the hematoma is evacuated versus letting it resorb on its own. Therefore, unless the pocket has dehiscence or significant wound tension, there should be conservative management. A pressure dressing may be helpful in this situation.

Prevention of hematomas is influenced by anticoagulation and antiplatelet medications. If possible, these agents should be stopped without bridging therapy. Low-molecular-weight heparin is significantly associated with hematoma development and should not be used.[65] It is better to do continuous warfarin than unfractionated heparin.[66,67] Preliminary data suggest that uninterrupted administration or holding one dose of a novel anticoagulant may also be safe,[68,69] and it is thought that the use of clopidogrel may also increase bleeding risk.[30,69]

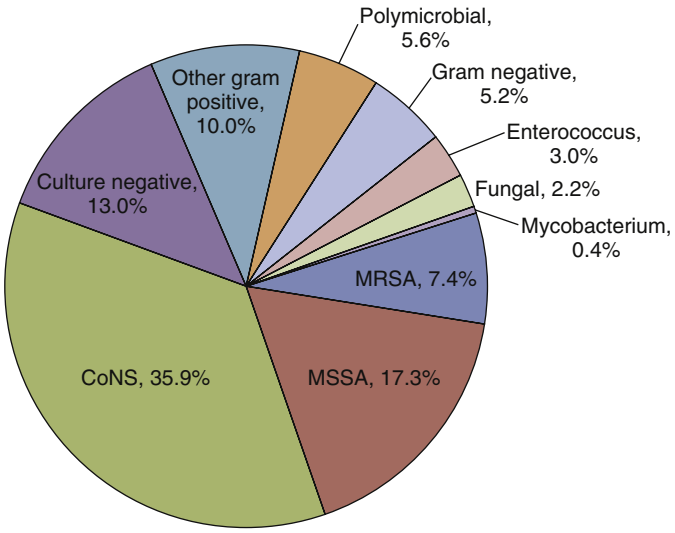
Some operators remove the pocket surrounding the generator and leads with the thought that this can reduce the chances of infection. Removing the pocket can potentially remove pathogenic organisms residing within it and may allow better penetration of inflammatory cells into the area surrounding the device. It is also possible, however, that this process will lead to a higher hematoma rate, which may lead to more infections. More research on this is needed.

Microbiology

Gram-positive bacteria are clearly the most common infectious agents (Fig. 14.1). Coagulase-negative staphylococci (CoNS) and *S. aureus* are the two infectious agents seen most frequently.[6,7,36,70–77] The relative contribution of each agent is dependent on the percentage of cases of pocket infection versus endocarditis and the duration of follow-up, because pocket infections are slightly more likely to be from CoNS, especially if followed for a long duration,[7] whereas *S. aureus* is more likely to be the agent for endocarditis.[70]

S. aureus comes in two broad categories: MSSA and MRSA. Both are very challenging to treat in CIED infections, but MRSA more so owing to its resistance to most β-lactam antibiotics. The proportion of infections caused by MRSA varies by practice location; knowing this local frequency is important. However, it represents a significant burden nationwide and may influence the choice of empiric antibiotics.

Other gram-positive organisms may be seen, with viridans group streptococci usually presenting early and enterococcus presenting late.[70] *Propionibacterium acnes*, corynebacteria, gram-negative bacteria, fungi (especially *Candida* species), nontuberculous mycobacteria, and other agents may be detected.[36] Polymicrobial infections may also occur, and culture-negative cases occur with some frequency. Multiple sets of blood cultures, especially before antibiotics are given; sending tissue for culture at the time of extraction; and working with an infectious diseases specialist may help to establish a firm microbiologic diagnosis.



• **Fig. 14.1** Infectious organisms cultured from cardiac implantable electronic device infections at the University of Washington Medical Center, 2000–2015. *CoNS*, coagulase-negative *Staphylococcus*; *MRSA*, methicillin-resistant *Staphylococcus aureus*; *MSSA*, methicillin-sensitive *Staphylococcus aureus*.

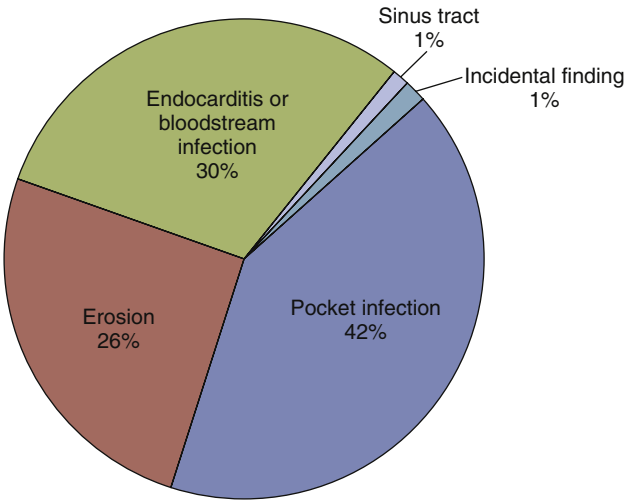
It is thought that in most cases the CIED is the initial focus of the infection. However, *S. aureus* bacteremia is known to frequently seed an otherwise noninfected CIED. In fact, up to 73% of patients with a CIED who develop bacteremia may develop a CIED infection,[78] especially if the patient has an ICD instead of a pacemaker, likely because of differences such as patient characteristics in this population,[79] although a difference in the properties of the ICD generator or lead that can dispose to seeding cannot be ruled out. Gram-negative bacteria rarely lead to secondary seeding of a CIED.[80]

Presentation

Patients with CIED infection can have a localized generator or lead erosion, a pocket infection, CIED-associated endocarditis, or unexplained bloodstream infection or sepsis in the presence of a device (Fig. 14.2).

Pocket infections present with erythema, tenderness, warmth, pain, or drainage at the generator site (Fig. 14.3). Some patients will also have systemic signs of fever, malaise, fatigue, or anorexia. A high degree of suspicion may sometimes be necessary to make the correct diagnosis. Those with a device erosion have an exposed portion of the generator or lead through the skin. Some patients may have impending erosion, where the skin appears thin and is tethered to the underlying CIED. In certain cases, a pocket revision may salvage the situation if the device is not yet infected. Most erosions, however, are due to subclinical pocket infection that eventually comes through the skin. If there is any portion of the CIED system that is exposed, the whole system is considered infected with skin flora or other microorganisms.

CIED-associated endocarditis or unexplained bloodstream infections in the setting of a cardiac device can present with acute illness and sepsis and can progress rapidly to acute respiratory distress syndrome and multiorgan failure. Other times, the presentation may be more insidious and difficult to diagnose.



• **Fig. 14.2** Types of cardiac implantable electronic device infection at University of Washington Medical Center, 2000–2015.

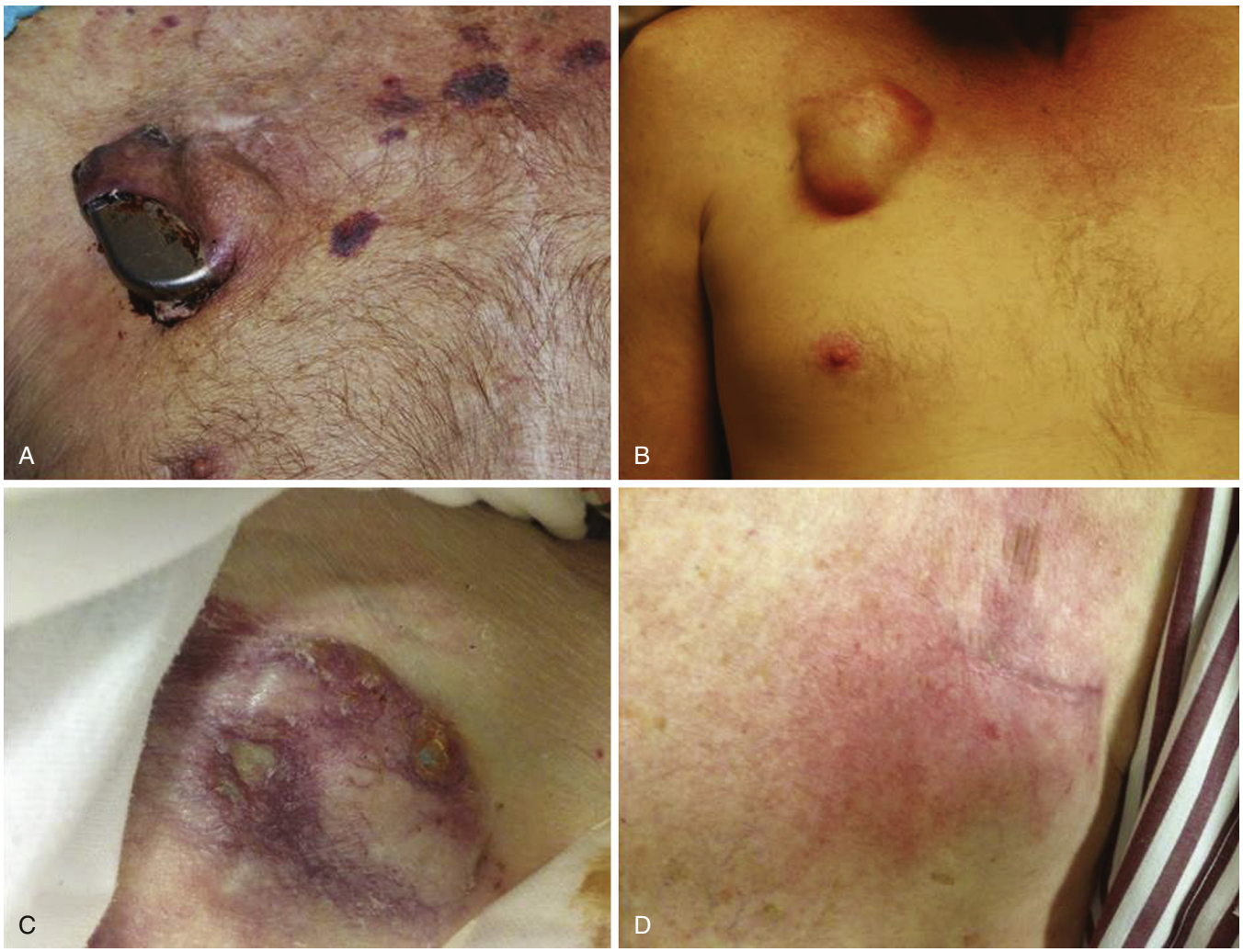
Symptoms including fever, dyspnea, and anorexia can be present. Patients may or may not have signs of heart failure, physical manifestations of infectious endocarditis such as Osler nodes or Janeway lesions, and embolic pulmonary events. Rarely, patients may have localized extension of an infection including chest wall abscess, clavicular osteomyelitis, or intracardiac abscess.

Diagnostic Evaluation

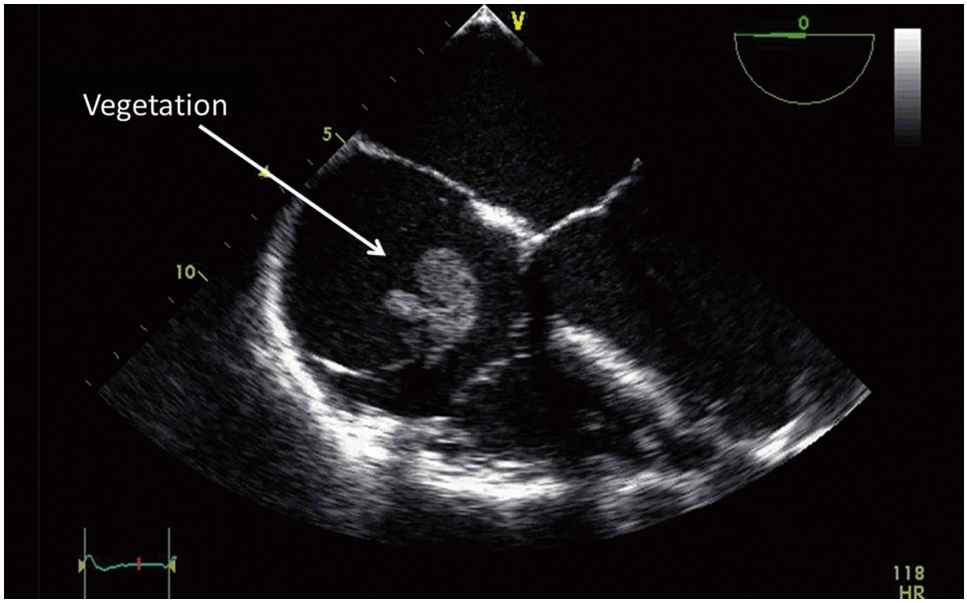
Laboratory Testing

All patients should undergo basic laboratory testing, including a complete blood count with differential to evaluate for leukocytosis. Erythrocyte sedimentation rate and C-reactive protein may also be checked if the diagnosis is in doubt, although the positive predictive value of elevations of these values is poor.

Two sets of blood cultures should be obtained before starting antibiotics even in those with only localized signs of pocket infection, because up to 30% of patients with a pocket infection may also have bacteremia, and management in the setting of bacteremia is different from simple soft tissue infection.[7]



• **Fig. 14.3** Four presentations of pocket infection. (A) Obvious pocket erosion. (B) Edema and intense erythema. (C) Hypervascularity, erythema, and erosion. (D) Mild erythema.



• **Fig. 14.4** Large vegetation on a lead in the right atrium seen on transesophageal echocardiography.

Imaging

A transthoracic echocardiogram (TTE) should be obtained on every patient with suspected CIED infection. Vegetations on leads or valves can sometimes be seen, but a TTE can also assess valvular and left ventricular function, pulmonary pressures, and pericardial effusion. TTE, however, cannot be used to rule out the presence of intracardiac vegetations, and in patients with positive blood cultures or in those whom antibiotics were started before blood cultures were taken, a transesophageal echocardiogram (TEE) should be obtained (Fig. 14.4).[36] The TEE can also be helpful to assess whether there is paravalvular extension of endocarditis, such as an intracardiac abscess, or whether there are left-sided valvular lesions, vegetations, or adhesions in the superior vena cava.

For some patients, TEE imaging can be completed at the time of lead extraction, as described later, because it is a useful adjunct during the extraction procedure. The benefit of preoperative TEE is if there is very large vegetation suspected that may be better treated by an open surgical procedure versus percutaneous extraction.

Intracardiac echocardiography (ICE) may be useful if there is a question of a vegetation.[81] ICE is more sensitive for picking up intracardiac masses than TEE [82] but involves intravascular access, with a small risk of vascular complications, increased cost, and need for operator experience. If it is used, it is done most commonly at the time of an extraction procedure.

There must be caution in interpreting all masses on leads as infections because thrombus is a possibility also. One autopsy study demonstrated that more than one-third of patients may have thrombi on leads, more commonly on the atrial lead. [83] If a mass is seen incidentally on a lead during TEE performed for other reasons, in the absence of clinical signs or symptoms of infection, it is unlikely to be an infection and antibiotics and/or extraction are not indicated. Blood cultures may be considered if there is any suspicion of infection for other clinical reasons.

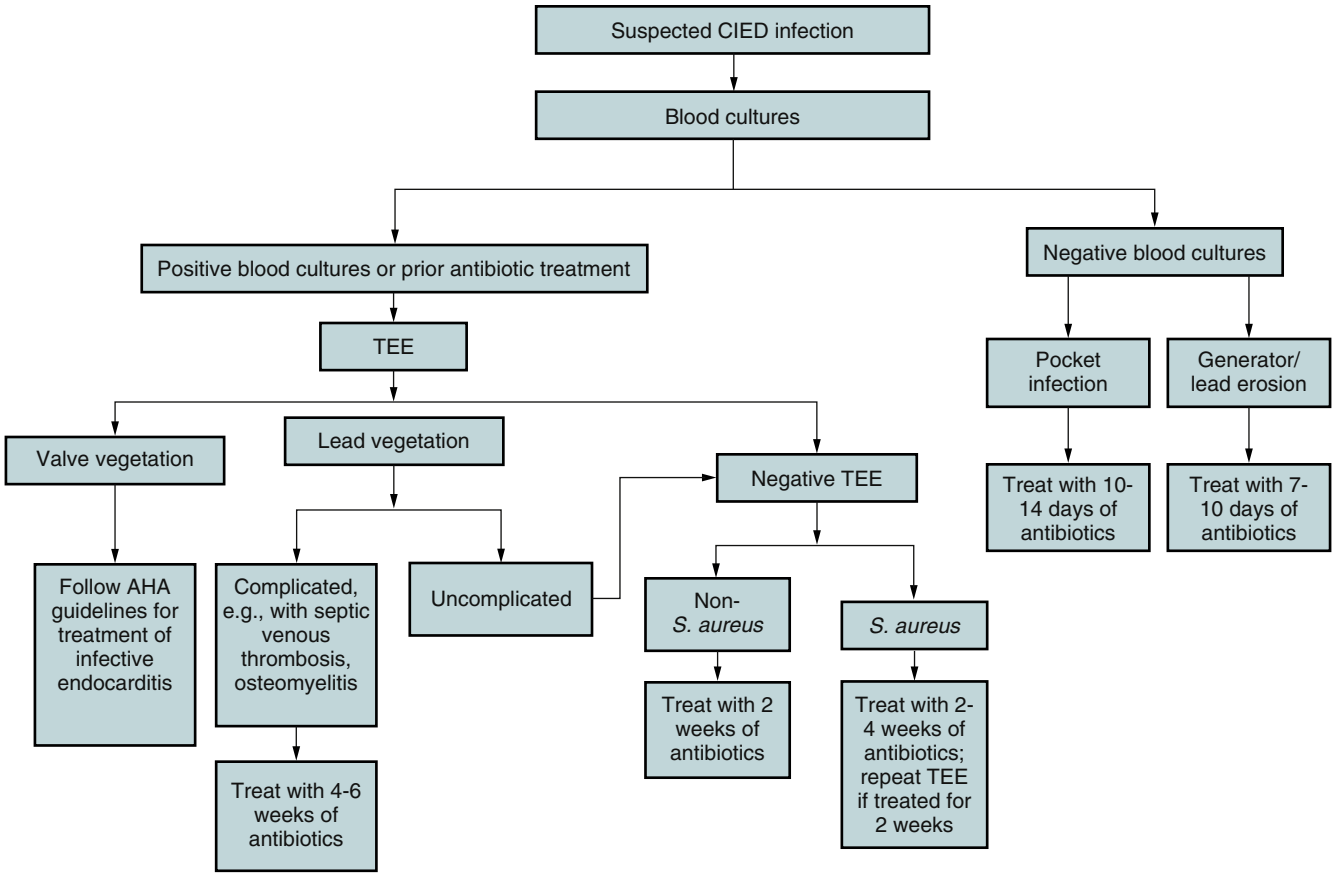
Some operators obtain an ultrasound to assess for fluid around the pocket. This, however, may be a nonspecific finding, especially if there has been recent pocket manipulation such as a new implant or generator change. A needle should definitely not be inserted into the pocket for aspiration because it has low yield, could introduce skin flora into a noninfected pocket, or cause damage to a lead, which may lead to an inappropriate shock or inhibition of pacing.[36]

Fluorine-18 fluorodeoxyglucose positron emission tomography (^{18}F-FDG PET) combined with computed tomography has been used to determine if there is a CIED infection if the diagnosis is in doubt.[84–87] Sarrazin et al.[86] demonstrated that those with a negative 18F-FDG PET with initial suspicion of infection could be treated with antibiotics alone without later development of CIED infection. Overall, the sensitivity was 0.89 and specificity was 0.86 when using a qualitative visual score. When the ratio between the maximum count rate of the device and the mean count rate of normal lung tissue was used, a value greater than 1.87 led to 100% specificity. A second study using a ratio of greater than 2.0 had a 97% sensitivity and 98% specificity for pocket infection.[84] Furthermore, this modality is expensive, and it can be difficult to obtain insurance approval of the test for this indication in the United States.

Management

The optimum treatment of CIED infections depends on the location of infection, the causal organism(s), and the overall goals of patient care.

The AHA has published a scientific statement with information on how to manage CIED infection (Fig. 14.5).[36] Patients who present with an erosion and exposure of the device should undergo device removal, as described later. However, if there are signs and symptoms of soft tissue infection and it is unclear



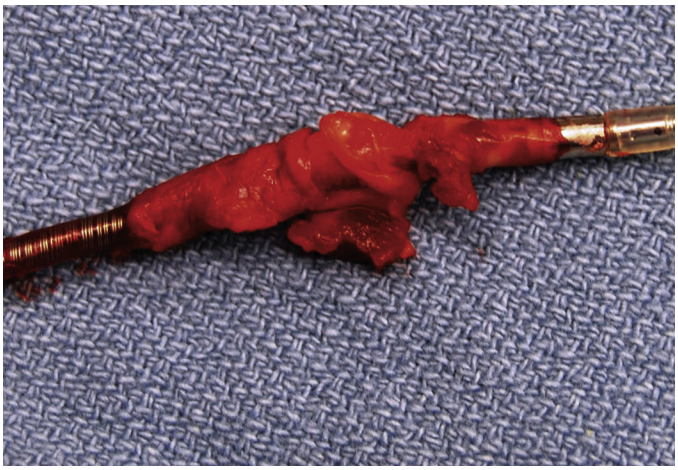
• **Fig. 14.5** Evaluation and management algorithm for suspected cardiac implantable electronic device (CIED) infection. Approach to management of adults with CIED infection. A history, physical examination, chest radiograph, electrocardiogram, and device interrogation are standard baseline procedures before CIED removal. Duration of antibiotics should be counted from the day of device explantation. (Modified from Sohail MR, Uslan DZ, Khan AH, et al. Management and outcome of permanent pacemaker and implantable cardioverter-defibrillator infections. *J Am Coll Cardiol.* 2007;49:1851-1859.)

if the CIED is infected, it may be reasonable to not remove the device up front and give a short course of antibiotics, assuming blood cultures are negative. If patients fail to improve, or if signs and symptoms return following cessation of antibiotics, then the device pocket itself is likely involved rather than just the overlying skin, and explantation is necessary.

For patients with positive blood cultures, erosions with device exposure, or failure to respond to appropriate antibiotics, device removal is indicated, combined with antibiotics, as described later.

CIED and Lead Extraction

All patients with an expected survival of greater than 1 year should undergo removal of the entire CIED system (Fig. 14.6). Removal of the CIED generator alone is not sufficient for treating the infection, because the leads will also have the infectious agent on them. Recurrence is common if the entire system is not removed.[4,88,89] To demonstrate the microbiologic extension of pocket infections, investigators in one study removed the pacemaker generator and extravascular portion of the leads from the pectoral region, but removed the intravascular portion of the leads using a snare from the femoral vein.[90] To try to prevent cross-contamination, one operator removed the extravascular portion and a second operator removed the intravascular segment. While 95% of extravascular lead cultures were positive, 79% of intravascular lead cultures were positive,



• **Fig. 14.6** Lead with scar tissue and vegetation that was removed by percutaneous extraction.

a difference that was not statistically significant. This demonstrates the high rate of organism growth on the intravascular portion of leads in those with pocket infection and the need for complete CIED system removal.

Explantation is defined as removal of a system that has been in place for less than 1 year and is removed from the pocket. In contrast, extraction is defined as the removal of leads that have been implanted for over 1 year, leads that must be removed using specialized tools such as an excimer laser, or leads that are removed from a location different from implantation, such as using a femoral snare.

For systems that have been implanted for less than 1 year, it is usually relatively simple to remove the system by manual traction. Leads that have been in place for a longer period can develop fibrosis and attachment to the vascular wall or heart, and manual traction will usually not be successful in removing the leads without a tear. Lead extraction must be performed at centers with experience, including by physicians who have been adequately trained, backup cardiothoracic surgeons available to perform an emergent sternotomy, an anesthesiologist to manage the patient's airway and hemodynamics, and perfusionists to manage a cardiopulmonary bypass machine. Transesophageal echocardiography and fluoroscopy should also be used.

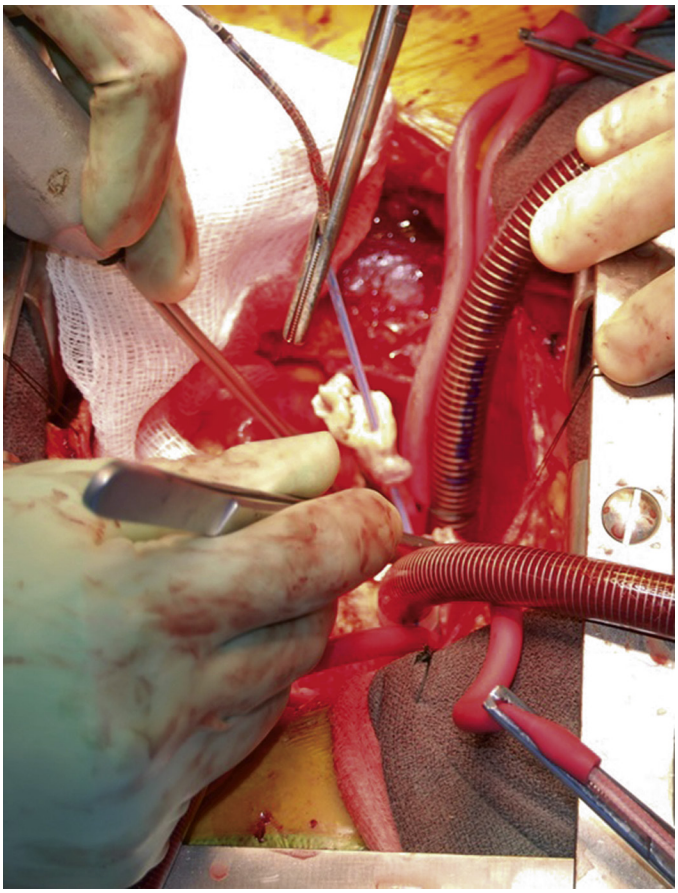
Complete clinical success for lead extraction is between 96% and 100%.[91,92] There is a 0.3% to 0.8% chance of death and a 1.0% to 4.3% chance of major complications, including cardiac or vascular incidents requiring thoracotomy, sternotomy, pericardiocentesis, or chest tube.[91,92] Methods for lead extraction are beyond the scope of this chapter, but several excellent reviews are available.[93–95]

Open Surgical Approach

Before the development of percutaneous lead extraction, an open surgical approach was needed to remove infected CIED systems. Percutaneous removal may not be possible for all patients; some may be too high risk, have failed percutaneous removal, have very large vegetations (Fig. 14.7), or have concomitant valvular endocarditis. These patients require an open surgical approach. In addition, there may be significant tricuspid valve damage requiring repair or replacement.

There is no definitive lead vegetation size in which an open approach is recommended, although many recommend it be greater than 3 cm.[93] It seems safe to do lead extraction in sizes less than 2 cm, and those up to 4 cm have been extracted percutaneously.[76] There may be a risk of paradoxical embolus in those with a patent foramen ovale, atrial septal defect, or ventricular septal defect, and some have advocated for an open approach in these patients.[93]

In one multicenter study, 8.9% of patients with CIED infection underwent an open surgical approach, one-third of which were a result of failed percutaneous extraction.[96] Of the group who went for a planned initial surgical approach, they were more likely to have systemic emboli, a prosthetic valve, lack of coronary artery disease, and a lower Charlson comorbidity index.[96]



• **Fig. 14.7** Intraoperative image of a large vegetation on a lead removed by open sternotomy.

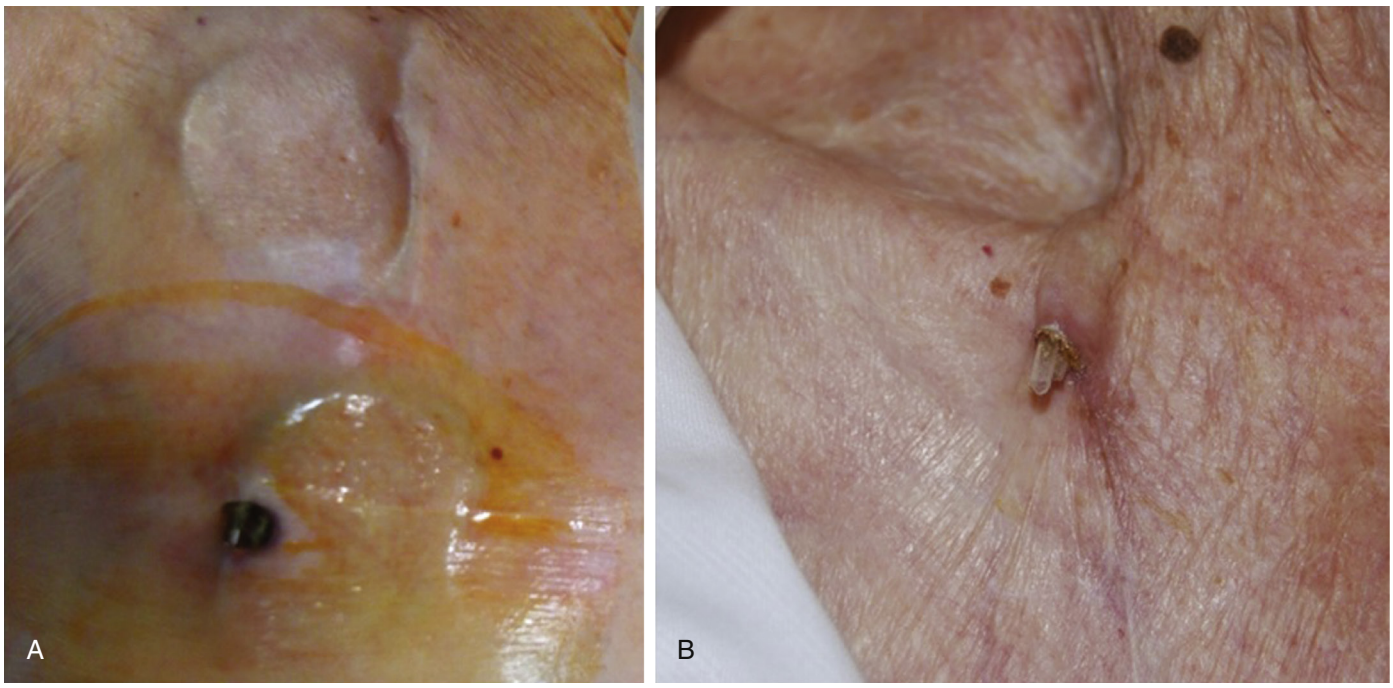
The surgical technique may be through a thoracotomy or sternotomy with cardiopulmonary bypass, depending on the clinical situation. The distal portions of the leads are typically removed through a right atriotomy while the proximal portion is pulled from the pocket. Depending on the clinical scenario, a permanent epicardial pacing system may be placed at the same time[97,98] because it is out of the bloodstream and less likely to be reinfected.

One study showed no difference in survival with open surgery versus percutaneous removal,[99] one showed an improved survival,[96] and one showed worse survival.[6] However, given differences in patient comorbidities and low numbers of cases, these results are difficult to interpret and the choice should be individualized for each patient.

Hospital stay is frequently shorter for patients undergoing percutaneous lead removal, which is one reason to pursue this approach first.[99] If the operators or institutions do not have significant experience with percutaneous removal, however, it may be reasonable to perform an open surgical approach as the primary treatment because it may have a more favorable risk-benefit profile.[93]

Long-Term Antibiotics

For patients who are believed to have high mortality risk from other comorbidities and are unlikely to survive more than



• **Fig. 14.8** Ninety-six-year-old woman with dementia and a pocket infection believed to be too high risk for extraction. (A) Initial erosion of right-sided pacemaker. Chlorhexidine is seen on the skin. The pacemaker was removed and the proximal portion of the lead cut, but an extravascular segment remained. A new pacemaker was placed on the left side. (B) Six months later, the retained lead fragment eroded through the skin.

1 year, it is reasonable to consider long-term suppressive antibiotics, as described later. Approximately 3% to 15% of patients are believed to be too high risk or refuse device system removal.[12] For patients with pocket infection, it may be reasonable to remove the generator and pocket, then cut the lead and allow it to retract into the vasculature. If the generator alone is removed without removing the lead or allowing it to retract, future erosion of the lead is possible (Fig. 14.8). For patients who still need a pacing system, it may be placed on the contralateral side.

Postoperative Management

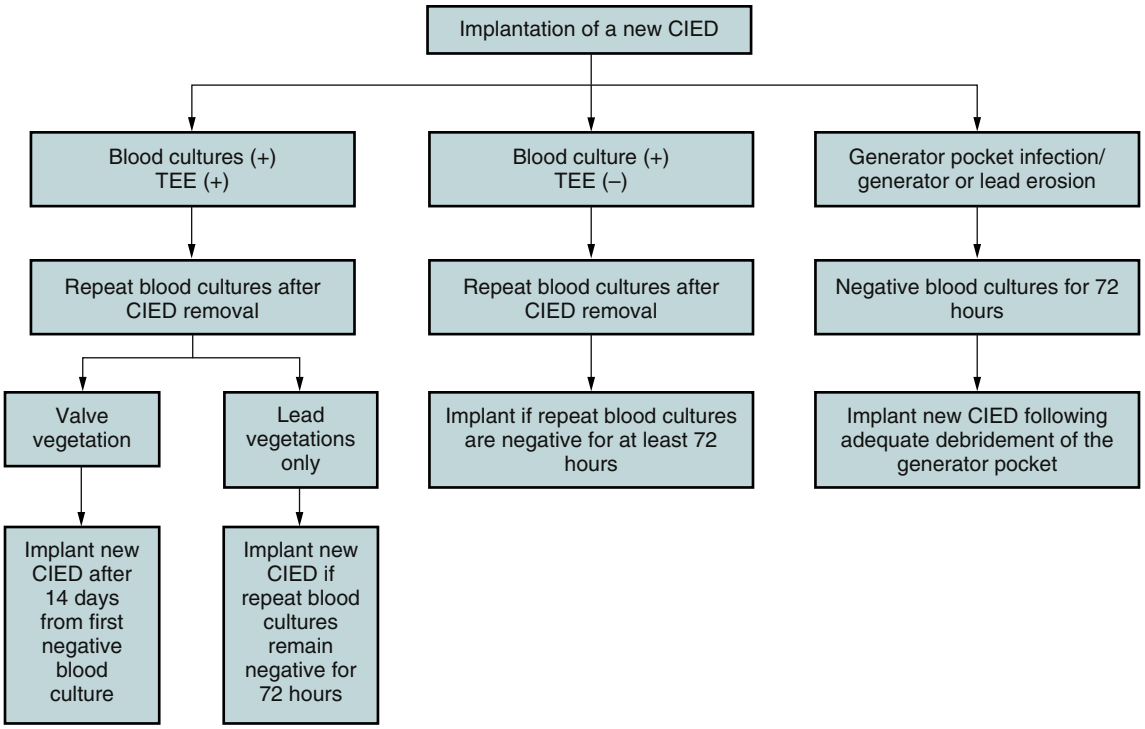
Antibiotics

Antibiotics are an essential part of postoperative management for these patients, and in most cases there are benefits to collaborating with a specialist in infectious diseases to choose the best drug, at the optimum dosage and duration. This specialist should also formulate a follow-up plan and in most cases provide direct follow-up antibiotic management, including toxicity monitoring and prevention of drug interactions.

After device removal, antibiotics should be used and tailored to the organism and resistance pattern. If the organism is unknown initially and cultures are not available yet, empiric therapy with an antistaphylococcal agent is appropriate. Based on the local resistance patterns, there should be strong consideration for using vancomycin as the initial agent, potentially in combination with an anti–gram-negative agent such as ceftriaxone, which also provides superior killing power against MSSA compared with vancomycin. In some circumstances, an oral fluoroquinolone may be chosen instead, especially if there are difficulties with intravenous access. Regardless of the regimen chosen, blood cultures should be obtained after device removal in case there is hematogenous spread by the removal surgery.

Patients with a pocket infection are treated for shorter periods compared with those with bacteremia and endocarditis. According to the AHA scientific statement (see Fig. 14.5), patients with uncomplicated CIED erosion without pocket inflammation can be treated for 7 to 10 days after explantation, and those with pocket inflammation should be treated for 10 to 14 days.[36] Our practice is to treat all patients with a pocket infection and negative blood cultures for 14 days.

For those with bacteremia alone and no valvular lesions on echocardiography, at least 2 weeks of intravenous antibiotics are recommended; longer durations may be used for more virulent organisms (e.g., *S. aureus*, *Pseudomonas*, *Candida*, mycobacteria) if remnant fragments are retained or if there are patient factors such as immunosuppression or other prosthetic materials. In fact, retained lead fragments may trigger discussion of much longer courses of antibiotic suppression. Typically, these patients are treated as though they have prosthetic valve endocarditis, with a long course of intravenous antibiotics in combination with oral rifampin when feasible, because of this drug's unmatched tissue penetration—including biofilms. Regardless of the regimen chosen, a small subset of these patients can develop recurrent bacteremia following discontinuation of antibiotics. At this point, there must be strong consideration of an indefinitely long course of oral medications when safe and feasible (often β-lactams, trimethoprim/sulfamethoxazole, or doxycycline) or removing the lead fragment percutaneously or



• **Fig. 14.9** Suggested management strategy for reimplantation of new cardiac implantable electronic device (CIED) following treatment for infection. (From Sohail MR, Uslan DZ, Khan AH, et al. Management and outcome of permanent pacemaker and implantable cardioverter-defibrillator infections. *J Am Coll Cardiol.* 2007;49:1851-1859.)

surgically. This is the exception to the rule, though, and most patients will not develop recurrent bacteremia.

If there is a lead vegetation with no evidence of a valvular vegetation and a non–*S. aureus* organism, 2 weeks of antimicrobial therapy may be used. If there is a valvular vegetation, *S. aureus*, or evidence of distant infection elsewhere such as septic venous thrombosis or osteomyelitis, a longer duration of 4 to 6 weeks should be used. However, decision making should be individualized based on the organism and patient factors.

Reimplantation

There is little data to guide when to reimplant CIEDs after extraction (Fig. 14.9); however, the first step is determining if the patient needs reimplantation. For instance, a patient may have had a primary prevention ICD implanted, never received an ICD shock, and had an improvement in ejection fraction. This patient does not need a new ICD. Similarly, some patients may have had an indication for pacing that has since resolved. Up to half of patients may not need reimplantation of a device.[100–102]

For patients who are pacemaker dependent, a "temporary permanent pacemaker" or "externalized permanent pacemaker" can be used as a bridge to reimplantation.[103]

Prognosis After CIED Infection

Several studies have demonstrated an increased mortality rate in those with a CIED who develop an infection of the device that persists for months or years after hospital discharge.[16,72,74,104,105] There is a significant range of all-cause mortality in studies based on the duration of follow-up. In-hospital or 30-day mortality rate is 2% to 15%, 6-month mortality rate is 4% to 29%, and 2-year or longer mortality rate is 6% to 35%.[12] Whether this risk is due to the infection or, more likely, comorbidities that increase the risk of CIED infection has not been determined.

The main risk factor for increased mortality rate with a CIED infection is failure to remove the CIED.[71,106,107] In addition, there is the suggestion that early CIED removal within 3 days of hospitalization can lead to a decreased mortality rate.[108] There are biases to these studies, however, because patients may be believed to be too high risk to undergo device removal either early in the hospitalization or at any time point, and it is the healthier patients who are getting the CIED removed.

Several studies have attempted to predict the risk of death with a CIED infection. Having bacteremia or lead endocarditis increases the risk.[106,108] Having an elevated Charlson comorbidity index, having an older device, using corticosteroids, and presenting with hypotension also increase the risk.[106] Another study suggested that higher creatinine, lower ejection fraction, and hypotension were risk factors.[108] Risk factors for dying over a longer-term follow-up include systemic embolization, moderate or severe tricuspid regurgitation, abnormal right ventricular function, and creatinine greater than 1.5 mg/dL.[109] In this series, the mortality rate after a CIED infection was 18% at 6 months, suggesting an elevated risk of death not just from infection but also from the underlying comorbidities.

Conclusion

CIED infections remain stubbornly common, with an incidence of roughly 1% to 2% per device, though it is substantially higher among patients who sustain a complication during implantation. Meticulous adherence to best practices will reduce the risk but cannot fully eliminate it. Although most infections present within 3 months of implantation, delayed presentations frequently happen, and there is no safe time when an infection will no longer occur. Virtually any organism can cause CIED infection, but the most common are skin commensals such as coagulase-negative staphylococci and *S. aureus*. A systematic approach to making the diagnosis is essential, so that all stakeholders—electrophysiologist, infectious diseases specialist, cardiac surgeon, patient, and family—can make the best management decisions.

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15

Postoperative Management

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Introduction

Comprehensive postoperative management of the patient following cardiac implantable electronic device (CIED) procedures presents unique challenges but is critical to ensure optimal outcomes. Although the early postoperative time usually requires nothing more than careful and directed observation, several specific issues must be addressed. Effective patient care requires more than the skill of the implanting physician; it requires a collaborative team of knowledgeable health care providers who are aware of expected outcomes and risks and who can manage problems that may occur following the device and/or lead implantation. These include the stability of the patient after the CIED system implantation, pain control, recognition of early and longer-term complications, management of underlying conditions, proper patient and family education, vigilance of appropriate early and late postprocedure device programming, wound management, and follow-up arrangements.

Postoperative Management Specific to CIED Type and Indications

The postoperative team must understand the type of CIED implanted and the indication for implantation. A patient with complete heart block who is otherwise healthy and has normal ventricular function quickly becomes dependent upon the device; thus, lead failure is a potentially catastrophic event. Conversely, for the patient with intermittent bradycardia, although surgical and lead-related complications remain important, associated comorbidities play a much larger role in outcomes following device implantation. Moreover, patients with diabetes and high serum glucose levels may have more difficulty with wound healing and be at greater risk for infection.

For patients with an implantable defibrillator, it is important to know whether a single- or dual-chamber device was implanted and if the patient requires pacing in addition to protection from tachyarrhythmias. Cardiac resynchronization devices are most often combined with an implantable cardioverter-defibrillator (ICD); these patients are then at risk for multiple lead issues, as well as both appropriate and inappropriate shocks. Maximizing left ventricular pacing is important before discharging the patient. Loss of capture or intolerable phrenic nerve stimulation may ultimately require early changes in programming or lead repositioning.

Lead dislodgements before discharge were reported in 1% of the patients entered into the National Cardiovascular Data Registry.[1] The specific rhythm disturbances that can result from lead dislodgements for all types of leads should be understood. For example, if a new atrial lead dislodges into the ventricle, ventricular sensing (paced or intrinsic) could be "interpreted" by the device as atrial sensed activity, resulting in loss of atrioventricular synchrony. In addition to lead dislodgement, patients with new endocardial transvenous leads are at risk for cardiac perforation and pneumothorax, situations that need to be rapidly assessed and acted upon. For some patients, lead extraction is part of the procedure that engenders unique and complex risks that require specific observation in the postoperative time interval.

In contrast to procedures involving new lead implantation, a planned generator change is unlikely to be associated with lead dislodgment. While generally straightforward, problems such as disproportionate pocket pain if a pocket revision was performed or excessive bleeding in the anticoagulated patient may be encountered. A discovery of an unexpected lead fracture at the time of the procedure could convert a generator replacement into a lead replacement procedure. Lead malfunction can occur after generator replacement owing to tenuous lead integrity that is worsened from manipulation of the lead and/or removal of scar during the procedure. This malfunction may not present until months later.[2]

"Header block" issues are another source of complications in the early postoperative time. These problems stem from inappropriate connection of the lead(s) within the header block ports. When not correctly connected, oversensing of resultant noise could cause loss of pacing and/or inappropriate ICD shocks. Additionally, atrial and ventricular pacing leads can be inadvertently inserted into the wrong port, resulting in pacing malfunction. For defibrillators, the risk is now less with DF4 leads, where the prior bifurcated leads are now a single four-pole inline connector.

Specific patient clinical factors are evaluated in reference to their baseline preoperative clinical status. For example, if a patient has chronic hypotension, low blood pressures postoperatively may have little meaning; however, a patient with chronic hypertension who develops hypotension in the early

postoperative period should be carefully evaluated for bleeding or cardiac tamponade. Similarly, an oxygen-dependent patient with severe chronic obstructive pulmonary disease (COPD) may be expected to have a low oxygen saturation, but the same situation would not be expected for an individual without prior respiratory compromise; thus, hypoxia may indicate a pneumothorax or worsening heart failure. Patients requiring anticoagulation, particularly with a low-molecular-weight heparin or standard heparin, are at higher risk of hematoma formation and pocket infections. It has become common practice to perform device implantations while patients maintain their chronic anticoagulation. Experience with warfarin has suggested this practice to be safe, although there is less experience with the non–vitamin K target-specific anticoagulant drugs.[3–5]

All members of the team should know how the pacemaker or implantable defibrillator is programmed. A patient who has a VVI pacemaker will only pace at times that are required based on a programmed fixed lower rate (unless there is hysteresis). For example, if the lower rate is programmed at 60 beats/min and the patient is found to have no evidence of pacing and an intrinsic conducted rhythm at 30 to 40 beats/min, it can be assumed that the device is not functioning as programmed. This situation should be urgently evaluated. For patients implanted with a defibrillator, the lowest tachycardia detection zone for which therapy (ICD shocks or antitachycardia pacing) is activated needs to be known and documented.

The complexity of CIED types and the patients receiving them necessitates team management with a communication plan. Responsibilities should be clearly defined to prevent critical abnormalities (including medication reconciliation) from being overlooked. The team leader, generally the implanting physician, and any allied professional providers and training fellows should meet regularly with the postoperative team to review the responsibilities of all team members and anticipated care requirements for individual patients.

Immediate Postoperative Care

The initial evaluation begins with a measurement of vital signs, oxygen saturation, and cardiac rhythm and a determination of symptoms including pain. For patients receiving conscious sedation or general anesthesia, the level of consciousness must be assessed, quantitated, and documented. An early determination of the need for continuous observation if the patient has any unstable vital signs is important. Also, many patients should remain in a supine position for a period of time until the effects of anesthesia have resolved and there is not a risk for adverse events should the patient try to independently stand. Additionally, patients may demonstrate mild confusion related to either the sedation or the stress of the procedure.

Unintended arm movements may place a patient at risk of lead dislodgement, and for some individuals, arm or body restraints may be needed. For those individuals who are anticoagulated or for whom hemostasis is challenging, a compressive dressing may be of some value; however, it may also mask the problem and delay recognition of an expanding hematoma if not checked frequently.

The patient's hemodynamic and respiratory status should be monitored during transfer from the electrophysiology laboratory or operating room to the postprocedure recovery unit. Similar to other surgical procedures, this is a critical time because of transfer of care to personnel less familiar with issues that may have transpired during the implant procedure. It is important for the implant team to provide a cogent and comprehensive summary of any concerns and expectations to the postoperative team caring for the patient.

Most institutional protocols include vital signs recording upon arrival to the postoperative unit, generally every hour for 2 hours, and then every 4 hours thereafter along with continuous telemetry monitoring. For patients receiving deep sedation or general anesthesia, the anesthesiology team will provide immediate postoperative monitoring in accordance with their protocols and practice and the patient's clinical status (see Chapter 7). Parameters that define the circumstances for notifying the implanting physician of the patient's clinical status are also generally dictated by established institutional protocols.

Situations that should trigger notification of the implanting physician or designee include hemodynamic deterioration, respiratory distress, expanding hematoma, bleeding, fever, important arrhythmia, or suspected failure of sensing or capture by the CIED. In addition to these, other acute complications include disproportionate pain, lead dislodgement, lead perforation with or without cardiac tamponade, device malfunction, and pneumothorax (Table 15.1). An immediate postprocedural anterior-posterior chest x-ray (CXR) is obtained to rule out the presence of a pneumothorax and to evaluate lead location and adequate redundancy, specifically in patients with new lead implants.

In the immediate postoperative period, adequate analgesia is given as needed and/or an ice pack can be placed gently over the CIED surgical site for comfort. Health care providers should understand the difference between typical surgical pain and a more serious problem such as pleural pain from an expanding pneumothorax or pericarditis from lead perforation.

The goals for respiratory management include oxygen therapy as needed to maintain an oxygen saturation (Spo_2) greater than or equal to 92%. For patients with sleep apnea, an overnight stay is recommended and continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) should be used. Patients with obstructive sleep apnea are encouraged to bring their unit from home.

Postoperative Recovery Unit to Hospital Discharge

While most patients receiving a CIED will recover without incident, this is a period in which important complications, including those during the immediate postoperative recovery phase discussed earlier, may occur. A change in lead function or frank dislodgement is frequently not observed until after the first 12 hours following implantation. Thus, a pacemaker-dependent patient with a new implant is generally not

TABLE 15.1 Selected Complications by Type and Time of Occurrence

Periprocedural (Intraprocedure to First 24 hr)	Early Postprocedural (After First 24 hr to 1 Week Postimplant)	Late Postprocedural (After 1 Week to 1 Year Postimplant)
Pneumothorax	Lead dislodgement	Device malfunction
Hemothorax	Diaphragm stimulation	Lead dislodgement
Lead perforation with pericarditis, pericardial effusion, or tamponade	Delayed perforation of leads	Delayed perforation of leads
RBBB or CHB from lead manipulation	Symptomatic subclavian vein thrombosis	Symptomatic subclavian vein thrombosis
Coronary sinus dissection or perforation	AV fistula	Lead failure and need for intervention
IV contrast or other drug reaction	Pocket pain	Device migration
Hemomediastinum	Hematoma or bleeding	Pocket pain
Air embolus	Superficial cellulitis, wound dehiscence, pocket infection, bloodstream infection, device endocarditis, or sepsis	Tricuspid valve damage
Arterial stick or lead placement	Thoracic duct injury with chylothorax or lymphatic fistula	Frozen shoulder
Lead crossing PFO or ASC or placed in CS	Renal dysfunction from IV contrast	Erosion, pocket infection, bloodstream infection, device endocarditis, or sepsis
Damage to leads during generator change or lead addition	Pulmonary embolus	Twiddler's syndrome
Death		SVC syndrome
Atrial or ventricular arrhythmias		AV fistula
Myocardial infarction		Keloid or hypertrophic scar
Stroke		
Pulmonary edema		
Cardiogenic shock		
Nerve injury		
Pulmonary embolus		
Local anesthesia overdose or injection into vessel		
Apnea		
Loose set screw		
Retained products		
Placing lead in wrong header port		
Lost guidewires in vessels		

ASC, atrial septal defect; AV, arteriovenous; CHB, complete heart block; CS, coronary sinus; IV, intravenous; PFO, patent foramen ovale; RBBB, right bundle branch block; SVC, superior vena cava.

discharged the same day. Loss of capture can be a life-threatening situation for a pacemaker-dependent patient, necessitating the immediate availability of resuscitative equipment with temporary transthoracic pacing and a CIED-trained response team member (physician, cardiology fellow, or other health care provider) who could interrogate the device and determine if an increase in the pacing output would be useful or if alternate emergency measures must be taken.

The nursing team needs to be aware that the patient could become acutely symptomatic from a complication at any time within the observational period and that symptoms may evolve. The patient should always be assessed by the implanting physician or designee if a significant change in clinical status occurs. There should be a low threshold for contacting the physician for a change in oxygen saturation, modest and persistent changes in vital signs or rhythm, or complaints of chest pain or dyspnea.

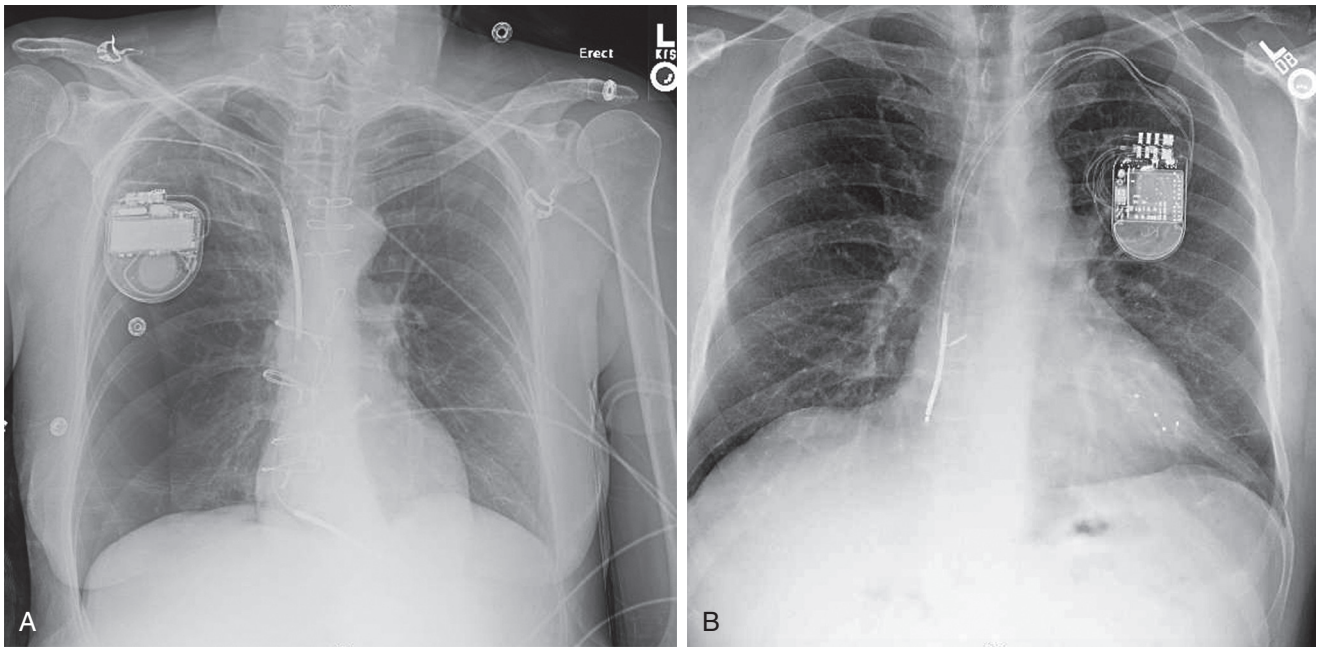
Health care providers less familiar with the potential complications of these procedures may erroneously ascribe symptoms to surgical site pain or the discomfort of having been immobilized for the procedure, potentially missing the signs and symptoms of a complication.

Other major medical events, albeit rarely occurring early after implantation, include myocardial infarction, pulmonary embolus, and stroke. Worsening heart failure due to fluid shifts and/or procedural complications represents a more common possibility. Rarely wound dehiscence and acute infection can occur early, though most infections occur later after implantation, even out to 1 year (see Chapter 14).

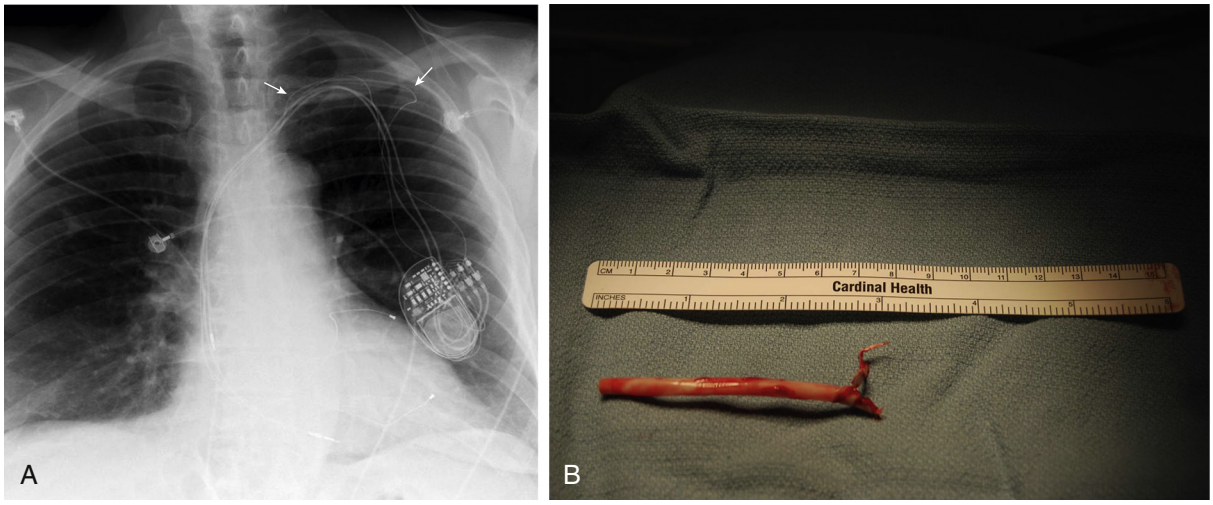
Specific Testing

Chest Radiograph

Because a pneumothorax or a lead dislodgement may not manifest immediately, implanting physicians often order an early postprocedural portable anterior-posterior (AP) CXR and a posteroanterior (PA) and lateral CXR the next day before discharge (Fig. 15.1A–B). Some physicians routinely discharge patients on the same day as the implant procedure and will perform a PA and lateral CXR before discharge. Other abnormalities that could be identified on the PA and lateral CXR include a foreign body (e.g., sponge, instrument), an anchoring sleeve introduced into the venous system when manipulating a lead without a hemostatic sheath in place, or an avulsed section of the hemostatic sheath (Fig. 15.2).



• **Fig. 15.1** Postoperative chest radiograph showing a large right-sided pneumothorax obtained during vascular access from the right subclavian vein (A) and dislodgement of a right ventricular defibrillator lead (B).



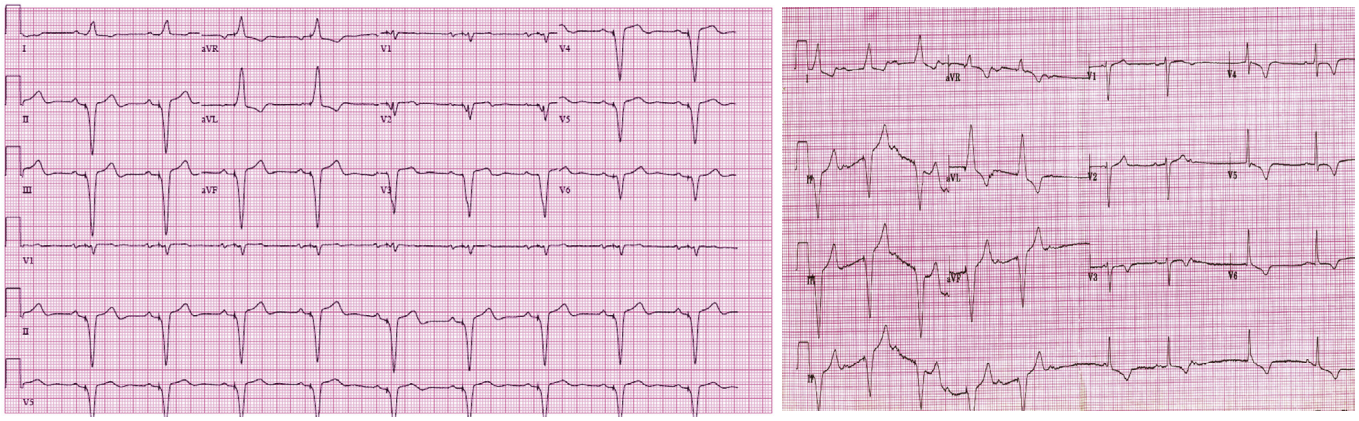
• **Fig. 15.2** Chest radiograph with (A) retained splittable sheath *(arrows)* and (B) retained splittable sheath extracted.

Electrocardiogram

An electrocardiogram (ECG) should be performed within the first 24 hours to document that the CIED device is functioning as intended. The morphology of the paced QRS complex should remain stable after implant, and if there is right ventricular–only pacing (not cardiac resynchronization therapy [CRT]), a left bundle branch block should be present (Fig. 15.3A–B). If a right bundle branch morphology is present in a patient who received a transvenous lead intended for the right ventricle, then consideration of errant placement of the lead into the left ventricle must be considered (through an atrial septal defect [ASD] or ventricular septal defect [VSD]) or that the lead has been placed into a branch of the coronary sinus. Rarely this could occur owing to a lead that perforates across the ventricular septum and into the left ventricle. Inadvertent placement of a lead across the aortic valve and into the left ventricle has been reported.[6,7]

Routine Laboratory Testing

Generally patients do not require routine blood tests such as a blood cell count or metabolic panel unless they require anticoagulation monitoring, have an expanding hematoma, or are actively bleeding, or if arrhythmias suggestive of an electrolyte, calcium, or magnesium abnormality are suspected.



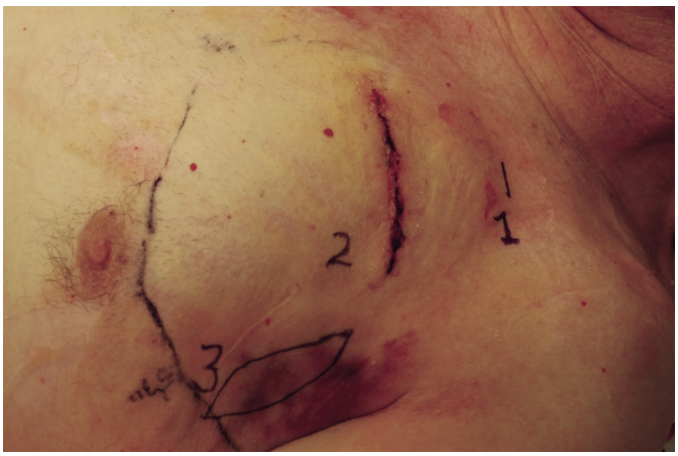
• **Fig. 15.3** Electrocardiograms (ECGs) showing right ventricular pacing with appropriate ventricular capture and left bundle branch morphology (A) and ECG from different patient demonstrating loss of right ventricular capture during pacing (B) (single-chamber pacemaker programmed VVIR at a rate of 60 beats/min).

Device Interrogation

Interrogation of the CIED is mandatory for all new device/lead implants before discharge to confirm stability of the system and adjust programmed settings as needed. A significant change in sensing or capture threshold may be due to lead dislodgement or perforation, requiring both patient and device reassessment. As there is a risk of acute elevation in pacing thresholds owing to edema (although rare now with steroid-tipped leads), pacing outputs are set higher than they would be for long-term pacing. Outputs are often programmed downward within 90 days after the implant procedure.

Pertinent findings during device interrogation include a significant decrease in the sensed R or P waves, changes in the pacing thresholds or lead impedance, and the occurrence of phrenic nerve or diaphragmatic pacing. Left ventricular (LV) leads placed for CRT devices are the most common cause of phrenic nerve stimulation. This can often be ameliorated with adjustment of pacing output or pacing vectors if available. In the case of LV leads, once the pacing threshold has stabilized during outpatient checks, the initial postoperative high pacing outputs may be minimized to near-capture threshold to extend battery longevity.

If diaphragmatic pacing occurs with right atrial or right ventricular leads, perforation should be considered. Atrial leads placed in a lateral position may also cause diaphragmatic stimulation owing to direct phrenic nerve stimulation without perforation (causing hiccups). Right ventricular leads may stimulate the diaphragm directly if seated deeply into the right ventricular (RV) apex without actual perforation being present. If evaluation with imaging studies and assessment of electrical parameters do not suggest perforation, the pacing output should be adjusted if possible to abolish diaphragmatic pacing but provide an adequate threshold for pacing stimulation. Strong consideration should be given to lead revision in the pacemaker-dependent patient.

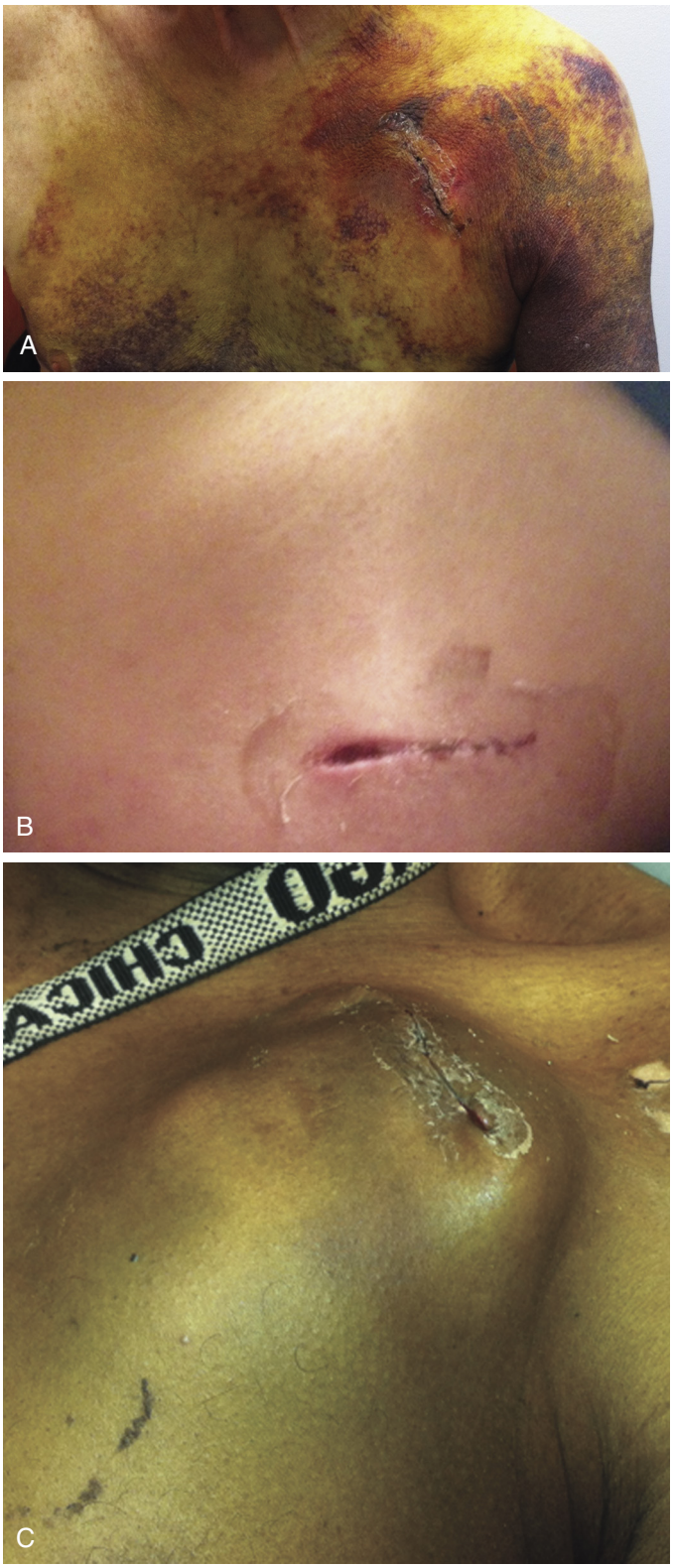


• **Fig. 15.4** Multiple postoperative findings. Marking pen used to identify areas of concern for nursing observation. *1* illustrates skin excoriation from dressing removal, *2* illustrates disruption of skin adhesive following removal of dressing, and *3* illustrates extent of pocket hematoma.

Wound Care

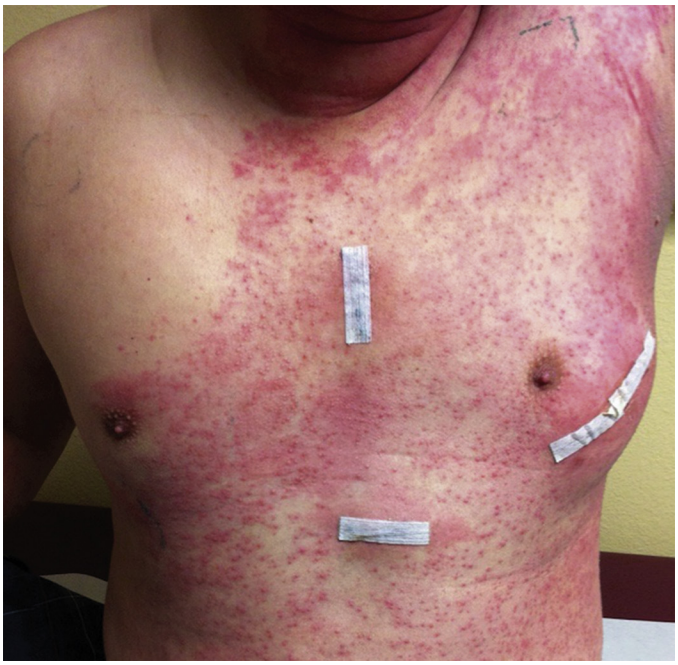
Following closure of the surgical incision, the wound may be dressed with skin adhesive (Dermabond or similar), skin closure strips (Steri-Strips), a nonwoven wound dressing (Primapore), or a combination of these. Regardless of whether skin adhesive or closure strips are used, the nonwoven dressing is left in place for 24 to 48 hours. During that period, the dressing should be inspected for active bleeding, and the generator pocket beneath the dressing inspected for evidence of an expanding hematoma. When the nonwoven dressing is removed, care should be taken to prevent disruption of the skin adhesive or closure strips below the dressing (Fig. 15.4). This may be problematic if the nonwoven dressing is applied before the skin adhesive had sufficient time to dry.

Once the nonwoven dressing has been removed, the perincisional site should be monitored for wound dehiscence, ecchymosis, bleeding, and generator pocket hematoma (Fig. 15.5A–C). An expanding hematoma in the early perioperative



• **Fig. 15.5** Examples of ecchymosis (A), wound dehiscence (B), and generator pocket hematoma (C).

period may delay discharge until the bleeding has abated. If a hematoma occurs, it is preferable to keep the incision intact and not reexplore as this may increase the risk of infection. If the hematoma continues to expand to the extent that pain is intolerable despite aggressive pain management, or if the incision dehisces because of pressure and begins draining, exploration and evacuation of the hematoma may become necessary.



• **Fig. 15.6** Allergic reaction to chlorhexidine surgical scrub. Note demarcation between prepped and unprepped areas.

In either case, extended intravenous antibiotic therapy is reasonable. A pressure dressing applied to the pocket hematoma may reduce further bleeding, and ice packs may help to reduce pain due to tension on surrounding tissue. Assessment of the patient's coagulation status including platelet count and an international normalized ratio (INR) level (depending on the type of anticoagulation) should be performed, and withholding anticoagulation therapy should be considered, if otherwise safe. In the early perioperative period, physical signs suggesting infection include erythema, induration, tenderness, and cellulitis. Allergic reactions to the prep solutions may also manifest; however, this may only present after discharge (Fig. 15.6). Any of the aforementioned findings warrant prompt intervention.

If the patient uses tobacco products, nicotine replacement therapy should be strongly encouraged, particularly in patients with peripheral vascular disease. Smoking can prolong the effects on inflammatory and reparative cell function, leading to delayed healing and complications.[8]

Activity

Early ambulation following CIED implantation is encouraged based on the patient's underlying condition once the effects of sedation have cleared.

Before leaving the electrophysiology laboratory or operating suite, a sling is typically placed on the upper extremity of the ipsilateral side of a new lead placement (note that some patients may have a new lead placed on the contralateral side

of the device and tunneled to the generator pocket). The sling is generally left in place for 24 hours or until discharge and the postoperative CXR has been examined. The purpose of the sling is to prevent excessive range of motion on the arm that could result in lead dislodgement or generator pocket bleeding and hematoma formation. Some physicians do not feel a sling prevents these outcomes and may choose not to use one. Once the patient goes home, he or she may want to wear the sling during sleep for additional comfort; however, prolonged use should be avoided as chronic immobilization of the shoulder may produce an adhesive capsulitis ("frozen shoulder"). For patients undergoing a generator change without new lead placement, a sling is not needed. Generally, the patient is instructed not to lift his or her arms higher than the shoulder for approximately 1 month or, at least, to avoid excessive upper extremity motion. Because restricted arm movements may be counterproductive and lead to adhesive capsulitis, physicians should decide whether to use a sling based on individual patient assessment.

Diet/Nutrition

Wound healing is dependent upon adequate nutrition because a postoperative anabolic state exists in the surgical patient. This is particularly important in the elderly population, which makes up a large portion of CIED implantations. Routine caloric requirements of 25 to 35 kcal/kg increase to 30 to 40 kcal/kg, while protein requirements increase from 0.8 to 1 g/kg to 1 to 1.8 g/kg. Patients with known hypoalbuminemia should have adequate protein replacement sufficient to maintain an anabolic state and match catabolism, but this must be carefully monitored in patients with renal and/or hepatic failure.

Diabetic patients may have decreased or impaired wound healing due to deficiencies in the cellular response to injury (activation of keratinocytes, fibroblasts, endothelial cells, macrophages, and platelets).[9] Careful control of serum glucose to avoid hyperglycemia has been shown to aid in wound healing.[10]

Vitamin supplementation following surgery remains controversial. However, vitamins A and C and zinc have been proposed as key nutrients for wound healing.[11] Vitamin A is considered to aid in cellular differentiation, proliferation, epithelialization, and collagen synthesis but should be avoided in renal failure owing to a significant increase in circulating blood levels as a result of the vitamin's ability to exceed the binding capacity of retinol-binding protein. Vitamin C plays an important role in collagen synthesis but should be avoided in patients with renal failure owing to the increased risk of renal oxalate stone formation. Zinc enhances protein synthesis, cellular replication, and collagen formation. Larger wounds can contribute to zinc losses. One multivitamin with minerals daily will compensate for any general micronutrient losses.

Venous Thromboembolism Prophylaxis

The Institute for Clinical Systems Improvement (ICSI) provides guidelines for patients 18 years of age or older hospitalized for venous thromboembolism.[12] Risk stratification with either the Caprini scoring method or American College of Chest Physicians guidelines may be used to determine risk.[13,14] Treatment options include early ambulation, pharmacologic anticoagulation, and mechanical prophylaxis (sequential compression devices).

Antibiotic Prophylaxis

Routine preoperative intravenous antimicrobial prophylaxis is warranted and even mandated immediately before implantation of pacemakers, implantable cardioverter-defibrillators, and cardiac resynchronization devices.[15] This is discussed in Chapter 14. A large randomized trial noted a significantly lower rate of skin and soft tissue infections among patients who received a single dose of cefazolin before device implantation.[16]

Risk factors for device-related infection include fever within 24 hours before implantation, temporary pacing before implantation, early intervention for hematoma or lead replacement, corticosteroid use for more than 1 month during the preceding year, presence of more than two leads, and development of pocket hematoma.[17,18] Typical choices for perioperative antibiotics include cefazolin 2 g given intravenously (IV) over 5 minutes and repeated intraoperatively at 3 hours, and vancomycin 1 g IV over 60 minutes and redosed at 6 hours if the procedure is ongoing. For patients with intolerance or allergies to these antibiotics, levofloxacin 500 mg IV every 24 hours for two doses is a reasonable alternative. Regardless of antibiotic choice, the intravenous infusion should be complete before skin incision. A follow-up postoperative dose of intravenous antibiotic at the appropriate time interval is often administered; however, there is no prospective controlled randomized data to support its use.

Pain Control

Postoperative pain is a confluence of somatic, sensory, and psychological responses to surgical injury. Low stimulation of unmyelinated nerve endings in the papillary dermis produces itching, while high stimulation results in pain. Scratching converts itching to a sensation of pain and eliminates pruritus. Application of cold also blunts somatic pain reflexes, making ice packs applied to the CIED implantation site a very useful and oftentimes overlooked simple therapy for pain control in the immediate and remote postoperative setting.

Treating postoperative pain can provide subjective relief and can blunt autonomic and somatic reflex responses so that patients remain functional until the wound otherwise heals. Complete resolution of pain by opioids or any other therapy is unlikely. However, psychological preparation of patients before CIED implant may help with tolerance of the pain. Preparation includes a careful description of the procedure, a description of sensations to be experienced, a plan to treat pain, and reassurance that sufficient analgesic treatment will be given. Common narcotic analgesics for immediate postoperative pain management include morphine, fentanyl, hydromorphone, oxycodone, and codeine–acetaminophen combinations. Nonsteroidal antiinflammatory agents may be effective. However, they should be avoided in patients taking anticoagulant

therapy, with cardiomyopathy, or with gastrointestinal sensitivity. A comparison of narcotic and nonnarcotic analgesic agents is listed in Table 15.2.

Discharge Planning

Overnight Observation

Elective CIED implantation patients admitted on the day of surgery are commonly discharged on the first postoperative day following a PA and lateral CXR, device interrogation, ECG, and physical examination. Before discharge, patients are given instructions for wound care. If Steri-Strips are in place, generally it is recommended that the wound should be kept clean and dry for at least a week. If Dermabond is used, the wound can get wet almost immediately as the glue forms an occlusive barrier. Some implanting physicians will ask the patient to refrain from showering for up to 48 hours following application of the skin adhesive, however (Table 15.3).

Same-Day Discharge

Efforts to save health care costs and free up inpatient beds have sparked interest in discharging patients with a new CIED implant on the same day the procedure was performed, as long as it is safe and does not interfere with patient outcomes. The choice for same-day discharge versus an overnight observation will vary depending on the patient's clinical condition and pacemaker dependency, the implant procedure type, and the suspected risk of complications. No specific evidence base supports one approach over another and the care at institutions may differ markedly from one to another.

Candidates for same-day discharge are chosen at the physician's discretion and typically exclude pacemaker-dependent patients with a new lead in the dependent chamber, those who require periprocedural bridging with heparin (best to avoid if at all possible), those who are unable to monitor for the presence of postoperative complications, patients who have intra- or postprocedural complications, those who have postprocedure device interrogations outside of expected parameters, and patients who live extended distances from a local emergency department. To facilitate expedient discharge of this patient group, postprocedural staff caring for the patient should be informed before the procedure. A sample same-day discharge protocol is provided in Table 15.4.

First Follow-up Clinic Visit

The first outpatient clinic visit following a CIED implant generally occurs within 7 to 14 days after discharge but can be scheduled up to 30 days. This visit should include a focused physical exam, wound check, device interrogation, review of precautions with device-magnet interactions, and activity restrictions. In addition, the function and role of the remote monitor given to the patient at the time of implantation should be reviewed, and the patient should be provided with an opportunity to voice any questions or concerns.

A focused history and physical exam should be performed to rule out symptoms suggesting infection. Inspection of the wound should focus on the presence of erythema, induration, fluctuance, cellulitis, or focal pain upon palpation, any of which may indicate early infection. The integrity of the surgical incision should be examined for structural integrity by ruling out the presence of tension or dehiscence. The generator pocket should be assessed for the presence of hematoma, which, if present, should be compared to the appearance of the

TABLE 15.2 Comparison of Common Narcotic and Nonnarcotic Analgesic Agents

Drug	Dose	Route	Frequency	Other
Morphine	2–4 mg	IV	q3h prn	Moderate to severe pain not responsive to oxycodone
Fentanyl	50–100 μg	IM	q1–2h prn	
Hydromorphone	2–10 mg	PO	q3–6h prn	
	2–4 mg	IM, SQ, IV	q4–6h prn	
Oxycodone	1–2 tabs	PO	q4h prn	Moderate to severe pain
Tylenol #3	1–2 tabs	PO	q4–6h prn	Not to exceed 4 g acetaminophen per day
Ketorolac	30–60 mg	30 mg IV, 60 mg IM	q6h prn	Not to exceed 120 mg/day
	10–20 mg	PO	q6h prn	Not to exceed 40 mg/day
Ibuprofen	400 mg	PO	q4–6h prn	
Naproxen	250–500 mg	PO	bid	
Tramadol	50–100 mg	PO	q4–6h	Max 400 mg/24 hr

generator pocket at the time of hospital discharge. As discussed previously, most hematomas are best treated with continued observation.

Other findings including pruritus and urticaria due to allergic reactions to surgical prep solutions or adhesive tape may be visible. If the patient's procedure was particularly difficult and long fluoroscopic times were required (such as with some CRT implantations) and in patients with a high body mass index (BMI), careful examination of the skin for radiation burns should be performed and documented (see Chapter 5).

A device interrogation is performed to determine the sensing and pacing characteristics as well as the impedance of each lead. These data are compared with the data at time of implantation. Device programming and the arrhythmia logbook should be reviewed for variances in programming or abnormalities requiring further assessment. The implanting physician should be aware of any deviances from previous device interrogations.

Following examination of the wound and interrogation of the device, patient education should be provided. Precautions regarding situations where electromagnetic interference may be encountered should be reviewed with the patient. These potential interactions are contained in the patient education materials supplied by the device manufacturer. While several manufacturers have U.S. Food and Drug Administration–approved magnetic resonance imaging (MRI) conditional CIEDs, institutional policies for performing an MRI on a patient with a conditional or non-MRI conditional CIED may vary considerably.

TABLE 15.3 Discharge Instructions After Cardiac Implantable Electronic Device Implantation

Call 911 for:	A shock from a defibrillator AND you do not feel well—for example, you have passed out or have dizziness, shortness of breath, or chest pain. More than 1 shock in a row from your device.
Call the physician's office immediately for:	Redness around the incision site. Swelling. Drainage or any separation of the incision. Fever >99.5°F (37.5°C). Chills. Any concerns or questions regarding your wound. If you receive a shock from your device AND you are feeling fine. *Keep a log of the shocks you receive. It is useful for your physician to know the number of shocks you received, how you feel after each shock, the time of day the shocks occurred, and what you were doing at the time the shocks were delivered.*
Wound care:	Remove your dressing (bandage) within 24–48 hr after your surgery. Do not put lotion or powder on your incision until it is completely healed. Avoid touching the area over or around your device. Do not poke at or twist your device. Add padding around your seatbelt and keep it in place until the wound is completely healed. If your wound is closed with skin glue, you may shower 48 hr after surgery. If Steri-Strips are used, you may shower 5 days following surgery. Do not soak or submerge your incision in water until it is completely healed. Do not allow anyone to poke or probe your incision with fingers or instruments without consulting the implanting physician first. Do not allow anyone to poke a needle into the device pocket without consulting the implanting physician first.
Activity restrictions:	Wear a sling for the first 24 hr following surgery if your doctor prescribed one. Afterward, wearing a sling during sleep is permissible for 1 week after implant, but no longer. If you have travel plans in the first 2 weeks after your surgery, please check with the implanting physician beforehand. For the first 30 days after your surgery: • Do not push, pull, strain, twist, or lift anything over your head with the arm on the side of your implant. • Try to avoid sudden and forceful movements with your arms or upper body. • Do not lift more than 10–15 pounds or lift your elbow higher than your shoulder on the side of your implant. • Try not to raise your arm over your head. • Avoid extending your arm behind your back.
Long term:	Check with your doctor before you do activities that involve repetitively swinging your arm on the side of your implant (such as swimming, golfing, tennis, or vacuuming) or engaging in activities that can injure your shoulder or the incision site (such as shooting a gun, wrestling, or playing football). These restrictions could last 3 months or longer, based on your treatment plan that your physician has outlined for you. Continue to avoid touching the area over or around your device. Do not poke at or twist your device. Questions regarding the use of specific types of equipment should be directed to the engineers who work for the manufacturer of your device. Their contact information will be on your Device Identification Card.

TABLE 15.4 Sample Same-Day Discharge Protocol for Cardiac Implantable Electronic Device Patients

Patients will be observed for 3–6 hr postprocedure in recovery or holding area:
- Immediate upright portable anterior-posterior CXR
- Posterior-anterior and lateral chest CXR performed 3–4 hr postprocedure to confirm stable lead position(s) and reevaluate for pneumothorax
- Implanting provider or designee examines the patient and reviews the device interrogation with documentation in the electronic medical record.
- Document patient's cell or home number for follow-up telephone call the next day.

Discharge orders should be completed early to allow for pain medication and antibiotic prescriptions to be filled and be ready by the time of discharge.
Postoperative IV antibiotics can be given per physician discretion.
Surgical site inspection performed immediately before discharge
Education before discharge:
- Patient teaching/instructions
- Sling instructions (not more than 1 day)
- Monitor instructions (plug in—whether to push button)
- Bandage instructions (take off next day)
- Review symptoms to call for (chest pain, SOB, palpitations, pocket swelling or drainage, and implantable cardioverter-defibrillator shocks).

Patients will receive and be paired with their manufacturer-specific home monitor.
- Training is provided by the industry-employed allied professional or the cardiac implantable electronic device–trained device clinic nurses.

Patients will be scheduled for a remote transmission on the day after discharge.
Patients receive a follow-up phone call on postoperative day 1 from device clinic RN.
- Ask about symptoms such as dyspnea, chest pain, swelling at site, and overall health.
- Check that the patient removed bandage and sling.
- Troubleshoot remote monitoring process if necessary.
- Document follow-up and device information in the electronic medical record.

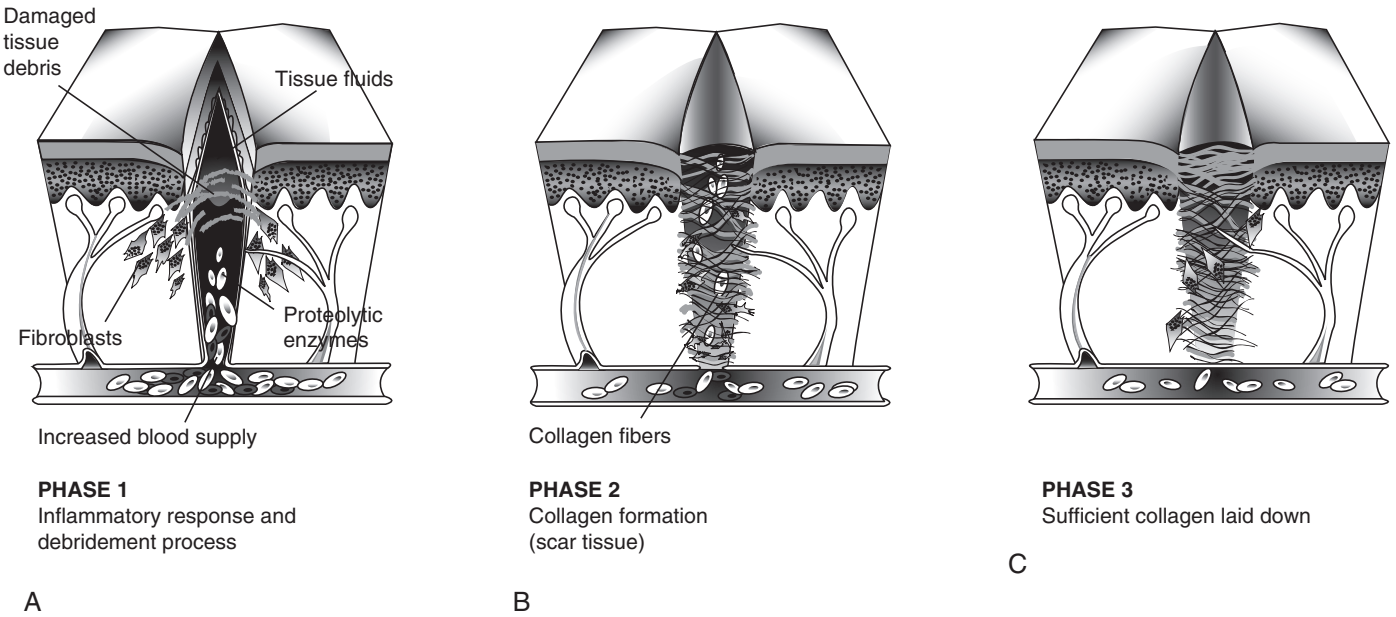
Patients may be seen in the clinic 1 day postoperatively for a follow-up posteroanterior and lateral CXR and device interrogation if the physician deems appropriate.
Patients will follow up in 10–14 days for a wound check and device interrogation.

CXR, chest x-ray; IV, intravenous; RN, registered nurse; SOB, shortness of breath.

TABLE 15.5 Traveling With a Cardiac Implantable Electronic Device

- Always carry your Device Identification Card.
- If you must pass through a metal detector, hand your identification card to security staff and tell them that you might set off the alarm.
- Body scanners that are used in some airports and other locations will not have any effect on your device.
- You may walk through the metal detection arch, but do not remain inside the arch or lean on the sides of the structure.
- If a metal detection hand wand is used, ask security staff to avoid waving or holding it over your device.
- If you feel dizzy, have fast heartbeats (palpitations), or are shocked by your defibrillator when you are near a metal detector, rapidly move farther away. Your defibrillator should begin to work properly right away.
- Always carry a complete list of your current medications. Include their dosage, how often you take them each day, and why you are taking them.
- Always carry your medications with you in your carry-on bags. Do not pack them in your checked luggage.
- Carry phone numbers for your health care providers in case of emergency.

The first postoperative visit also provides an excellent opportunity to provide a systematic review of the function of remote monitoring. It should be emphasized to the patient that remote monitoring is not a substitute for face-to-face evaluation, and that remote monitoring does not provide "real-time" cardiac monitoring. Explaining to the patient that the health care team may not be notified of recorded events until the following morning or next business day is critical so that patients will know to access medical care urgently for serious events. Patient questions should also be addressed at this appointment, including a reminder that for any critical events (acute symptoms such as chest pain, shortness of breath, syncope, or ICD shocks), the patient should seek immediate medical attention.



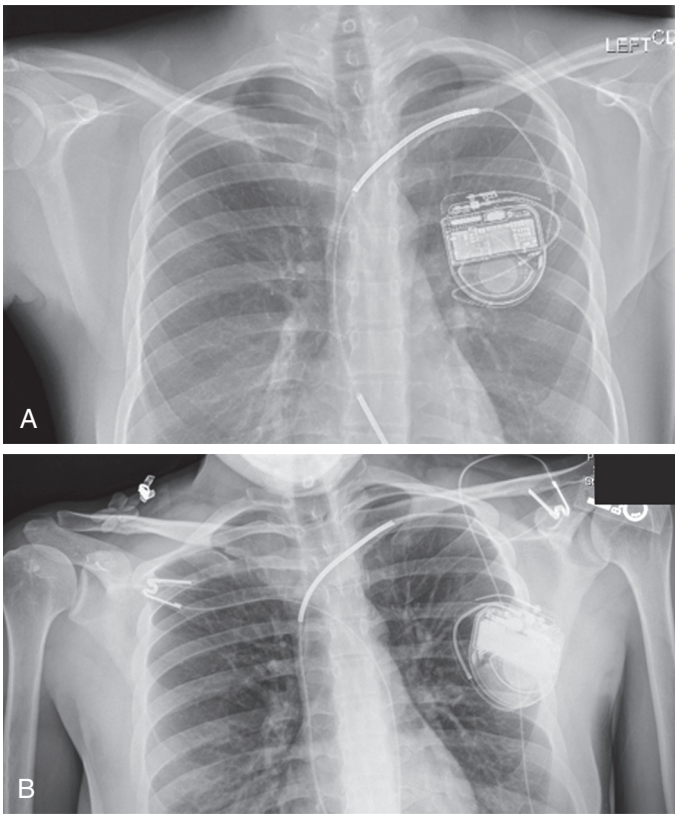
• **Fig. 15.7** Wound healing phases over time: phase 1 (A), phase 2 (B), and phase 3 (C). (From Pieknik R. *Suture and Surgical Hemostasis: A Pocket Guide*. Philadelphia: Saunders; 2006.)

Special circumstances, such as those encountered with traveling, should be discussed if applicable. Some of these considerations are listed in Table 15.5. Taking the time to provide adequate education to the patient and review precautions will go a long way to allay patient concerns, alleviate anxiety, and diminish perceived pain.

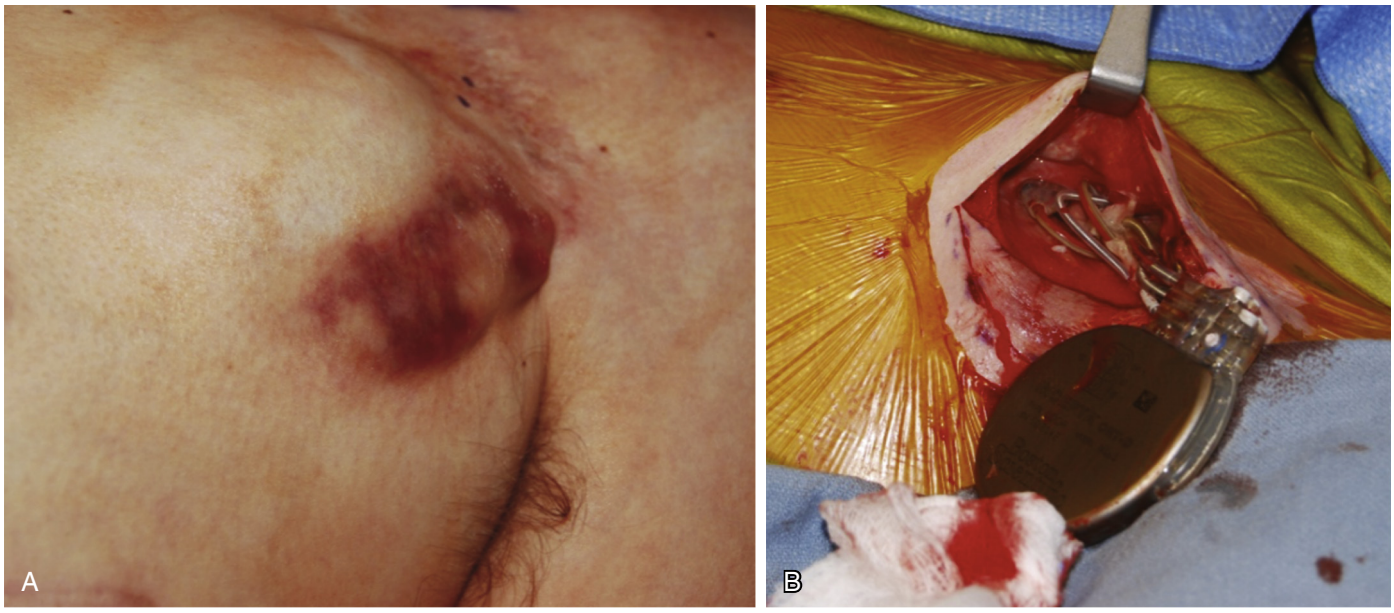
First Year After CIED Implantation

Routine follow-up clinic or remote monitoring appointments are scheduled based on institutional device follow-up protocols, the needs of the patient, and the directive of the implanting physician.

By 3 months, the wound should be well healed, and any hematoma and ecchymosis present from liquefaction of the hematoma should be resolved. As epithelium is regenerated, new capillary loops are formed and macrophages decrease in number, resulting in maturation of the surgical incision. In the late phase of wound healing, the epithelium is healed completely and collagen fibers mature. The process of collagen replacement and scar remodeling continues for years (Fig. 15.7). Care should be taken to inquire into symptoms and findings that may suggest late lead dislodgement, delayed myocardial perforation, or migration of the generator (Fig. 15.8). Device-related infections could occur out to a year or more following CIED implantation (see Chapter 14). Some patients will present with chronic pocket discomfort late after implantation. An infectious etiology must always be considered; however, occasionally the cause of the pocket discomfort may be pulse generator migration with impingement on neighboring structures (Fig. 15.9A–B).



• **Fig. 15.8** Defibrillator generator migration into left axilla during vigorous exercise: initial postimplant position (A) and after migration (B).



• **Fig. 15.9** Patient presenting with chronic pain in defibrillator pocket presenting 4 months following implantation. (A) Examination of pocket demonstrates ecchymosis over lateral border. (B) Surgical exploration demonstrates purulence within the generator pocket.

Summary

Postoperative management of the patient with a CIED implant begins immediately after the implantation and continues well beyond discharge. An understanding of the procedure, wound management, nutritional status, device function and interrogation, and potential complications and their presentation are paramount to deliver competent patient care.

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