1 Pacing and Defibrillation: Clinically Relevant Basics for Practice

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Anatomy and physiology of the cardiac conduction system

The cardiac conduction system consists of specialized tissue involved in the generation and conduction of electrical impulses throughout the heart. Knowledge of the normal anatomy and physiology of the cardiac conduction system is critical to understanding appropriate utilization of device therapy.

The sinoatrial (SA) node, located at the junction of the right atrium and the superior vena cava, is normally the site of impulse generation (Fig. 1.1). The SA node is composed of a dense collagen matrix containing a variety of cells. The large, centrally located P cells are thought to be the origin of electrical impulses in the SA node, which is surrounded by transitional cells and fiber tracts extending through the perinodal area into the remainder of the right atrium. The SA node is richly innervated by the autonomic nervous system, which has a key function in heart rate regulation. Specialized fibers, such as Bachmann's bundle, conduct impulses throughout the right and left atria. The SA node has the highest rate of spontaneous depolarization and under normal circumstances is responsible for generating most impulses. Atrial depolarization is seen as the P wave on the surface electrocardiogram (ECG; Fig. 1.1).

Atrial conduction fibers converge, forming multiple inputs into the atrioventricular (AV) node, a small subendocardial structure located within the interatrial septum (Fig. 1.1). The AV node also receives abundant autonomic innervation, and it is histologically similar to the SA node because it is composed of a loose collagen matrix in which P cells and transitional cells are located. Additionally, Purkinje cells and myocardial contractile fibers may be found. The AV node allows for physiologic delay between atrial and ventricular contraction, resulting in optimal cardiac hemodynamic function. It can also function as a subsidiary "pacemaker" should the SA node fail. Finally, the AV node functions (albeit typically suboptimally) to regulate the number of impulses eventually reaching the ventricle in instances of atrial tachyarrhythmia. On the surface ECG, the majority of the PR interval is represented by propagation through the AV node and through the His–Purkinje fibers (Fig. 1.1).

Purkinje fibers emerge from the distal AV node to form the bundle of His, which runs through the membranous septum to the crest of the muscular septum, where it divides into the various bundle branches. The bundle branch system exhibits significant individual variation. The right bundle is typically a discrete structure running along the right side of the interventricular septum to the moderator band, where it divides. The left bundle is usually a large band of fibers fanning out over the left ventricle, forming functional fascicles. Both bundles eventually terminate in individual Purkinje fibers interdigitating with myocardial contractile fibers. The His–Purkinje system has correspondingly less autonomic innervation than the SA and AV nodes.

Because of their key function and location, the SA and AV nodes are the most common sites of conduction system failure; it is understandable, therefore, that the most common indications for pacemaker implantation are SA node dysfunction and high-grade AV block. It should be noted, however, that conduction system disease is frequently diffuse and may involve the specialized conduction system at multiple sites.

Electrophysiology of myocardial stimulation

Stimulation of the myocardium requires initiation of a propagating wave of depolarization from the site of initial activation, whether from a native "pacemaker" or from an artificial stimulus. Myocardium exhibits "excitability," which is a response to a stimulus out of proportion to the strength of that stimulus.[1] Excitability is maintained by separation of chemical charge, which results in an electrical transmembrane potential. In cardiac myocytes, this electrochemical gradient is created by differing intracellular and extracellular concentrations of sodium (Na^+) and potassium (K^+) ions; Na^+ ions predominate extracellularly, and K^+ ions predominate intracellularly. Although this transmembrane gradient is maintained by the high chemical resistance intrinsic to the lipid bilayer of the cellular membrane, passive leakage of these ions occurs across the cellular membrane through ion channels. Passive leakage is offset by two active transport mechanisms, each transporting three positive charges out of the myocyte in exchange for two positive charges that are moved into the myocyte, producing cellular polarization.[2,3] These active transport mechanisms require energy and are susceptible to disruption when energy-generating processes are interrupted.

The chemical gradient has a key role in the generation of the transmembrane action potential (Fig. 1.2). The membrane potential of approximately −90 mV drifts upward to the threshold potential of approximately −70 to −60 mV. At this point, specialized membrane-bound channels modify their conformation from an inactive to an active state, which allows the abrupt influx of extracellular Na^+ ions into the myocyte,[4,5] creating phase 0 of the action potential and rapidly raising the transmembrane potential to approximately +20 mV.[6,7] This rapid upstroke creates a short period of overshoot potential (phase 1), which is followed by a plateau period (phase 2) created by the

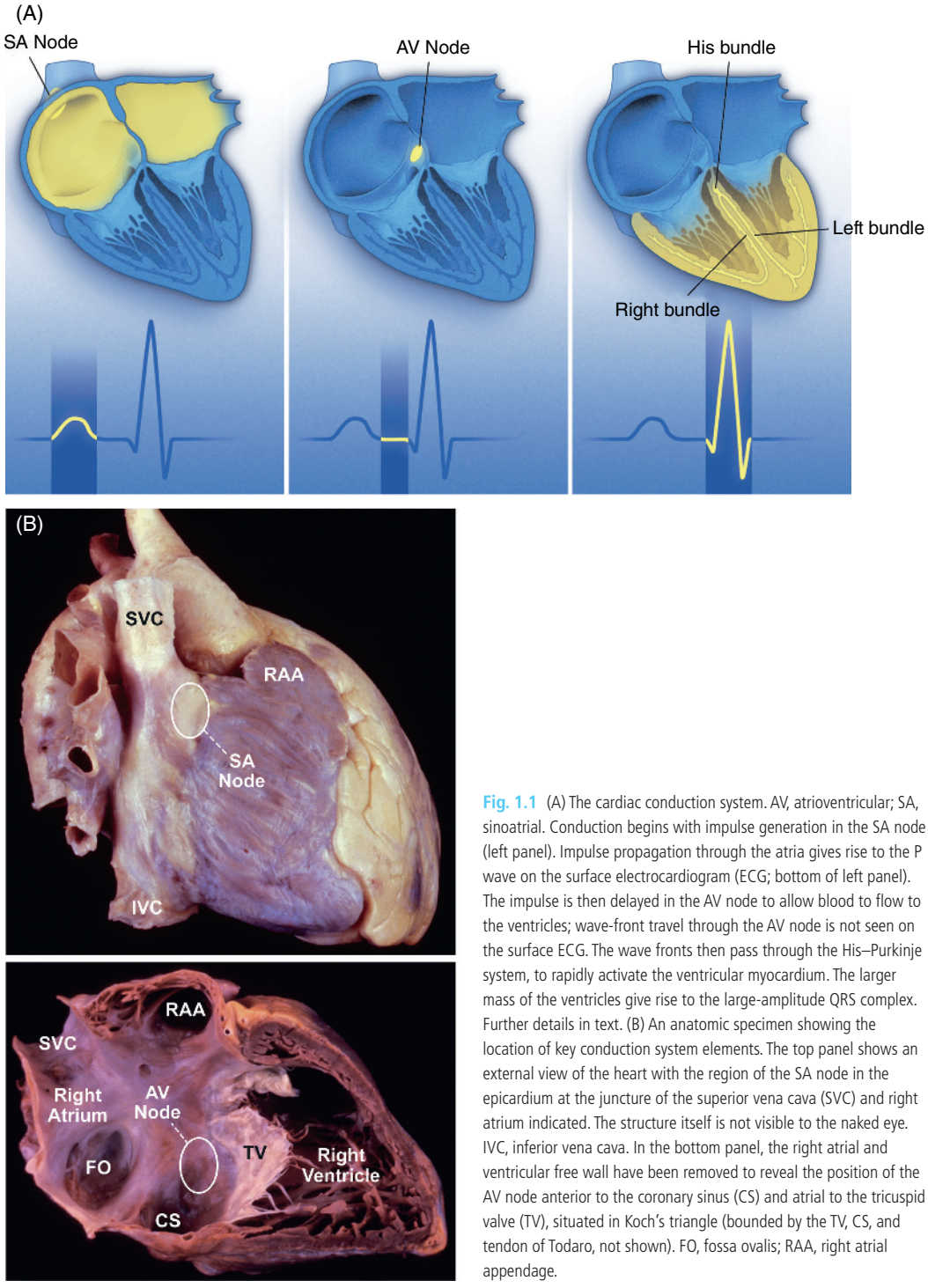


Fig. 1.1 (A) The cardiac conduction system. AV, atrioventricular; SA, sinoatrial. Conduction begins with impulse generation in the SA node (left panel). Impulse propagation through the atria gives rise to the P wave on the surface electrocardiogram (ECG; bottom of left panel). The impulse is then delayed in the AV node to allow blood to flow to the ventricles; wave-front travel through the AV node is not seen on the surface ECG. The wave fronts then pass through the His–Purkinje system, to rapidly activate the ventricular myocardium. The larger mass of the ventricles give rise to the large-amplitude QRS complex. Further details in text. (B) An anatomic specimen showing the location of key conduction system elements. The top panel shows an external view of the heart with the region of the SA node in the epicardium at the juncture of the superior vena cava (SVC) and right atrium indicated. The structure itself is not visible to the naked eye. IVC, inferior vena cava. In the bottom panel, the right atrial and ventricular free wall have been removed to reveal the position of the AV node anterior to the coronary sinus (CS) and atrial to the tricuspid valve (TV), situated in Koch's triangle (bounded by the TV, CS, and tendon of Todaro, not shown). FO, fossa ovalis; RAA, right atrial appendage.

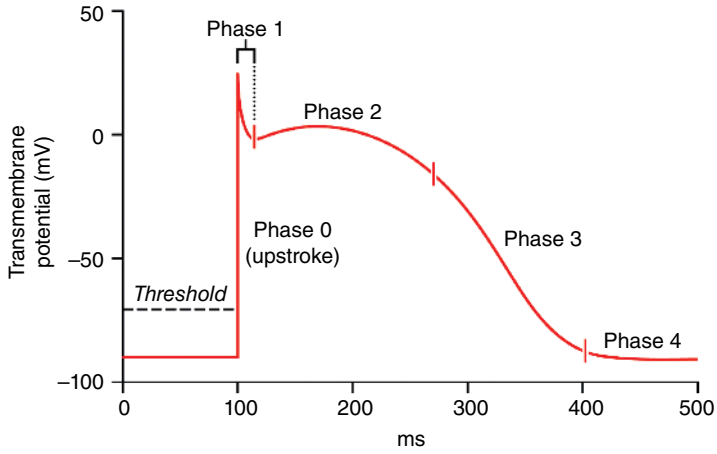


Fig. 1.2 Action potential of a typical Purkinje fiber, with the various phases of depolarization and repolarization (described in the text). (Source: From Stokes KB, Kay GN. Artificial electric cardiac stimulation. In: Ellenbogen KA, Kay GN, Wilkoff BL, eds. Clinical Cardiac Pacing. Philadelphia: WB Saunders Co., 1995: 3–37, by permission of the publisher.)

inward calcium (Ca^{2+}) and Na^{+} currents balanced against outward K^{+} currents.[8–10] During phase 3 of the action potential, the transmembrane potential returns to normal, and during phase 4 the gradual upward drift in transmembrane potential repeats. The shape of the transmembrane potential and the relative distribution of the various membrane-bound ion channels differ between the components of the specialized cardiac conduction system and working myocytes, allowing for their differing regional purposes and functions.

Depolarization of neighboring cells occurs as a result of passive conduction via low-resistance intercellular connections called "gap junctions," with active regeneration along cellular membranes.[11,12] The velocity of depolarization throughout the myocardium depends on the speed of depolarization of the various cellular components of the myocardium and on the geometric arrangement and orientation of the myocytes. Factors such as myocardial ischemia, electrolyte imbalance, metabolic abnormalities, myocardial scar, diseased tissue, and drugs affect depolarization and depolarization velocity.

Pacing basics

Understanding cardiac pacing requires an intimate consideration of two key elements: stimulation threshold (i.e., the minimum amount of output required to stimulate a sufficient quantity of cells so as to facilitate initiation of a propagating impulse) and sensing (i.e., the ability of a device to detect a propagating impulse amidst the cellular milieu). These elements are facilitated by a variety of factors related to lead design, interface between leads and myocardial tissue, and other factors, as outlined later in this chapter.

Stimulation threshold

Artificial pacing involves delivery of an electrical impulse from an electrode of sufficient strength to cause depolarization of the myocardium in contact with that electrode and propagation of that depolarization to the rest of the myocardium. The minimal amount of energy required to produce this depolarization is called the stimulation threshold. The components of the stimulus include the pulse amplitude (measured in volts) and the pulse duration (measured in milliseconds). An exponential relationship exists between the stimulus amplitude and the duration, resulting in a hyperbolic strength–duration curve. At short pulse durations, a small change in the pulse duration is associated with a significant change in the pulse amplitude required to achieve myocardial depolarization; conversely, at long pulse durations, a small change in pulse duration has relatively little effect on threshold amplitude (Fig. 1.3). Two points on the strength–duration curve should be noted (Fig. 1.4). The *rheobase* is defined as the smallest amplitude (voltage) that stimulates the myocardium at an infinitely long pulse duration (milliseconds). The *chronaxie* is the threshold pulse duration at twice the rheobase voltage. The chronaxie is important in the clinical practice of pacing because it approximates the point of minimum threshold energy (microjoules) required for myocardial depolarization.

The relationship of voltage, current, and pulse duration to stimulus energy is described by the formula

$$E = \frac{V^2 \times t}{R}$$

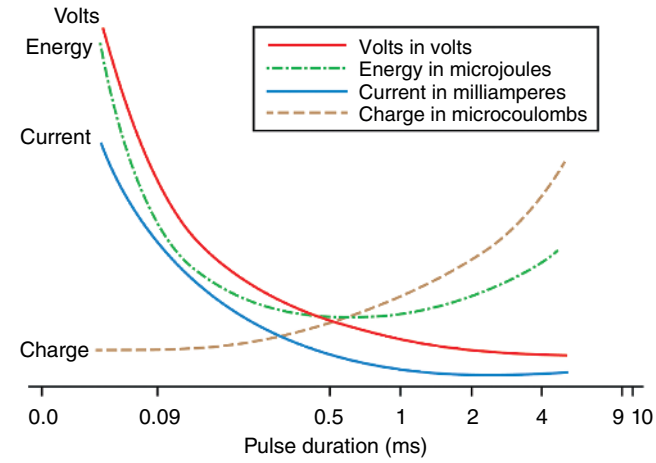


Fig. 1.3 Relationship of charge, energy, voltage, and current to pulse duration at stimulation threshold. As the pulse duration is shortened, voltage and current requirements increase. Charge decreases as pulse duration shortens. At threshold, energy is lowest at a pulse duration of 0.5–1.0 ms and increases at pulse widths of shorter and longer duration. (Source: Modified from Furman S. Basic concepts. In: Furman S, Hayes DL, Holmes DR Jr, eds. A Practice of Cardiac Pacing. Mount Kisco, NY: Futura Publishing Co., by permission of the publisher.)

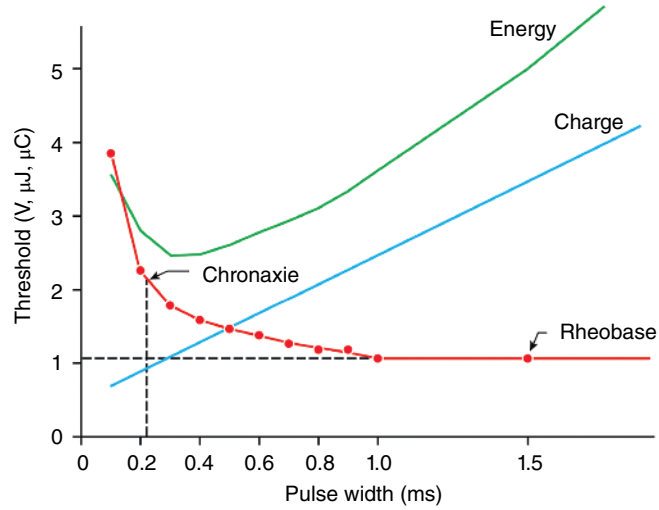


Fig. 1.4 Relationships among chronic ventricular strength–duration curves from a canine, expressed as potential (V), charge (μC), and energy (μJ). Rheobase is the threshold at infinitely long pulse duration. Chronaxie is the pulse duration at twice rheobase. (Source: From Stokes K, Bornzin G. The electrode–biointerface stimulation. In: Barold SS, ed. Modern Cardiac Pacing. Mount Kisco, NY: Futura Publishing Co., 1985: 33–77, by permission of the publisher.)

in which E is the stimulus energy, V is the voltage, R is the total pacing impedance, and t is the pulse duration. This formula demonstrates the relative increase in energy with longer pulse durations. The energy increase due to duration is offset by a decrement in the voltage needed. The strength–duration curve discussed thus far has been that of a constant-voltage system, which is used in all current pacemakers and defibrillators. Constant-current devices are no longer used. The singular exception to this is biologic pacemakers, which allow for use of cells repurposed to recreate the body's intrinsic conduction system. However, this approach is not presently available for human use and thus will not be discussed here.

Impedance is the term applied to the resistance to current flow in the pacing system. Ohm's law describes the relationship among voltage, current, and resistance as

$$V = IR$$

in which V is the voltage, I is the current, and R is the resistance. Although Ohm's law is used for determining impedance, the impedance and resistance are technically not interchangeable terms. Impedance implies inclusion of all factors that contribute to current flow impediment, including lead conductor resistance, electrode resistance, resistance due to electrode polarization, capacitance, and inductance. Technically, the term "resistance" does not include the effects of capacitance

(storage of charge) or inductance (storage of current flow) to impede current flow. Nevertheless, Ohm's law (substituting impedance for *R*) is commonly used for calculating impedance. In constant-voltage systems, the lower the pacing impedance, the greater the current flow; conversely, the higher the pacing impedance, the lower the current flow. Lead conductors are designed to have a low resistance to minimize the generation of energy-wasting heat as current flows along the lead, and electrodes are designed to have a high resistance to minimize current flow and to have negligible electrode polarization. Decreasing the electrode radius minimizes current flow by providing greater electrode resistance and increased current density, resulting in greater battery longevity and lower stimulation thresholds.[13]

"Polarization" refers to layers of oppositely charged ions that surround the electrode during the pulse stimulus. It is related to the movement of positively charged ions (Na^+ and H_3O^+) to the cathode; the layer of positively charged ions is then surrounded by a layer of negatively charged ions (Cl^-, HPO_4^{2-}, and OH^-). These layers of charge develop during the pulse stimulus, reaching peak formation at the termination of the pulse stimulus, after which they gradually dissipate. Polarization impedes the movement of charge from the electrode to the myocardium, resulting in a need for increased voltage for stimulation. As polarization develops with increasing pulse duration, one way to combat formation of polarization is to shorten the pulse duration. Electrode design has incorporated the use of materials that minimize polarization, such as platinum black, iridium oxide, titanium nitride, and activated carbon.[14] Finally, polarization is inversely related to the surface area of the electrode. To maximize the surface area (to reduce polarization) but minimize the radius (to increase electrode impedance), electrode design incorporates a small radius but a porous, irregular surface construction.[15] Fractal coatings on the lead tip increase the surface area 1000-fold without the need to increase the axial diameter. Leads designed to maximize these principles are considered "high-impedance" leads.

Variations in stimulation threshold

Myocardial thresholds typically fluctuate, occasionally dramatically, during the first weeks after implantation. After implantation of earlier generations of endocardial leads, the stimulation threshold would typically rise rapidly in the first 24 h and then gradually increase to a peak at approximately 1 week (Fig. 1.5). Over the ensuing 6–8 weeks, the stimulation threshold would usually decline to a level somewhat higher than that at implantation, but less than the peak threshold, known as the "chronic threshold."[16,17] The magnitude and duration of this early increase in threshold was highly dependent on lead design, the interface between the electrode and the myocardium, and individual patient variation, but chronic thresholds would typically be reached by 3 months. The single most important lead design change to alter pacing threshold evolution was the incorporation of steroid elution at the lead tip, to blunt the local inflammatory response (Fig. 1.6). With steroid

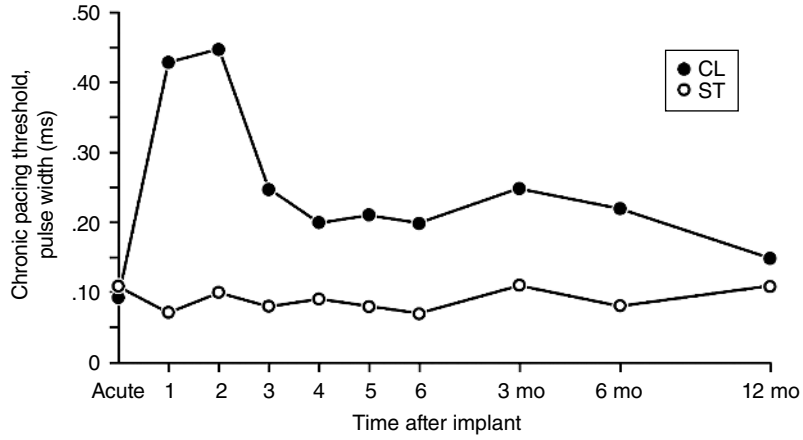


Fig. 1.5 Long-term pacing thresholds from a conventional lead (CL; no steroid elution) and a steroid-eluting lead (ST). With the conventional lead, an early increase in threshold decreases to a plateau at approximately 4 weeks. The threshold for the steroid-eluting lead remains relatively flat, with no significant change from short-term threshold measurements. (Source: From Furman S. Basic concepts. In: Furman S, Hayes DL, Holmes DR Jr, eds. A Practice of Cardiac Pacing, 2nd edn. Mount Kisco, NY: Futura Publishing Co., 1989: 23–78, by permission of Mayo Foundation.)

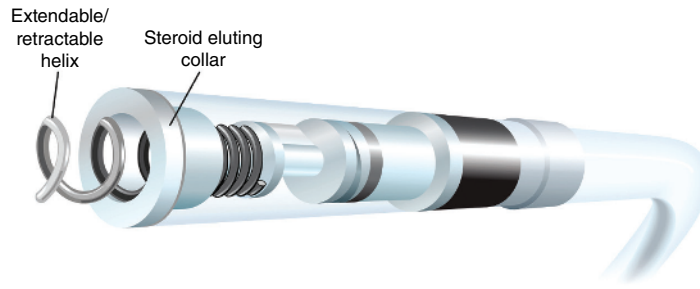


Fig. 1.6 Diagram of a steroid-eluting active fixation lead. The electrode has a porous, platinized tip. A silicone rubber collar is impregnated with 1 mg of dexamethasone sodium.

elution there may be a slight increase in thresholds postimplantation, with subsequent reduction to almost that of acute thresholds.[18,19]

Transvenous pacing leads have used passive or active fixation mechanisms to provide a stable electrode–myocardium interface. Active fixation leads may have higher initial pacing thresholds acutely at implantation, but thresholds frequently decline significantly within the first 5–30 min after placement.[16] This effect has been attributed to hyperacute injury due to advancement of the screw into the myocardium. On a cellular level, implantation of a transvenous pacing lead results in acute injury to cellular membranes, which is followed by the development of myocardial edema and coating of the electrode surface with platelets and fibrin. Subsequently, various chemotactic factors are released, and an acute inflammatory reaction develops, consisting of mononuclear cells and polymorphonuclear leukocytes. After the acute response, release of proteolytic enzymes and oxygen free radicals by invading macrophages accelerates cellular injury. Finally, fibroblasts in the myocardium begin producing collagen, leading to production of the fibrotic capsule surrounding the electrode. This fibrous capsule ultimately increases the effective radius of the electrode, with a smaller increase in surface area.[20,21] Steroid-eluting leads are believed to minimize fibrous capsule formation. In both atrial and ventricular active fixation leads, steroid elution results in long-term reduction in energy consumption with maintenance of stimulation thresholds, lead impedance values, and sensing thresholds.[22,23]

The stimulation threshold may vary slightly with a circadian pattern, generally increasing during sleep and decreasing during the day, probably reflecting changes in autonomic tone. The stimulation threshold may also rise after eating; during hyperglycemia, hypoxemia, or acute viral illnesses; or as a result of electrolyte fluctuations. In general, these threshold changes are minimal. However, in the setting of severe hypoxemia or electrolyte abnormalities they can lead to loss of capture. Certain drugs used in patients with cardiac disease may also increase pacing thresholds – see Chapter 8 (Programming: Maximizing Benefit and Minimizing Morbidity Programming).

The inflammatory reaction and subsequent fibrosis that occur after lead implantation may act as an insulating shield around the electrode. These processes effectively increase the distance between the electrode and the excitable tissue, allowing the stimulus to disperse partially before reaching the excitable cells. These changes result in an increased threshold for stimulation and attenuate the amplitude and slew rate of the endocardial signal being sensed. This is a process termed "lead maturation." Improvements in electrode design and materials have reduced the severity of the inflammatory reaction and, thus, improved lead maturation rates.[18,24] When the capture threshold exceeds the programmed output of the pacemaker, exit block will occur; loss of capture will result if the capture threshold exceeds the programmed output of the pacemaker.[16,25] Exit block, a consequence of lead maturation, results from the progressive rise in thresholds over time.[16,25] This phenomenon occurs despite initial satisfactory lead placement and implantation thresholds, often but not always occurs in parallel in the atrium and ventricle, and usually recurs with placement of subsequent leads. Steroid-eluting leads prevent exit block in most, but not all, patients (Fig. 1.6).

Clinical considerations when considering threshold

Several clinical considerations are needed when evaluating stimulation threshold and using this to guide programming of the device. One key consideration is that of cathodal versus anodal stimulation and how this relates to local myocardial capture. In cathodal stimulation, electrons "enter" the muscle from the electrode, resulting in relatively low capture thresholds and minimal electrode corrosion. Specifically, capture occurs at the tip electrode. During anodal stimulation, myocardial capture occurs at the anode, which, in bipolar pacing configurations in which both cathode and anode are in contact with myocardium, generally occurs at the level

of the ring electrode. Capture during cathodal stimulation occurs at the onset of the impulse (or the leading edge). In turn, anodal stimulation occurs at the trailing edge of the stimulus. One key issue with anodal stimulation is that the resulting myocardial refractory period tends to be shorter, and one particular concern (particularly at faster heart rates) is an R-on-T phenomenon that could result in ventricular fibrillation (VF). Risk of anodal stimulation is minimized by making the anode as large as possible to reduce electron density.

The clinical area in which anodal stimulation is of particular importance in modern pacing systems is cardiac resynchronization devices. This is of specific importance in cases where resynchronization devices are set to pace from the tip of the left ventricular lead (cathode) to the right ventricular (RV) ring (anode). This pacing modality is often used when phrenic pacing is present during bipolar left ventricular lead pacing. At higher outputs, it is possible to have anodal stimulation from the RV ring, in which case three wave fronts are possible: one from the left ventricular tip (cathodal, left ventricle paced), one from the RV tip (cathodal, right ventricle paced), and one from the RVr ring (anodal). These three wave fronts can result in less than desirable pacing, resulting in nonresponse to resynchronization. The risk of this is minimized by using an integrated RV lead, where the shocking coil is the ring electrode, making anodal capture extremely unlikely due to the large surface area of the anode.

Threshold-specific programming

The threshold defines specific safety margins for programming of a device. Specifically, by understanding the minimum output needed to consistently capture the myocardium, one can program a device at a level that will ideally consistently capture myocardium even if thresholds change for reasons described previously. Generally, at the time of initial implant, programming at a relatively high output (as much as five times the threshold) may be considered. However, this may be limited by higher initial thresholds. Over chronic use, when it is felt threshold has more or less stabilized, programming a two times safety factor above the stimulation threshold may be reasonable. As already described above, however, one key consideration is the risk of anodal stimulation in resynchronization devices. Programming too-high outputs based on stimulation thresholds may be guided by anodal stimulation seen at higher outputs, or even the potential for capture of surrounding tissues of interest (phrenic nerve, diaphragm, etc.). Thus, a stimulation threshold may be considered in both directions: the minimum output at which the myocardium is captured, and the maximum output at which undesirable capture of surrounding tissues or anodal stimulation may occur. Programming of a device would thus require consideration of these lower and upper thresholds while simultaneously allowing for a sufficient safety margin. It should also be noted that programmed output of pacing at the time of initial device implant (when it cannot be certain how the threshold will stabilize), at the time of chronic follow-up (when it may be hoped that thresholds are more or less stabilized), and when considering the clinical situation at the time of pacing is critical. In the last scenario, one can specifically consider the situation of postdefibrillation pacing, when stimulation thresholds may be increased due to tissue ischemia during the preceding tachyarrhythmia or at the time of defibrillation.

Sensing

The first pacemakers functioned as fixed-rate, VOO devices. All contemporary devices offer demand-mode pacing, which pace only when the intrinsic rate is below the programmed rate. For such devices to function as programmed, accurate and consistent sensing of the native rhythm are essential.

Intrinsic cardiac electrical signals are produced by the wave of electrical current through the myocardium (Fig. 1.7). As the wave front of electrical energy approaches an endocardial unipolar electrode, the intracardiac electrogram records a positive deflection. As the wave front passes directly under the electrode, a sharp negative deflection is recorded, referred to as the intrinsic deflection.[26] The intrinsic deflection is inscribed as the advancing wave front passes directly underneath a unipolar electrode. Smaller positive and negative deflections preceding and following the intrinsic deflection represent activation of surrounding myocardium. The analog on the surface ECG is the peak of the R wave, referred to as the intrinsicoid deflection, because the electrical depolarization is measured at a distance (from the surface), rather than directly on the myocardium. However, the intrinsic deflection is a local endocardial event; it does not necessarily time with the intrinsicoid deflection in any ECG lead. Bipolar electrograms (Fig. 1.7) represent the difference in potential recorded between two closely spaced intracardiac electrodes. Owing to the close spacing of two typically small electrodes, far-field signals (i.e., signals not generated by the tissue the lead electrode is in contact with) are smaller and thus more easily rejected by pacemakers and defibrillators. Ventricular electrograms typically are much larger than atrial electrograms because ventricular mass is greater. Typical amplitude ranges for ventricular electrograms are 5–25 mV, and for atrial electrograms 1.5–5 mV (Fig. 1.8). The maximum frequency densities of electrograms in sinus rhythm are in the range of 80–100 Hz in the atrium and 10–30 Hz in the ventricle (these frequencies

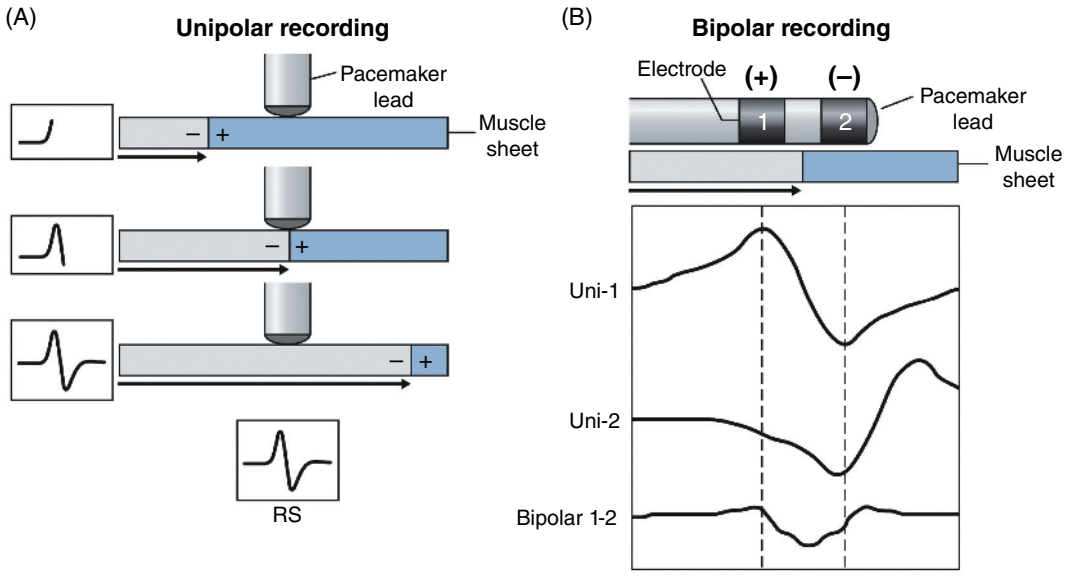


Fig. 1.7 Schema of the relationship of the pacing lead to the recorded electrogram with (A) unipolar and (B) bipolar sensing. (A) Top: as the electrical impulse moves toward the cathode (lead tip), a positive deflection is created in the electrogram. Middle: as the electrical impulse passes the cathode, the deflection suddenly moves downward, at the intrinsic deflection. Bottom: as the impulse moves away from the cathode, a negative deflection occurs. (B) The same phenomena occur for each electrode of a bipolar lead when considered independently (uni-1 and uni-2). When these are put together as a bipolar signal, the resultant tracing is seen at the bottom (bipolar 1–2). (Source: Adapted from Stevenson WG, Soejima K. Recording techniques for clinical electrophysiology. J Cardiovasc Electrophysiol 2005; 16:1–6.)

may differ slightly depending on leads and/or technologies). Pulse generator filtering systems are designed to attenuate signals outside these ranges. Filtering and use of blanking and refractory periods (discussed later) have markedly reduced unwanted sensing, although myopotential frequencies (ranging from 10 to 200 Hz) considerably overlap with those generated by atrial and ventricular depolarization and are difficult to filter out, especially during sensing in a unipolar configuration.[27–29] Shortening of the tip-to-ring spacing has also improved atrial sensing and rejection of far-field R waves.

A second important metric of the intracardiac electrogram in addition to amplitude is the slew rate; that is, the peak slope of the developing electrogram[30] (Fig. 1.8). The slew rate represents the maximal rate of change of the electrical potential between the sensing electrodes and is the first derivative of the electrogram (dV/dt). An acceptable slew rate should be at least 0.5 V/s in both the atrium and the ventricle. In general, the higher the slew rate, the higher the frequency content and the more likely the signal will be sensed. Slow, broad signals, such as those generated by the T wave, are less likely to be sensed because of a low slew rate and lower frequency density.

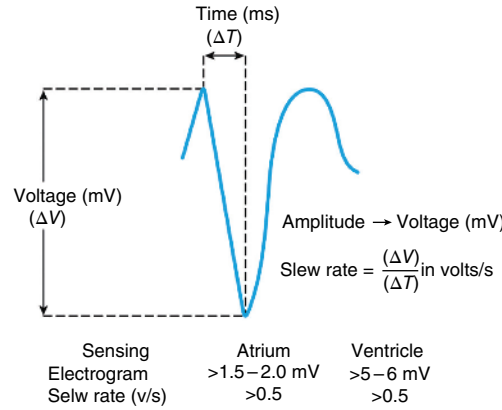


Fig. 1.8 In the intracardiac electrogram, the difference in voltage recorded between two electrodes is the amplitude, which is measured in millivolts. The slew rate is volts per second and should be at least 0.5.

Polarization also affects sensing function. After termination of the pulse stimulus, an excess of positive charge surrounds the cathode, which then decays until the cathode is electrically neutral. Afterpotentials can be sensed, resulting in inappropriate inhibition or delay

of the subsequent pacing pulse (Fig. 1.9). The amplitude of afterpotentials is directly related to both the amplitude and the duration of the pacing pulse; thus, they are most likely to be sensed when the pacemaker is programmed to high voltage and long pulse duration in combination with maximal sensitivity.[30] The use of programmable sensing refractory and blanking periods has helped to prevent the pacemaker from reacting to afterpotentials, although, in dual-chamber systems, atrial afterpotentials of sufficient strength and duration to be sensed by the ventricular channel may result in inappropriate ventricular inhibition (crosstalk), especially in unipolar systems.[31,32] Afterpotentials may be a source of problems in devices with automatic threshold measurement and capture detection; the use of leads designed to minimize afterpotentials may increase the effectiveness of such algorithms.[33]

"Source impedance" is the impedance from the heart to the proximal portion of the lead, and it results in a voltage drop from the site of the origin of the intracardiac electrogram to the proximal portion of the lead.[34] Components include the resistance between the electrode and the myocardium, the resistance of the lead conductor material, and the effects of polarization. The resistance between the electrode and the myocardium, as well as polarization, is inversely related to the surface area of the electrode; thus, the effects of both can be minimized by a large electrode surface area. The electrogram actually seen by the pulse generator is determined by the ratio between the sensing amplifier (input) impedance and the lead (source) impedance. Less attenuation of the signal from the myocardium occurs when there is a greater ratio of input impedance to source impedance. Clinically, impedance mismatch is seen with insulation or conductor failure, which results in sensing abnormalities or failure.

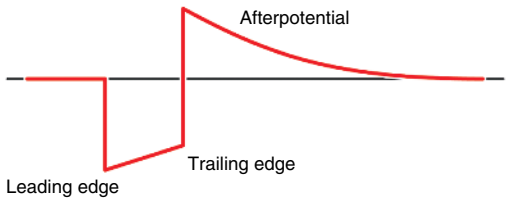


Fig. 1.9 Diagram of a pacing pulse, constant voltage, with leading edge and trailing edge voltage and an afterpotential with opposite polarity. As described in the text, afterpotentials may result in sensing abnormalities.

Sensing-specific programming

When programming the sensitivity of the device, it is important to consider the rhythm being sensed as well as the desire to marry avoidance of oversensing of nonmyocardial signals or myocardial signals beyond the chamber of interest as well as undersensing of signals within the chamber of interest. When evaluating sensing, it is of particular importance to consider the rhythm being sensed. For example, local atrial or ventricular signals may have higher amplitudes during normal rhythm than during an arrhythmia, such as atrial fibrillation or VF. Thus, appropriate sensing during periods of arrhythmia may require greater sensitivity settings. In turn, oversensing of ancillary phenomena (repolarization during ventricular sensing, ventricular signals during atrial sensing) may result in inappropriate withholding or delivery of therapies. Thus, a balance in sensitivity settings is often required.

Lead design

Traditional pacing lead components include the electrode and fixation device, the conductor, the insulation, and the connector pin (Figs 1.10 and 1.11). However, novel device and lead designs, allowing for leadless pacemakers and other novel approaches to cardiac stimulation, afford different form factors that, nevertheless, rely on similar principles to traditional leads.

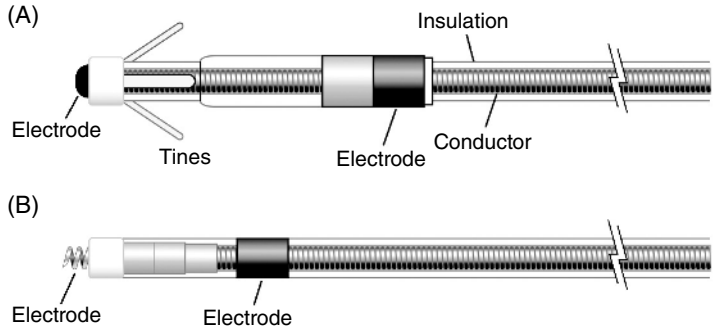


Fig. 1.10 (A) Basic components of a passive fixation pacing lead with tines. (B) Active fixation lead in which the helix serves as the distal electrode.

Leads are subject to biologic, chemical, and mechanical repetitive stress. They must be constructed of materials that provide mechanical longevity, stability, and flexibility; they must satisfy electrical conductive and resistive requirements; they must be insulated with material that is durable and that has a low friction coefficient, to facilitate implantation; and they must include an electrode that provides good mechanical and electrical contact with the myocardium. Industry continues to improve lead design to achieve these goals.

Optimal stimulation and sensing thresholds favor an electrode with a small radius and a large surface area. Electrode shape and surface composition have evolved over time. Early models utilized a round, spherical shape with a smooth metal surface. Electrodes with an irregular, textured surface allow for increased surface area without an increase in electrode radius.[15,33,35] To achieve increased electrode surface area, manufacturers have used a variety of designs, including microscopic pores, coatings of microspheres, and wire filament mesh.

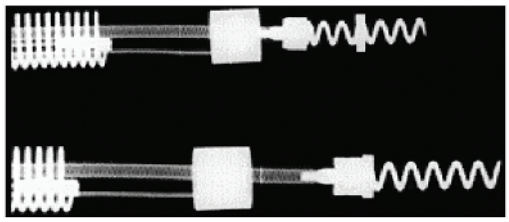


Fig. 1.11 Radiographic example of an active fixation screw-in lead with a retractable screw rather than a screw that is always extended. The screw is extended in the lower image.

Unfortunately, relatively few conductive materials have proven to be satisfactory for use in pacing electrodes. Ideally, electrodes are biologically inert, resist degradation over time, and do not elicit a marked tissue reaction at the myocardium–electrode interface. Certain metals, such as zinc, copper, mercury, nickel, lead, and silver, are associated with toxic reactions with the myocardium. Stainless steel alloys are susceptible to corrosion. Titanium, tantalum, platinum, and iridium acquire a surface coating of oxides that impedes current transfer. Materials currently in use are platinum–iridium, platinized titanium-coated platinum, iridium oxide, and platinum (Fig. 1.12). Carbon electrodes seem to be least susceptible to corrosion. Also, they are improved by activation, which roughens the surface to increase the surface area and allow for tissue ingrowth.[36]

Lead fixation with traditional leads may be active or passive. Passive fixation endocardial leads usually incorporate tines at the tip that become ensnared in

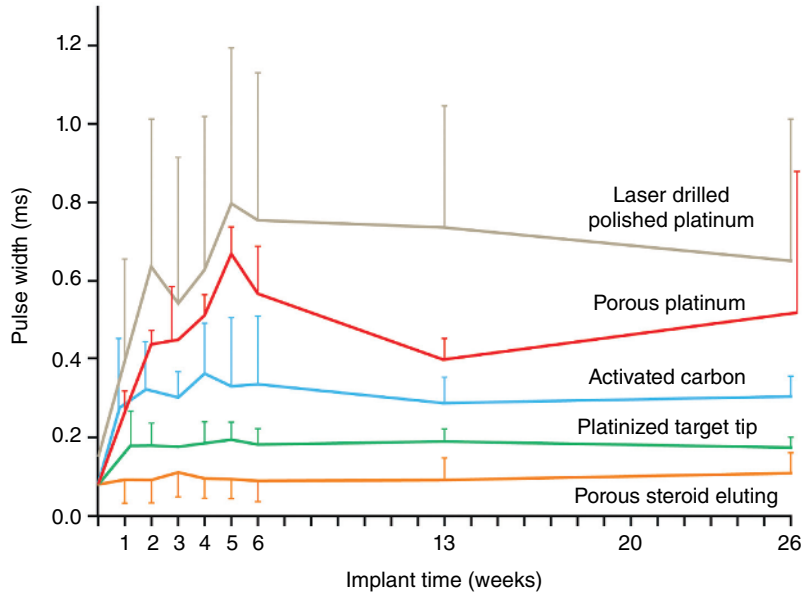


Fig. 1.12 Capture thresholds from implantation to 26 weeks from a variety of unipolar leads with similar geometric surface area electrodes. From top to bottom, the curves represent laser drilled polished platinum; porous surface platinum; activated carbon; platinized target tip; and porous steroid eluting leads. (Source: From Stokes KB, Kay GN. Artificial electric cardiac stimulation. In: Ellenbogen KA, Kay GN, Wilkoff BL, eds. Clinical Cardiac Pacing. Philadelphia: WB Saunders Co., 1995: 3–37, by permission of the publisher.)

trabeculated tissue in the right atrium or ventricle, providing lead stability. Leads designed for coronary venous placement usually incorporate a design that wedges the lead against the wall of the coronary vein. Active fixation leads deploy an electrically active screw into the myocardium to provide lead stability. There are advantages and disadvantages to active and passive designs. Passive fixation leads are simple to deploy. However, considerable myocardial and fibrous tissue enveloping the tip typically develops with passive fixation leads. The encasement of the tines of a passive fixation lead by fibrous tissue often makes the extraction of passive fixation leads more difficult than that of active fixation leads. Active fixation leads are often preferable in patients with distorted anatomy, such as those with congenital cardiac defects or those with surgically amputated atrial appendages. Active fixation leads are also preferable in patients with high right-sided pressures. As alternative site pacing has evolved (i.e., the placements of leads outside the right atrial appendage and RV apex), screw-in leads have become more popular because of the ability to stabilize them mechanically in nontraditional locations.

In active fixation leads, various mechanism are used to keep the screw unexposed (to avoid tissue injury) until it is in position for fixation. In many leads, the helix is extendable and retractable by rotation of the proximal connector using a simple tool (Bisping screwdriver). This allows the operator to control the precise time and location of helix deployment. Another approach entails covering a fixed helix with a material such as mannitol which dissolves in the bloodstream after approximately 5 min. This permits placement of the lead atraumatically at the desired location. Fixation is accomplished by rotating the entire lead body.

Conductors are commonly of a multifilament design to provide tensile strength and reduce resistance to metal fatigue (Fig. 1.13). Alloys such as MP35N (cobalt, nickel, chromium, and molybdenum) and nickel silver are typically used in modern pacing leads. Bipolar leads may be of coaxial design, with an inner coil extending to the distal electrode and an outer coil terminating at the proximal electrode (Fig. 1.14). This design requires that the conductor coils be separated by a layer of inner insulation. Coaxial designs remain commonly used in the treatment of bradyarrhythmias. Some bipolar leads are coradial, or "parallel-wound"; that is, two insulated coils are wound next to each other. Leads may also be constructed with the conductor coils parallel to each other (multiluminal), again separated by insulating material (Fig. 1.14). This type of design is typically used for tachyarrhythmia leads. Additionally, leads may use a combination of coils and cables. The coil facilitates the passage of a stylet for lead implantation, and the cable allows a smaller lead body.

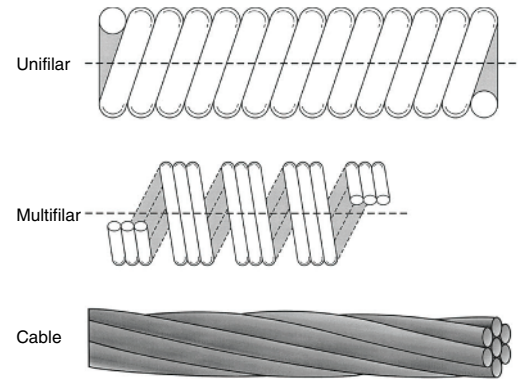


Fig. 1.13 Conductor coils may be of unifilar, multifilar, or cable design. The multifilar and cable designs allow the conductor to be more flexible and more resistant to fracture.

Two materials have predominated in lead insulation: silicone and polyurethane. Each has its respective advantages and disadvantages, but the overall performances of both materials have been excellent.[37]

The two grades of polyurethane that have had the widest use are Pellathane 80A (P80A) and Pellathane 55D. Early after the introduction of polyurethane as an insulating material, it became clear that clinical failure rates with specific leads were higher than acceptable; further investigation revealed that the failures were occurring primarily in leads insulated with the P80A polymer.[35,38] Microscopic cracks developed in the P80A polymer, initially occurring as the heated polymer cooled during manufacture; with additional environmental stress, these cracks propagated deeper into the insulation, resulting in failure of the lead insulation.

Polyurethane may also undergo oxidative stress in contact with conductors containing cobalt and silver chloride, resulting in degradation of the lead from the inside and subsequent lead failure. Some current leads use silicone with a polyurethane coating, incorporating the strength and durability of silicone with the ease of handling of polyurethane while maintaining a satisfactory external lead diameter. Silicone rubber is well known to be susceptible to abrasion wear, cold flow due to cyclic compression, and wear from lead-to-lead and lead-to-can contact. Current silicone leads have surface modifications that improve lubricity and reduce friction in blood. Preliminary studies have suggested that a hybrid coating of silicone and polyurethane may offer improved wear.[39] Despite lead improvements, laboratory testing, and premarketing, clinical trials have been inadequate to predict the long-term performance of

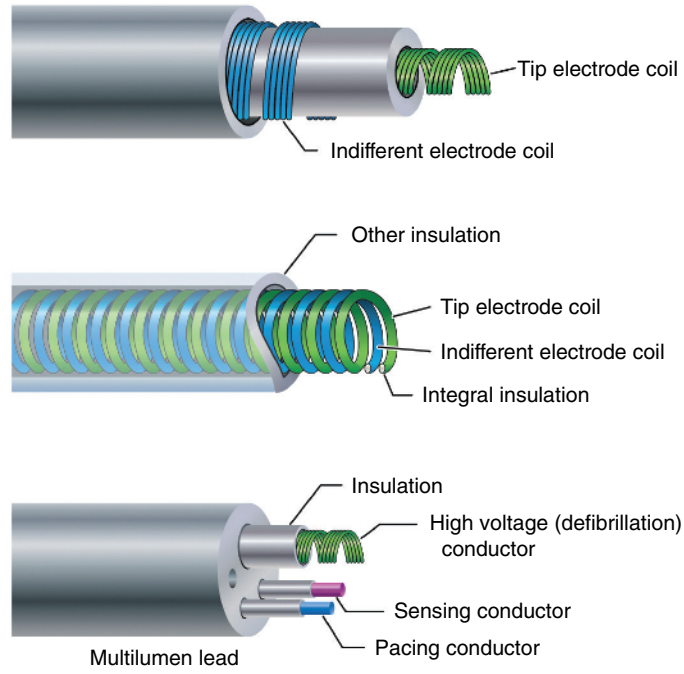


Fig. 1.14 Varieties of conductor construction. Top: bipolar coaxial design with an inner multifilar coil surrounded by insulation (inner), an outer multifilar coil, and outer insulation. Middle: individually insulated wires wound together in a single multifilar coil for bipolar pacing. Bottom: multilumen lead body design in which each conductor has its own lumen.

leads, so that clinicians implanting the devices or performing follow-up in patients with pacing systems must vigilantly monitor lead status. Increasingly, the use of internet-enabled remote monitoring and pulse-generator-based algorithms permits automatic alert generation in the event of impending lead fracture.

Contemporary leads and connectors are standardized to conform to international guidelines (IS-1 standard), which mandate that leads have a 3.2 mm diameter in-line bipolar connector pin.[40] These standards were established many years ago because some leads and connector blocks were incompatible, requiring the development of multiple adaptors. The use of the IS-1 standard permits using one manufacturer's leads with another manufacturer's pulse generator. Similarly, the DF-1 standard insures a common site for high-voltage connections in defibrillators. The newer IS-4 standard permits a single in-line connection of four low-voltage electrodes, permitting coronary sinus leads to include four (rather than two) pacing sites, increasing the likelihood of a lead having an acceptable threshold and/or pacing site. The DF-4 connectors (Fig. 1.15A) contain two high-voltage and two low-voltage connections so that a single connector (with single screw) can provide pace-sense and dual coil defibrillation support, significantly decreasing pocket bulk. The limitation introduced by the DF-4 connector is the inability to use a separate lead and connect it to the proximal coil port in the header. Though not commonly required, this is useful when the defibrillation threshold (DFT) is high and a strategy of placing a defibrillation coil in the coronary sinus, azygous vein, or subcutaneous tissues is planned.

Bipolar and unipolar pacing and sensing

In unipolar pacing systems, the lead tip functions as the cathode and the pulse generator as the anode (unipolar vs. bipolar leads; Fig. 1.15B). In bipolar systems, the lead tip serves as the cathode and a lead ring acts as the anode (Fig. 1.15B). Unipolar leads are of simpler design (only one conductor) and have a smaller external diameter. Unipolar leads have historically demonstrated greater durability than bipolar leads. In recent years, the difference in durability has been less distinct. Unipolar leads do not offer the option of bipolar function. Although unipolar and bipolar leads are readily available, present usage of transvenous leads is almost exclusively bipolar. Bipolar leads may function in the unipolar mode if the pacemaker is so programmed. They are available in several designs, generally coaxial or multiluminal. Regardless of design, the external diameter of a bipolar lead is usually greater than that of unipolar leads because each coil must be electrically separated by insulating material. Bipolar pacing is generally preferred over unipolar pacing because it rarely causes extracardiac stimulation at the pulse generator (pectoralis muscle stimulation), which may more often

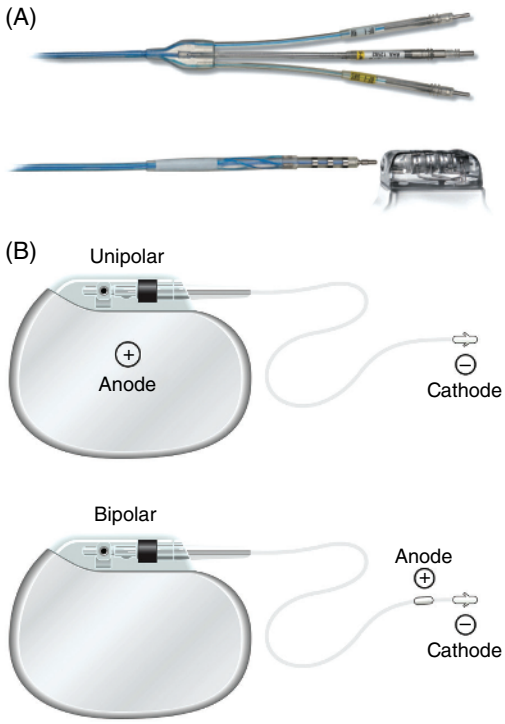


Fig. 1.15 Lead connectors and configurations. (A) Connector types in defibrillator leads. The top image shows the proximal end of a defibrillation lead with three connectors. Top and bottom pins are DF-1 connectors used for high-voltage shock delivery for defibrillation, whereas the middle pin is an IS-1 connector used for pacing and sensing. Bottom image shows a DF-4 connector, in which all four conductors (two for defibrillation and two for pace/sense) are mounted on a single pin. (B) Unipolar versus bipolar leads pacing leads. In a unipolar configuration, the pacemaker case serves as the anode, or (+), and the electrode lead tip as the cathode, or (–). In a bipolar configuration, the anode is located on the ring, often referred to as the "ring electrode," proximal to the tip, or cathode. The distance between tip and ring electrode varies among manufacturers and models.

occur with unipolar pacing due to current returning to the generator. Also, because closely spaced electrodes result in a smaller "antenna," bipolar sensing is less susceptible to myopotential and far-field oversensing and to electromagnetic interference.[41] All implantable defibrillators utilize bipolar sensing to minimize the risk of inappropriate shock caused by oversensing.

There are historical controversies regarding unipolar versus bipolar pacing and sensing configurations and which, if either, is superior.[41] Nonetheless, the majority of leads implanted are bipolar. There are certain advantages with unipolar leads. They employ a simpler design and smaller size. Smaller, more compliant and flexible unipolar leads can be placed in difficult coronary sinus venous tributaries. Traditionally, they have very low failure rates.[42] Unipolar leads are less prone to short circuit when there are insulation breaches (due the absence of an adjacent conductor), although this benefit may be outweighed by their susceptibility to oversensing. Importantly, a lead that is malfunctioning in the bipolar mode may function satisfactorily when programmed to the unipolar configuration – see Chapter 8 (Programming: Maximizing Benefit and Minimizing Morbidity Programming).

All pulse generators offer independently programmable pacing and sensing in each channel; however, bipolar programming of a device attached to a unipolar lead results in no output. Bipolar leads can function in the unipolar mode; the converse is not true.

Left ventricular and His bundle pacing leads

Cardiac resynchronization therapy with biventricular pacing is an established treatment for patients with chronic moderate–severe congestive heart failure, low left ventricular ejection fraction, and New York Heart Association class III or IV heart failure.[43] In order to pace the left ventricle, a pacing lead is implanted transvenously through the coronary sinus and one of its venous tributaries to stimulate the left ventricular free wall. Resynchronization is obtained by stimulating both ventricles to contract with minimal intraventricular delay, thereby improving the left ventricular performance.[44]

New technologies have emerged to assist in the placement of leads to targeted anatomic sites. Catheter-delivered systems use a deflectable sheath that is braided to allow the simultaneous ability to torque and advance the catheter. A second, smaller lumen sheath can be used within the first sheath to enhance access to the coronary sinus and its venous tributaries, as well as serve as a conduit for contrast injections and lead delivery. A second technology developed to reach difficult anatomic targets is to use an over-the-wire lead delivery system (Fig. 1.16). With this system, the lead can be advanced to a stable position over a guidewire used initially to navigate tortuous regions of the coronary veins similar to techniques used extensively for coronary angiography. Using stiffer wires, like a stylet that does not exit the left ventricular lead, the leads can be pushed into position as well as have their relative geometries changed by the constraints of the stiff wire. Tip geometry changes allow the operator to change the early contour of the lead system dynamically to allow passage through tortuous veins. Flexibility in tool selection improves access to target sites across a broad range of anatomies and decreases injury to coronary venous structures. Through availability and/or combining of these multiple technologies, access to target sites has

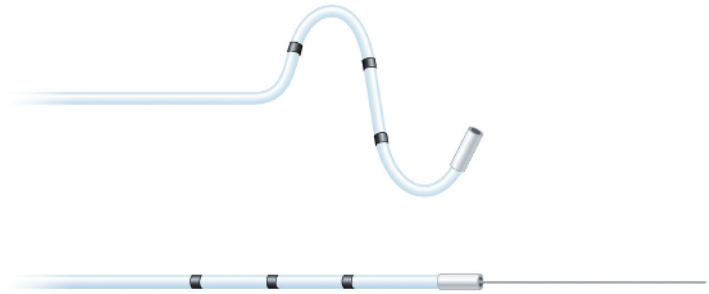


Fig. 1.16 Over-the-wire leads to facilitate placement in coronary vein branches. Top: lead with wire advanced beyond the distal end. The wire acts as a track over which the lead is advanced to provide stability. Bottom: lead with wire removed for final deployment.

improved greatly, particularly coronary vein subselection for left ventricular lead placement.

Modifications of tip geometries as well as a family of left ventricular leads to choose from have improved the stability of these passive leads. Furthermore, newer multipolar left ventricular leads provide a broad array of pacing configurations to facilitate favorable pacing thresholds and avoid phrenic nerve stimulation.[45] Establishing a well-positioned left ventricular lead position, and avoiding apical pacing, favorably influence long-term outcomes with cardiac resynchronization.[46]

Another novel approach to using traditional leads for resynchronization involves utilizing the native His–Purkinje system to facilitate rapid activation of the ventricles synchronously. So called His bundle pacing theoretically allows for more physiologic ventricular activation; however, this approach may be limited by anatomic complexity, variability in degree of disease of the native conduction system, and lead stability in the region of interest. Unlike conventional active fixation leads, leads used for His bundle pacing rely on a fixed helix screw that consists of a smaller electrode to facilitate selective His bundle capture and minimize local nonselective myocardial activation. In addition, deployment typically relies on use of either a preformed, nonsteerable sheath with dual plane shaping or a steerable sheath, which optimizes the ability to direct the lead to the septal region. Currently, the lead used most often is a nonstylet-driven exposed 4.1 French helical screw.

Epicardial leads

Though most commonly used leads consist of endocardially placed leads, epicardially placed leads are not uncommon, particularly in cases of leads placed in the operating room, and particularly in cases of difficult endocardial left ventricular lead placement or congenital heart disease in which endocardial pacing is not desired. These leads may involve a variety of fixation mechanisms distinct from that of endocardial leads, including epicardial stab-in, myocardial screw-in, or suture-on leads. Though steroid-eluting leads are standard for endocardial placement, there is no clear effect on longevity for steroid- versus nonsteroid-eluting epicardial leads. Similar to endocardial leads, both unipolar and bipolar configurations are possible, with the attendant benefits and risks seen with endocardial pacing also seen with epicardial leads.

Defibrillator leads – special considerations

As described previously, most defibrillator leads consist of similar design to that of endocardial pacing systems, albeit with addition of a coil to facilitate delivery of a high-voltage shock to restore normal rhythm. Novel defibrillators consist of entirely subcutaneous leads with longer shock coils that do not rely on direct myocardial contact or fixation. These leads consist of a distal sensing electrode, an integrated anchoring hole, an 8 cm defibrillator coil, a proximal sensing electrode located just proximal to the coil, and a connector pin. Electrodes are polyurethane, and the suture sleeve is silicone. Subcutaneous lead form factors are currently evolving, with future designs being constructed to facilitate pacing as well as defibrillation and to optimize form factor for delivery into the substernal space.

Leadless pacemakers

Though most of the discussion has focused on lead design related to traditional leads, advances in battery technology, component design, communication technologies, and delivery tools has allowed for the development of entirely self-contained leadless pacemakers. The principles outlined previously in terms of surface area of exposure to the myocardial interface and role of steroid-eluting systems are applicable to leadless devices as well. Currently, leadless devices only exist for RV pacing and consist of fully self-contained units. Either lithium carbon monofluoride or lithium–silver–vanadium oxide/carbon monofluoride battery systems are used. Both active fixation and passive fixation systems are used, depending on the manufacturer. Similarly, deployment and retrieval mechanisms may vary based on the manufacturer.

Pulse generators

All pulse generators include a power source, an output circuit, a sensing circuit, a timing circuit, and a header with a standardized connector (or connectors) to attach a lead (or leads) to the pulse generator.[47] The exception to this is leadless pacemakers, which are entirely self-contained, generally displacing about 1 cm^3 in blood volume, and residing in the right ventricle (though multichamber leadless devices are in development). However, all devices are capable of storing some degree of diagnostic information that can be retrieved at a later time. Most pacemakers also incorporate some type of a rate-adaptive sensor. The variety of novel sensors balanced against miniaturizing battery technology has led to a variable effect on the size and potential longevity of pulse generators.

Many power sources have been used for pulse generators over the years. Lithium iodine cells (consisting of varying types) have been the energy source for almost all contemporary pacemaker pulse generators. Newer pacemakers and implantable cardioverter-defibrillators (ICDs) that can support higher current drains for capacitor charging and high-rate antitachycardia pacing (ATP) use lithium–silver oxide–vanadium chemistries. Lithium is the anodal element and provides the supply of electrons; iodine is the cathodal element and accepts the electrons. The cathodal and anodal elements are separated by an electrolyte, which serves as a conductor of ionic movement but a barrier to the transfer of electrons. The circuit is completed by the external load (i.e., the leads and myocardium). The battery voltage of the cell depends on the chemical composition of the cell; at the beginning of life for the lithium iodine battery, the cell generates approximately 2.8 V, which decreases to 2.4 V when approximately 90% of the battery life has been used. The voltage then exponentially declines to 1.8 V as the battery reaches end-of-life. However, the voltage at which the cell reaches a specific degree of discharge is load dependent. The elective replacement voltages were chosen based on the shape of the discharge curves under expected operating conditions. When the battery is at end-of-service, most devices lose telemetry and programming capabilities, frequently reverting to a fixed high-output pacing mode to maintain patient safety. This predictable depletion characteristic has made lithium-based power cells common in current devices. Nickel–cadmium technology, as well as other metal-based systems, are being considered again, however.

The battery voltage can be measured by communication with the pulse generator. In addition, most devices provide battery impedance (which increases with battery depletion) for additional information about battery life. The battery life can also be estimated by the magnet rate of the device, which changes with a decline in battery voltage. Unfortunately, the magnet rates are not standardized, and rate change characteristics vary tremendously among manufacturers and even among devices produced by the same manufacturer. Therefore, it is important to know the magnet rate characteristics of a given device before using this feature to determine battery status.

The longevity of any battery is determined by several factors, including chemical composition of the battery, size of the battery, external load (pulse duration and amplitude, stimulation frequency, total pacing lead impedance, and amount of current required to operate device circuitry and store diagnostic information), amount of internal discharge, and voltage decay characteristics of the cell. The basic formula for longevity determination is

$$114 \times \frac{\text{Battery capacity}\,(\text{A h})}{\text{Current drain}\,(\mu\text{A})} = \text{Longevity}\,(\text{years})$$

However, this formula is subject to how the power cell's ampere-hours is specified by the manufacturer; thus, the longevity will vary somewhat by company. High-performance leads, automatic capture algorithms, and programming options that minimize pacing may further enhance device longevity if not offset by energy consumption from running the software.[48,49]

The pacing pulse is generated first by charging an output capacitor with subsequent discharge of the capacitor to the pacing cathode and anode. Because the voltage of a lithium iodine cell is fixed, obtaining multiple selectable pulse amplitudes requires the use of a voltage amplifier between the battery and the output capacitor. Contemporary pulse generators are constant-voltage (rather than constant-current) devices, implying delivery of a constant-voltage pulse throughout the pulse duration. In reality, some voltage drop occurs between the leading and the trailing edges of the impulse; the size of this decrease depends on the pacing impedance and pulse duration. The lower the impedance, the greater the current flow from the fixed quantity of charge on the capacitor and the greater the voltage drop throughout the pulse duration.[50] The voltage drop is also dependent on the capacitance value of the capacitor and the pulse duration.

The output waveform is followed by a low-amplitude wave of opposite polarity: the afterpotential. The afterpotential is determined by the polarization of the electrode at the electrode–tissue interface; formation is due to electrode characteristics, as well as to pulse amplitude and duration. The sensing circuit may sense afterpotentials of sufficient amplitude, especially if the sensitivity threshold is low. Newer pacemakers

use the output circuit to discharge the afterpotential quickly, thus lowering the incidence of afterpotential sensing. The afterpotential also helps to prevent electrode corrosion.

The intracardiac electrogram results from current conducted from the myocardium to the sensing circuit via the pacing leads, where it is then amplified and filtered. The input impedance must be significantly larger than the sensing impedance to minimize attenuation of the electrogram. A bandpass filter attenuates signals on either side of a center frequency, which varies between manufacturers (generally ranging from 20 to 40 Hz).[51,52] After filtering, the electrogram signal is compared with a reference voltage: the sensitivity setting; signals with an amplitude of this reference voltage or higher are sensed as true intracardiac events and are forwarded to the timing circuitry, whereas signals with an amplitude below the reference amplitude are categorized as noise, extracardiac or other cardiac signal, such as T waves.

Sensing circuitry also incorporates noise reversion that causes the pacemaker to revert to a noise reversion mode (asynchronous pacing) whenever the rate of signal received by the sensing circuit exceeds the noise reversion rate. This feature is incorporated to prevent inhibition of pacing when the device is exposed to electromagnetic interference. Pulse generators also use Zener diodes designed to protect the circuitry from high external voltages, which may occur, for example, with defibrillation. When the input voltage presented to the pacemaker exceeds the Zener voltage, the excess voltage is shunted back through the leads to the myocardium.

The timing circuit of the pacemaker is an electronic clock that regulates the pacing cycle length, refractory periods, blanking periods, and AV intervals with extreme accuracy. The output from the clock (as well as signals from the sensing circuitry) is sent to a timing and logic control board that operates the internal clocks, which in turn regulate all the various timing cycles of the pulse generator. The timing and logic control circuitry also contains an absolute maximal upper rate cut-off to prevent "runaway pacing" in the event of random component failure.[53,54]

Each new generation of pacemakers contains more microprocessor capability. The circuitry contains a combination of read-only memory (ROM) and random-access memory (RAM). ROM is used to operate the sensing and output functions of the device, and RAM is used in diagnostic functions. Larger RAM capability has allowed devices to store increased amounts of retrievable diagnostic information and patient-specific longitudinal data, with the potential to allow downloading of new features externally into an implanted device.

External telemetry is supported in all implantable devices and in some pacemakers. The pulse generator can receive information from the programmer and send information back by radiofrequency signals. Each manufacturer's programmer and pulse generator operate on an exclusive radiofrequency, preventing the use of one manufacturer's programmer with a pacemaker from another manufacturer. Through telemetry, the programmer can retrieve both diagnostic information and real-time information about battery status, lead impedance, current, pulse amplitude, and pulse duration. Real-time electrograms and marker channels can also be obtained with most devices. The device can also be directed to operate within certain limits and to store specific types of diagnostic information via the programmer.

The most recent change in telemetry is that of "remote" capability. Information exchange has traditionally occurred by placing and leaving the programming "head" of the programmer over the pulse generator for the duration of the interrogation and programming changes. New telemetry designs allow the programming "head" or "wand" to be placed briefly over the pulse generator, or in the near vicinity of the device, to establish the identity of the specific model and pulse generator and then complete the bidirectional informational exchange at a distance; that is, the "wand" does not need to be kept in a position directly over the pulse generator. Finally, even the use of a wand for certain pulse generators is not required for remote programming. These technology advances have allowed remote monitoring of all implantable devices and, in some pacemakers, the use of home telemetry systems that upload patient- and device-specific data to a central, secure database. With home monitoring, devices can be routinely monitored, patient alerts transmitted in real time, and patient cardiac status updates communicated on a programmable criteria basis. Remote monitoring of patients with ICDs improves survival and readily identifies risk markers of mortality.[55]

Pacemaker nomenclature

A lettered code to describe the basic function of pacing devices, initially developed by the American Heart Association and the American College of Cardiology, has since been modified and updated by the members of the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group (currently the Heart Rhythm Society).[56] This code has five positions to describe basic pacemaker function, although it obviously cannot incorporate all of the various special features available on modern devices (Table 1.1).

Table 1.1 The North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group (NBG) code.

I	II	III	IV	V
Chamber(s) paced	**Chamber(s) sensed**	**Response to sensing**	**Programmability, rate modulation**	**Multisite pacing**
O = None	O =None	O = None	O = None	O = None
A = Atrium	A = Atrium	T = Triggered	P = Simple programmable	A = Atrium
V = Ventricle	V = Ventricle	I = Inhibited	M = Multiprogrammable	V = Ventricle
D = Dual (A + V)	D = Dual (A + V)	D = Dual (T + I)	C = Communicating	D = Dual (A + V)

Source: Modified from Bernstein AD, Daubert JC, Fletcher RD, *et al.* The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group. Pacing Clin Electrophysiol 2002; 25:260–4, by permission of Futura Publishing Company.

The first position describes the chamber or chambers in which electrical stimulation occurs. **A** reflects pacing in the atrium, **V** implies pacing in the ventricle, **D** signifies pacing in both the atrium and the ventricle, and **O** is used when the device has ATP or cardioversion-defibrillation capability but no bradycardia pacing capability.

The second position describes the chamber or chambers in which sensing occurs. The letter code is the same as that in the first position, except that an **O** in this position represents lack of sensing in any chamber; that is, fixed-rate pacing. (Manufacturers may use an **S** in both the first and the second positions to indicate single-chamber capability that can be used in either the atrium or the ventricle.)

The third position designates the mode of sensing; that is, how the device responds to a sensed event. **I** indicates that the device inhibits output when an intrinsic event is sensed and starts a new timing interval. **T** implies that an output pulse is triggered in response to a sensed event. **D** indicates that the device is capable of dual modes of response (applicable only in dual-chamber systems).

The fourth position reflects both programmability and rate modulation. **O** indicates that none of the pacemaker settings can be changed by noninvasive programming, **P** suggests "simple" programmability (i.e., one or two variables can be modified), **M** indicates multiprogrammability (three or more variables can be modified), and **C** indicates that the device has telemetry capability and can communicate noninvasively with the programmer (which also implies multiprogrammability). Finally, an **R** in the fourth position designates rate-responsive capability. This means that the pacemaker has some type of sensor to modulate the heart rate independent of the intrinsic heart rate. All modern devices are multiprogrammable and have telemetry capability; therefore, the **R** to designate rate-responsive capability is the most commonly used currently.

The fifth position was originally used to identify antitachycardia treatment functions. However, this has been changed, and antitachycardia options are no longer included in the nomenclature. The fifth position now indicates whether multisite pacing is not present (**O**), or present in the atrium (**A**), ventricle (**V**), or both (**D**). Multisite pacing is defined for this purpose as stimulation sites in both atria, both ventricles, more than one stimulation site in any single chamber, or any combination of these.

All pacemaker functions (whether single, dual, or multi-chamber) are based on timing cycles. Even the function of the most complex devices can be readily understood by applying the principles of pacemaker timing intervals. This understanding is critical for accurate interpretation of pacemaker ECGs, especially during troubleshooting. Pacemaker timing cycles are described in detail in Chapter 7 (Timing Cycles).

Essentials of defibrillation

In 1899, Prevost and Battelli[57] noted that the "fibrillatory tremulations produced in the dog" could be arrested with the reestablishment of the normal heartbeat if one submitted the animal "to passages of current of high voltage." With the development of internal defibrillators in the late 1970s came a greater need to quantify defibrillation effectiveness, to understand the factors governing waveform and lead design, and to determine the effect of pharmacologic agents on defibrillation.

A few hypotheses have been proposed to explain how an electric shock terminates fibrillation: critical mass, upper limit of vulnerability (ULV), progressive depolarization, and virtual electrode depolarization. These hypotheses, which are not entirely mutually exclusive, are summarized in the following subsections.

In its resting state, the myocardium is excitable, and a pacing stimulus, or current injected by the depolarization of a neighboring myocyte, can bring the membrane

potential to a threshold value, above which a new action potential ensues. The ability of the action potential of a myocyte to depolarize adjacent myocardium results in propagation of electrical activity through cardiac tissue. Importantly, immediately after depolarization, the myocardium is refractory and cannot be stimulated to produce another action potential until it has recovered excitability. The interval immediately after an action potential, during which another action potential cannot be elicited by a pacing stimulus, is referred to as the "refractory period."

VF results when an electrical wavebreak induces reentry and results in a cascade of new wavebreaks. In patients with a structurally abnormal or diseased heart, the underlying tissue heterogeneity results in a predisposition to wavebreak, then reentry, and finally fibrillation.[58] These wandering wavelets are self-sustaining once initiated. In the 1940s, Gurvich and Yuniev[59] predicted that electric shocks led to premature tissue stimulation in advance of propagating wave fronts, preventing continued progression of the wave front. This concept of defibrillation as a large-scale stimulation remains a central tenet of many of the currently held theories of defibrillation.

Critical mass

The critical mass theory proposed that shocks need only eliminate fibrillatory wavelets in a critical amount of myocardium to extinguish the arrhythmia. Experiments in canine models found that injection of potassium chloride (which depolarizes myocardium, rendering it unavailable for fibrillation) into the right coronary artery or the left circumflex artery failed to terminate VF as often as injection into both the left circumflex and the left anterior descending arteries together. Similarly, electrical shocks of equal magnitude terminated fibrillation most frequently when the electrodes were positioned at the RV apex and the posterior left ventricle, as opposed to two RV electrodes. Thus, it was concluded that if a "critical mass" of myocardium was rendered unavailable for VF, either by potassium injection or by defibrillatory shock, then the remaining excitable tissue was insufficient to support the wandering wavelets, and the arrhythmia terminated.[60] However, it was not critical to depolarize every ventricular cell to terminate fibrillation. This is an important consideration when implanting a defibrillator, as the overall quantity of heart muscle contained within the region through which a defibrillatory shock travels is critical to the success of a shock to successfully restore normal rhythm. Thus, placement of the lead or epicardial patches relative to the generator in such a position as to encompass the largest volume of myocardium is one means by which to optimize defibrillatory success.

Upper limit of vulnerability

Studies mapping electrical activation after failed shocks led to several observations not accounted for by the critical mass hypothesis, giving rise to the ULV theory. First, an isoelectric interval (an electrical pause) was seen after failed shocks before resumption of fibrillation. The relatively long pause suggested that VF was terminated by the shock and then secondarily regenerated by it.[61] The concept that failed shocks are unsuccessful because they reinitiate fibrillation rather than because they fail to halt continuing wavelets was further buttressed by a second observation: that post-shock conduction patterns were not the continuation of preshock wave fronts.[62] If a failed shock resulted from the inability to halt continuing fibrillation, the assumption was that the post-shock wave fronts should be a continuation of the propagating wave fronts present before shock delivery and that new wave fronts at sites remote from the preshock wave fronts would not be expected. Furthermore, VF was frequently reinitiated in the regions of lowest shock intensity, suggesting that these low-intensity regions were responsible for reinitiating fibrillation. Shocks that fall into the vulnerable period (which overlaps the T wave during normal rhythm) with an energy above the lower limit of vulnerability and below the ULV induce VF. Shocks with energies above the ULV never induce VF, and thus defibrillate (Fig. 1.17).

Elegant mapping studies demonstrated that shocks with potential gradients less than a minimum critical value (termed the ULV; 6 V/cm for monophasic shocks, 4 V/cm for biphasic shocks) could induce fibrillation when applied to myocardium during its vulnerable period. Low-energy shocks did so by creating regions of functional block in vulnerable myocardium at "critical points" that initiated reentry and subsequent fibrillation.[63] Importantly, this theory permits linking of defibrillation and fibrillation. In sinus rhythm, low-energy shocks delivered during the vulnerable period (the T wave) induce VF; higher energy shocks – with energy above the ULV – do not (Fig. 1.18). Because at any given time during fibrillation a number of myocardial regions are repolarizing and thus vulnerable, a shock with a potential gradient below the ULV may create a critical point and reinitiate fibrillation. Conversely, a shock with a gradient above the ULV across the entire myocardium does not reinduce VF and should, therefore, succeed. During defibrillator testing, shocks are intentionally delivered in the vulnerable zone to induce fibrillation (Fig. 1.18); the zone of vulnerability has been defined in humans.[64] The fact that the vulnerable zone exists and that the ULV has been correlated with the DFT supports the ULV hypothesis as a mechanism of defibrillation, and permits its use at implant testing.[65]

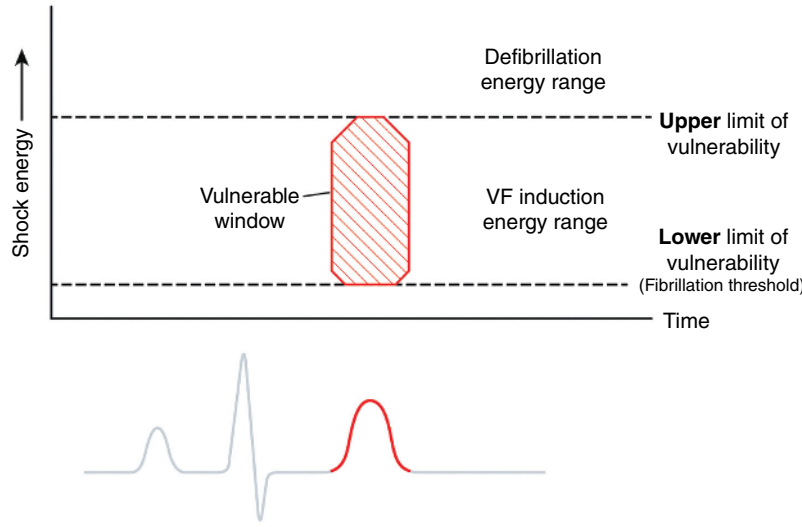


Fig. 1.17 Window of vulnerability during sinus rhythm. During sinus rhythm, the ventricles are vulnerable to ventricular fibrillation (VF) when a shock is delivered on the T wave, in the vulnerable window. To induce fibrillation, the shock energy must be greater than the lower limit of vulnerability (the fibrillation threshold) and below the upper limit of vulnerability (ULV). Shocks with energy above the upper limit of vulnerability do not induce fibrillation. Because during VF there is dyssynchrony of activation, at any given instant a number of regions are repolarizing (equivalent to the T wave in sinus rhythm), so that a shock with a gradient that is less than the ULV can reinduce fibrillation in these regions. In contrast, shocks with energy above the ULV throughout the myocardium cannot reinitiate VF and are successful. The ULV is correlated with the defibrillation threshold. Further details appear in the text.

Progressive depolarization

A third theory of defibrillation, the progressive depolarization theory (also referred to as the "refractory period extension theory"), incorporates some elements of both critical mass and ULV theories. Using voltage-sensitive optical dyes, Dillon and Kwaku[66] have demonstrated that shocks of sufficient strength were able to elicit responses, even from supposedly refractory myocardium. Thus, as seen in Fig. 1.19, the duration of an action potential can be prolonged (and the refractory period extended) despite refractory myocardium when a sufficiently strong shock is applied.[67] This phenomenon may result from sodium channel reactivation by the shock. The degree of additional depolarization time is a function of both shock intensity and shock timing.[68] Because the shock stimulates new action potentials in myocardium that is late in repolarization and produces additional depolarization time when the myocardium is already depolarized, myocardial resynchronization occurs. This is manifested by myocardial repolarization at a constant time after the shock (second dashed line in Fig. 1.19, labeled "constant repolarization time"). Thus, the shock that defibrillates extends overall ventricular refractoriness, limiting the excitable tissue available for fibrillation. Thus, it extinguishes continuing wavelets and resynchronizes repolarization, so that distant regions of myocardium become excitable simultaneously, preventing dispersion of refractoriness and renewed reentry. Experimental evidence has demonstrated that shocks with a potential gradient above the ULV result in time-dependent extension of the refractory period. In contrast, lower energy shocks may result in a graded response that could create transient block and a critical point, thereby reinducing fibrillation.[68] Note, whereas progressive depolarization and virtual electrode depolarization (discussed next) address cellular mechanisms of shock induced reentry, the ULV and critical mass hypotheses do not postulate a specific mechanism, but only that shock-induced reentry is an important mechanism of failed subthreshold shocks.

Virtual electrode depolarization

More recently, optical signal measurements of transmembrane potentials have demonstrated the concept of the "virtual electrode."[69] The virtual electrode effect refers to stimulation of tissue far from the implanted electrode. This effect makes the defibrillation electrode effectively much larger than the physical electrode. In the virtual electrode, the anode cells are brought close to their resting potential, increasing their responsiveness to stimulation. More importantly, the region of depolarization or hyperpolarization near the physical electrode is surrounded by regions with opposite polarity. Anodal shocking produces a wave front of

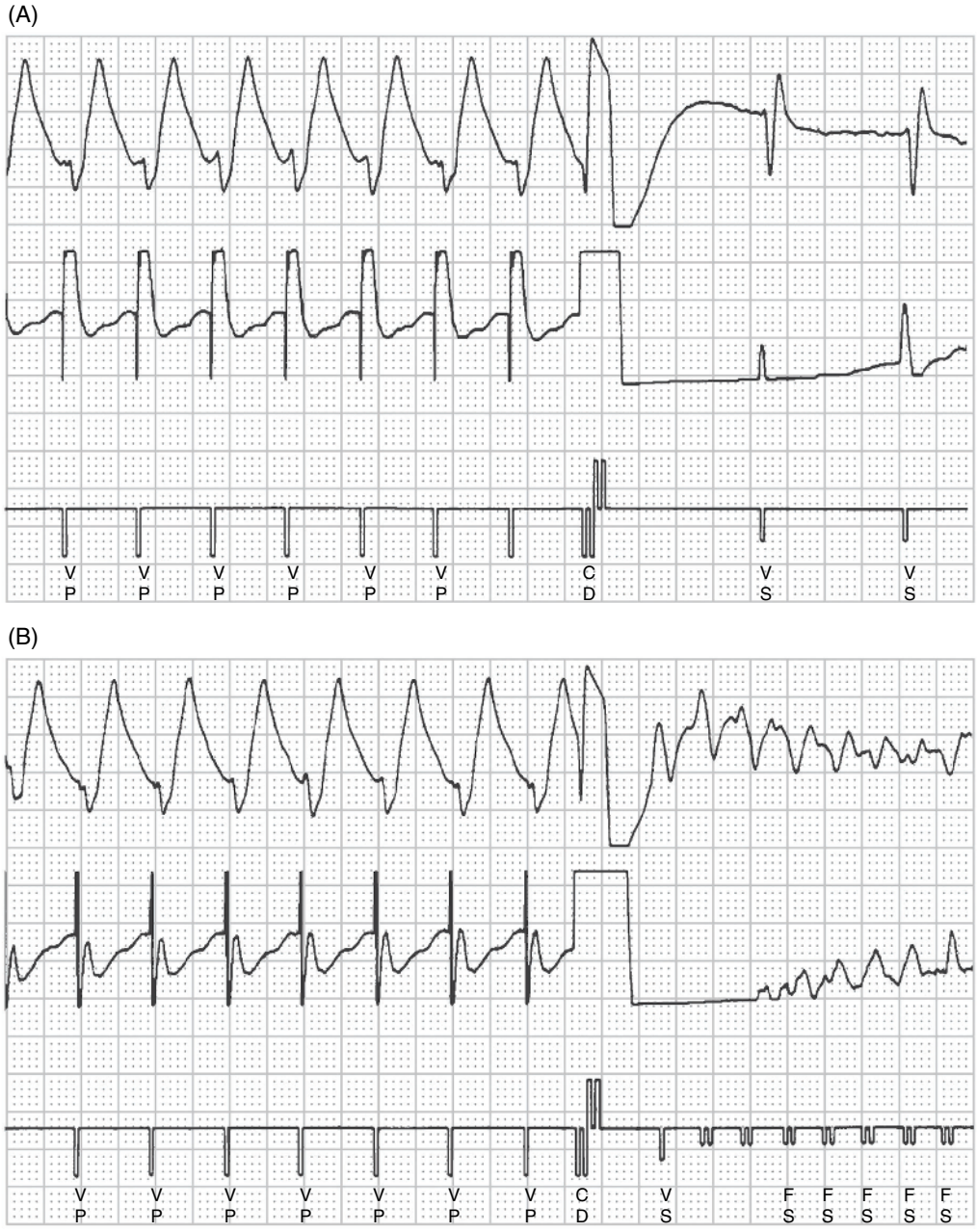


Fig. 1.18 Induction of ventricular fibrillation by a T-wave shock during testing of an implantable defibrillator. (A) A 1 J shock is delivered 380 ms after the last paced beat. Fibrillation is not induced, because this shock is delivered outside the window of vulnerability. (B) The timing of the shock is adjusted to 300 ms after the last paced complex, so that it is delivered more squarely on the T wave, in the window of vulnerability, and fibrillation is induced. The window of vulnerability is defined by both shock energy and timing. CD, charge delivered; FS, fibrillation sense; VP, ventricular pacing; VS, ventricular sensing.

depolarization that begins at the boundary of positively charged regions and then spreads toward the negatively charged region of the physical anode.[70] This produces "collapsing" wave fronts that frequently collide and neutralize one another and thereby are less likely to result in a sustained arrhythmia (Fig. 1.20).[71]

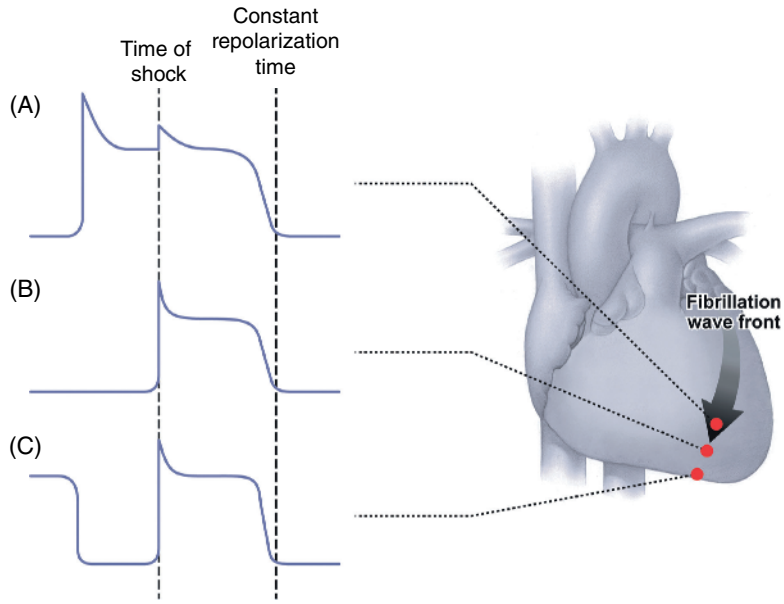


Fig. 1.19 Progressive depolarization. A fibrillatory wave front is depicted by the arrow, and the action potential response to a defibrillatory shock is demonstrated at several points surrounding the wave front. The fibrillatory wave front has just passed through a myocyte at point A when the shock is delivered. The myocyte is in its plateau (phase 2), when it would ordinarily be refractory to additional stimulation. However, when a sufficiently strong shock is delivered, the myocyte can generate an active response with prolongation of the action potential and of the refractory period. The response is referred to as "additional depolarization time." The tissue at point B is at the leading edge of the fibrillatory wave front. The shock strikes this myocardium at the time of the upstroke (phase 0) and has little effect on the action potential. The tissue at point C is excitable (it is the excitable gap that the fibrillatory wave front was about to enter) when the shock is delivered. The shock elicits a new action potential in this excitable tissue. Despite the different temporal and anatomical locations of the three action potentials depicted, after the shock there is resynchronization by the "constant repolarization time." This resynchronization helps prevent continuation of fibrillation.

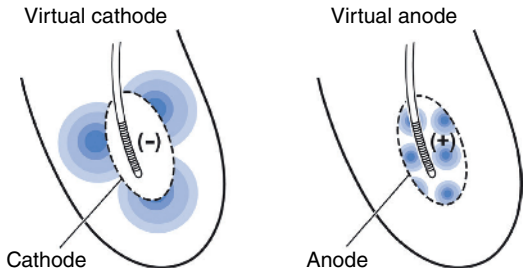
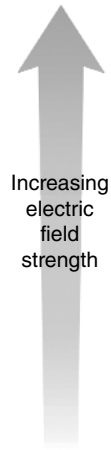


Fig. 1.20 The cathodal shocks (left) produce wave fronts that expand and propagate away from the right ventricular coil. In comparison, anodal shocks (right) produced wave fronts that collapse and propagate toward the right ventricular coil (Source: Adapted from Figure 4, Kroll MW, Efimov IR, Tchou PJ. Present understanding of shock polarity for internal defibrillation: the obvious and non-obvious clinical implications. Pacing Clin Electrophysiol 2006; 29:885–91, with permission.)

In addition to the foregoing, it is postulated that the coronary vessels may play a role in this virtual electrode effect. In a rabbit model, it was shown that defibrillation resulted in successive break excitations (after each shock phase) emanating from opposing myocardial surfaces (in the septum and left ventricle), which rapidly closed down excitable gaps. At stronger shocks, virtual electrodes formed around vessels, rapidly activating intramural tissue because of break excitations, assisting the main defibrillation mechanism. However, this was dependent on larger vessels being present (>200 μm in diameter). Thus, larger intramural blood vessels facilitate a more effective elimination of existing wave fronts, rapid closing down of excitable gaps, and successful defibrillation. This may also partly explain differential defibrillatory success in patients with more extensive intramural coronary disease.[72]

Electroporation as a mechanism for defibrillation

It is well recognized that electric fields can result in permeabilization of cellular membranes. At lower energy levels, reversible poration of cell membranes can occur such that cells continue to be viable. At higher energy levels, irreversible electroporation leading to apoptotic cell death may occur; and at very high field strengths,



Tissue Effect	Clinical Effect
Myocardial damage	Post-shock block Initiation of new arrhythmias
Refractory period extenstion Constant repolarization time	Defibrillation zone
Upper limit of vulnerability/defibrillation threshold	
Creation of critical points (transient block due to graded response)	Ventricular fibrillation induction
Action potential stimulation	Pacing pulse
No physiological effect	No effect

Fig. 1.21 Effects of increasing shock (electrical field) strength on myocardial tissue.

necrotic cell death may occur. It has been shown that, with a defibrillatory shock, there was transient depolarization, reduction of action potential amplitude, and upstroke dV/dt. Furthermore, epicardially, electroporation was localized to the region around the electrode, whereas endocardially an extensive electroporative effect is seen, involving in particular the papillary muscles, bundle branches, and trabeculae. Interestingly, preconditioning the myocardium with high-energy shocks could prevent reinduction of fibrillation with lower energy shocks that were previously within the window of vulnerability.[73] Thus, it is possible that an electroporative effect on myocardium may play a role in defibrillatory success.

Defibrillation theory summary

To summarize, the effects of the application of a voltage gradient across myocardium are a function of field strength and timing. Although the biologic effects of shocks may overlap, this concept is summarized in Fig. 1.21. Simply, extremely low energy pulses may have no effect on the myocardium, whereas stronger pulses (in the microjoule region), such as those used for cardiac pacing, result in action potential generation in nonrefractory myocardium, which leads to a propagating impulse. With further increases in electric field strength (to the 1 J area), VF can be induced with shocks delivered during the vulnerable period in normal rhythm. Increasing the shock strength above the ULV (and above the DFT) then puts the shock in the defibrillation zone. It is important to note, however, that very high energy shocks can lead to toxic effects, including disruption of cell membranes, post-shock block, mechanical dysfunction, and new tachyarrythmias.[68]

The importance of waveform

The shape of a defibrillating waveform can affect its defibrillation efficacy. As in pacing, the battery serves as the source of electrical charge for cardiac stimulation in defibrillation. Before a high-energy shock can be delivered, the electrical charge must be accumulated in a capacitor, because a battery cannot deliver the amount of charge required in the short time of a defibrillation shock. A capacitor stores charge by means of two large surface area conductors separated by a dielectric (poorly conducting) material, and capacitor size is an important determinant of implantable defibrillator volume, typically accounting for approximately 30% of device size. If fluid analogies are used for electricity (voltage as water pressure, and current as water flow), the capacitor is analogous to a water balloon, which has a compliance defined by the ratio of volume to pressure. To increase the amount of water put into the balloon, one can increase the pressure or, alternatively, use a balloon with a greater compliance (more stretch for a given amount of pressure). Similarly, the charge stored can be increased by increasing capacitance or by applying greater voltage. The trend in implantable devices has been toward smaller capacitors to create smaller devices.

The charge Q stored by a capacitor is defined by

$$Q = C \times V$$

where C is the capacitance and V the voltage. The voltage waveform of a capacitor discharged into a fixed-resistance load (Fig. 1.22) is determined by

$$V(t) = V_i e^{-t/RC}$$

where R is the resistance, t is time, and V_i is the initial voltage. The energy E associated with the waveform is given by

$$E = 0.5CV^2$$

Because the "tail" of the waveform in longer pulses (≥10 ms) refibrillates the ventricle, truncated waveforms have been used clinically. The classic monophasic truncated waveform is shown in Fig. 1.22B. The waveform is characterized by the initial voltage V_i, the final voltage V_f, and the pulse width or tilt. Tilt is an expression of the percentage decay of the initial voltage. The tilt of a waveform is a function of the size of the capacitor used, the resistance of the leads and tissues through which current passes, and the duration of the pulse. Tilt is defined by the percentage decrease of the initial voltage:

$$\text{Tilt}\ (\%) = \frac{V_i - V_f}{V_i} \times 100$$

Tilt can have an important effect on defibrillation efficacy, with progressive improvement in defibrillation efficacy with decreasing tilt for a trapezoidal waveform of constant duration. For monophasic waveforms formerly used clinically, the optimal tilt was 50–80%.

Biphasic waveforms

Appropriately characterized biphasic shocks can result in significant improvement in defibrillation efficacy,

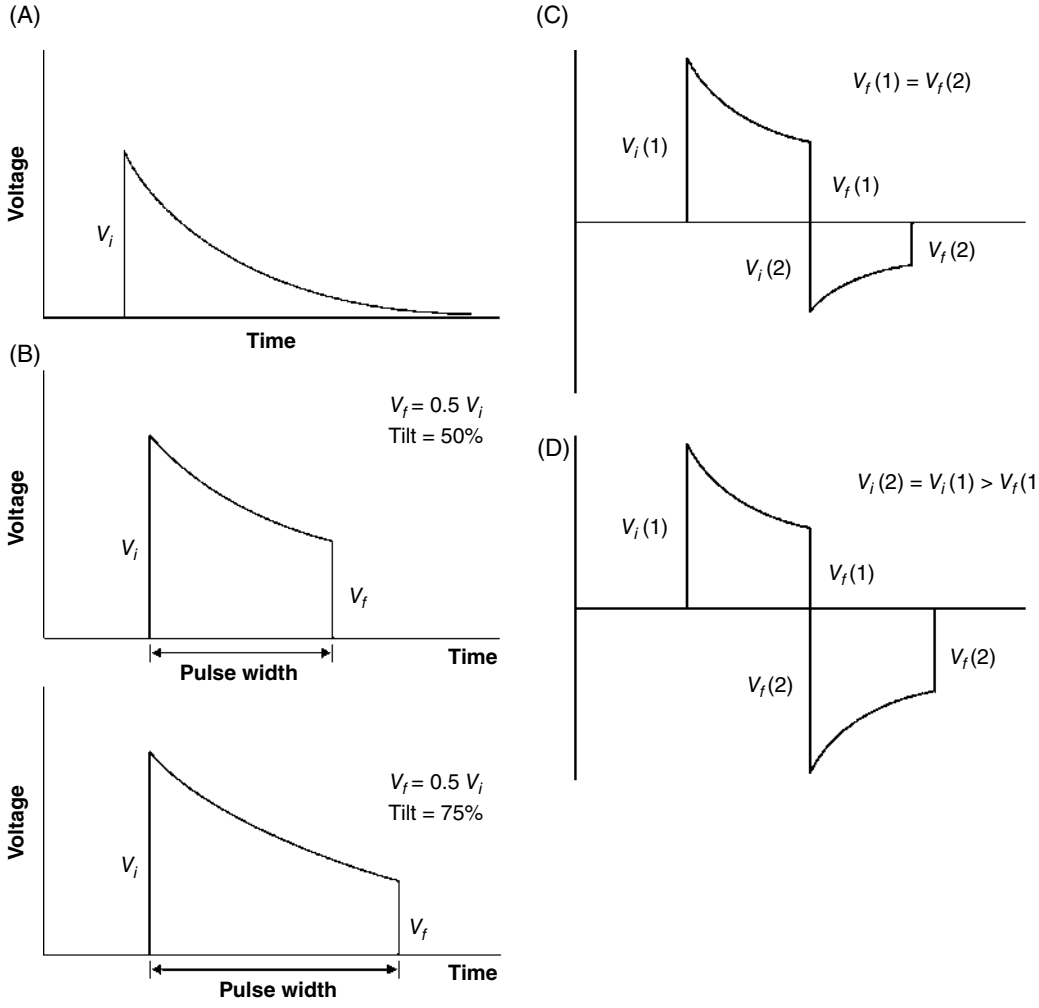


Fig. 1.22 Defibrillation waveforms. (A) Standard capacitor discharge. (B) Monophasic truncated waveform with initial voltage V_i, final voltage V_f, and pulse width labeled. Top waveform has 50% tilt, and bottom waveform has 75% tilt. (C) Biphasic waveform with leading edge of the first pulse $V_{i(1)}$, trailing edge of the first pulse $V_{f(1)}$, leading edge of the second pulse $V_{i(2)}$, and trailing edge of the second pulse $V_{f(2)}$ labeled. As $V_{i(2)} = V_{f(1)}$, this waveform can be generated by reversing the polarity of a single capacitor after the first pulse is completed. (D) In contrast, $V_{i(2)} > V_{f(1)}$, so that a second capacitor is needed to create this waveform.

with reductions in DFTs (a measure of defibrillation energy requirements) of 30–50%.[74] All currently available commercial defibrillators use biphasic waveforms; a typical biphasic waveform is shown in Fig. 1.22C. Biphasic waveforms have numerous clinical advantages, all stemming from their improved defibrillation efficacy. Biphasic waveforms have been shown to result in higher implantation success rates because of their lower DFTs, and thereby higher safety margins.[75] Because safety margins are increased, most patients do not require high-energy shocks, and smaller devices can be designed.[76] The improved efficacy of biphasic waveforms permits a greater tolerance in electrode positioning than that required for monophasic waveforms, facilitating the implanting procedure. Additionally, biphasic shocks have been shown to result in faster post-shock recurrence of sinus rhythm and to have greater efficacy than monophasic shocks in terminating VF of long duration.[77,78]

With the development of biphasic defibrillation waveforms, the energy required for defibrillation has been reduced.[79–81] Simultaneously, advances in capacitor and battery technology have allowed for a reduction in pulse generator size. Further advances that will reduce the generator size will occur when the energy required for defibrillation is reduced.[79]

Phase duration and tilt

In most commercially available ICDs, pulse duration and tilt are preset to values found to be optimal based on experimental evidence (Fig. 1.23). Some devices permit individualization of the pulse widths, based on the concept that individual variations in cellular time constants result in varying optimal pulse durations. Anecdotal observations and small studies support pulse width optimization in high DFT patients.[82,83] Waveform optimization is used infrequently in clinical practice, but may be useful in some high DFT patients.

Polarity and biphasic waveforms

Polarity is an important determinant of monophasic defibrillation, with lower DFTs found for transvenous systems when the RV electrode is the anode (+).[84,85] The results of studies of biphasic polarity are less uniform, with some reports showing an effect of biphasic polarity but others indicating no effect.[86,87] However, all studies demonstrating a polarity effect have found that waveforms with a first phase in which the RV electrode is the anode (+) are more effective. Additionally, biphasic polarity has the greatest effect on patients with elevated DFTs. In a study of 60 patients, use of biphasic waveforms with a RV anodal first phase resulted in a 31% reduction in DFT in patients with DFT ≥ 15 J, whereas polarity made no difference in patients with DFT < 15 J.[88] Despite the fairly uniform population improvement in DFT with a ventricular anodal first-phase polarity among studies in which an effect was seen, there is clearly individual variability, so that if an adequate safety margin cannot be found in a patient then a trial of the opposite polarity is reasonable, particularly if the initial polarity tested was not anodal in the right ventricle for the first phase.

Mechanism of improved efficacy with biphasic waveforms

Several theories have been proposed to explain the observed superiority of biphasic over monophasic waveforms. None provide a complete explanation for the

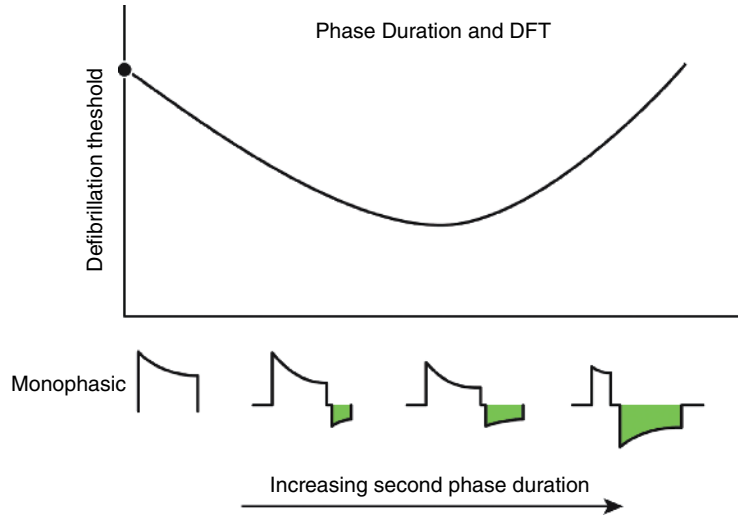


Fig. 1.23 Idealized curve demonstrating the relationship between second phase duration and defibrillation threshold (DFT). Details are in the text. (Source: From Wessale JL, Bourland JD, Tacker WA, Geddes LA. Bipolar catheter defibrillation in dogs using trapezoidal waveforms of various tilts. J Electrocardiol 1980; 13:359–65, by permission of Churchill Livingstone.)

benefits seen, and the fundamental mechanism remains to be determined. However, the clinical superiority of biphasic shocks has been a consistent and reproducible finding. All ICDs today use biphasic defibrillation.

Measuring shock dose

The shape of the waveform is a function of the initial voltage, the size of the capacitor, and the resistance of the load. If a smaller capacitor is used to diminish device size, a larger initial voltage may be needed to deliver an equivalent amount of charge into the fibrillating tissue. Thus, two waveforms may have different leading-edge voltages but have the same energy if there are differences in capacitance (Fig. 1.24). Therefore, the question of how to determine the "dose" of a shock arises. The "dose" of defibrillation is usually given in units of energy (joules) on the basis of tradition and ease of measurement. Physiologically, however, energy has little bearing on defibrillation; the voltage gradient is the factor that affects membrane channel conductance;, and at the tissue level, several decades of animal and human research have shown current to be the most important factor for generating action potentials and for defibrillation.[68] To add to the complexity, energy can be described as the stored energy – the amount of energy stored in the capacitor before shock delivery – or the energy delivered. Because the waveforms are truncated, usually around 10% of the stored energy is not delivered. Additionally, although the term is used clinically, "delivered energy" is highly variable, depending on where the delivery is recorded; energy delivered at the lead surface is not the same as energy delivered only a few millimeters into the tissue. Some device manufacturers, in fact, simply report an arbitrary percentage of the stored energy as the delivered energy.

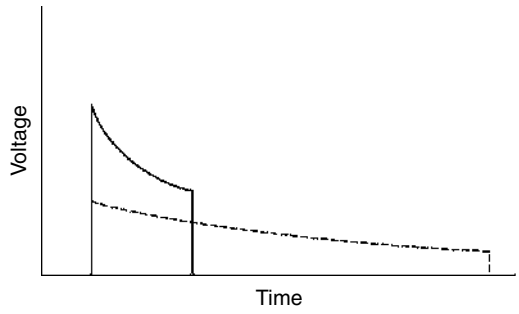


Fig. 1.24 Two waveforms with different voltages but the same energy. The solid waveform has a higher initial voltage but a smaller capacitance and, consequently, a shorter pulse width. The dashed waveform starts with a lower voltage but has a greater capacitance and pulse width, resulting in the same energy delivery despite the marked differences in the voltages. Further details in the text.

Over the range of clinically utilized capacitor size and biologic tissue resistance in a given system, a change in energy up or down is reflected by a similar change in voltage and current. In practice, "energy" is the most commonly used term to indicate shock dose.

Measuring the efficacy of defibrillation

Threshold and dose–response curve

A measure frequently used to assess the ability of a system to terminate VF is the DFT. The term "threshold" suggests that there is a threshold energy above which defibrillation is uniformly successful and below which shocks fail (Fig. 1.25A). The multitude of factors that affect whether a shock will succeed – patient characteristics, fibrillation duration, degree of ischemia and potassium accumulation, distribution of electrical activation at the time of the shock, circulating pharmacologic agents, and others – result in defibrillation behavior that is best modeled as a random variable, with a calculable probability of success for any given shock strength. Thus, defibrillation is more accurately described by a dose–response curve, with an increasing probability of success as the defibrillation energy increases (Fig. 1.25B). The curve can be characterized by its slope and intercept, and specific points on the curve can be identified, such as ED_{50}, the energy dose with a 50% likelihood of success. Factors adversely affecting defibrillation shift the curve to the right, so that a higher dose of energy is required to achieve a 50% likelihood of success, and improvements in defibrillation (such as superior lead position and improved waveforms or lead design) shift the curve to the left (Fig. 1.26). Because of the large number of fibrillation episodes required to define a curve (30–40 inductions), the dose–response curve is not determined in clinical practice, but it remains a useful research tool and conceptual framework. However, because the term DFT is widely used in the literature, it is adopted in this chapter.

Relationship between defibrillation threshold and dose–response curve

The probability of successful defibrillation at the DFT energy depends on the steps taken to define the threshold. Consider a step-down to failure DFT, in which shocks are delivered beginning at a relatively high energy (e.g., energy with a 99% success rate) and decremented by several joules with each VF induction until a shock fails (at which point a rescue shock is delivered). The DFT in this protocol is defined as the lowest energy shock that succeeds (Fig. 1.27). Because the initial energies tested are at the upper end of the dose–response curve, successive shocks may have a 98%, 95%, 88%, 85% (and so on) likelihood of success, depending on the starting energy and size of the steps taken. Despite

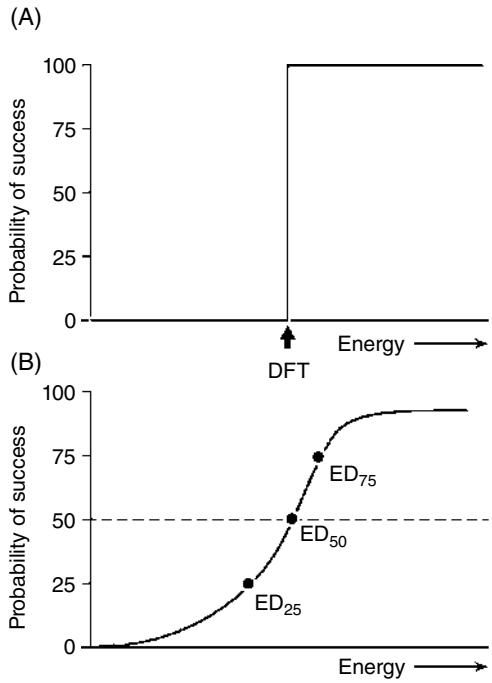


Fig. 1.25 Defibrillation "threshold" (DFT). (A) The expected response to shock if a true threshold value existed. In reality, the likelihood of success is a sigmoidal dose–response curve, as shown in (B). The ED_{50} is the energy dose with a 50% likelihood of success, and so on.

the fairly high likelihood of success for each shock individually, the sheer number of shocks delivered in this range, on average, result in a shock failing (thus defining the DFT) at a relatively high point on the curve. If this process is repeated many times, a population of DFTs is created, with a mean and expected range. In humans, step-down to failure algorithms have a mean DFT with likelihood of success near 70%, but with a standard deviation near 25%.[89,90] Thus, the likelihood of success of a shock delivered at the energy defined as the DFT at a single determination ranges from 25% to 88%, with an average of 71%.[90] In other words, if a defibrillator is programmed to the step-down to failure DFT energy for its first shock, the likelihood that the first shock will succeed can range from 25% to 88%, but on average will be 71%.

In contrast to the step-down to failure DFT, in a step-up to success DFT, low-energy shocks are delivered during VF with incremental doses of energy until a first success occurs, which defines the DFT. In this case, despite the fairly low likelihood of success at each low-energy shock, if enough shocks are delivered, one is likely to succeed, defining the DFT. With this protocol, the mean DFT has a likelihood of success near 30%. Iterative increment–decrement DFT or binary search algorithms that begin in the middle zone of the curve have been shown to approximate the ED_{50}. In this type of protocol, if the first shock defibrillates the heart, the

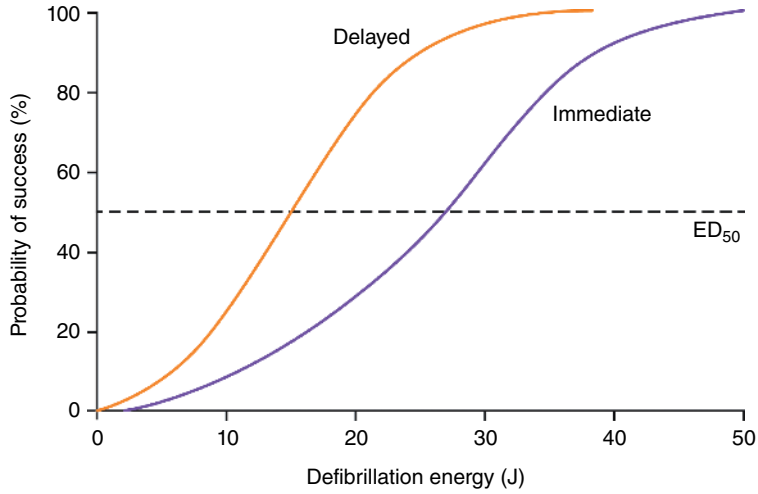


Fig. 1.26 Use of a dose–response curve to measure effects of an intervention on defibrillation efficacy. The graph shows the effect of thoracotomy on defibrillation in a canine model. The "immediate" group had defibrillation threshold testing performed immediately after thoracotomy. Note that the curve is shifted to the right and that the energy with a 50% probability of success is 27 J, compared with 15 J for the "delayed" group, which was allowed 48–72 h recovery before defibrillation testing. Defibrillation is more effective in the "delayed" group because the probability of success at a given energy is higher in this group. Thus, the curves graphically display diminished defibrillation efficacy immediately after thoracotomy. (Source: From Friedman PA, Stanton MS. Thoracotomy elevates the defibrillation threshold and modifies the defibrillation dose–response curve. J Cardiovasc Electrophysiol 1997; 8:68–73, by permission of Futura Publishing Company.)

first shock of the next fibrillation episode uses a lower energy. If the first shock does not defibrillate the heart, a second shock at a higher energy is delivered. Regardless of the DFT protocol, a DFT determination is best conceptualized as a means of approximating a point on the dose–response curve, with the specific point estimated being a function of the DFT algorithm chosen.

Patient-specific defibrillation threshold and safety margin testing – clinical indications

Patient-specific DFT testing determines the lowest energy that reliably defibrillates an individual patient. This permits programming a low first shock strength. The rationale for adopting this strategy is that the lower shock strength will result in the shortest charge time and consequent battery preservation, and diminished risk of syncope, post-shock AV block, myocardial damage, and impaired sensing.[91,92] With safety margin testing, the goal is to deliver the minimum number of shocks or induce the fewest possible VF episodes to determine whether a sufficient safety margin exists between the maximum ICD shock strength and reliable defibrillation. Following a safety margin test, the first shock is typically programmed to maximum output. However, it is still unclear with modern devices whether there is a role for continued DFT testing. Higher voltage devices make it unlikely that a shock will fail in most scenarios. A modern biphasic active pulse generator device placed in the left pectoral position has a 95% probability of passing a 10 J safety margin test, and most patients who fail an implant test do so because of a false-negative result.[93] The utility of DFT testing at a de novo implant is unclear, though there may be a modest benefit.[94] It stands to reason that the greatest benefit would be in patients with right-sided or atypical locations for device implant, or in whom anatomic abnormalities may suggest risk for defibrillation failure. A recent meta-analysis shows that routine DFT testing over follow-up after implant does not confer any significant benefit.[95]

In deciding whether to perform DFT testing, the risks and benefits of the procedure must be considered. Risks of testing include the risks attributable to anesthesia, to VF itself, and to shock delivery in patients with significant cardiovascular disease and comorbidities. In a recent study from Canada, in 19,067 ICD implantations, eight serious DFT testing-related complications occurred (three deaths and five strokes).[96] These data suggest that the risks are low, even in high-risk patients, when the testing is performed by experienced practitioners. A risk of not performing testing includes failure to identify a patient who will not be adequately defibrillated. Clinical variables, including baseline ejection fraction, do not accurately identify patients who may have a high DFT.[97] Testing is favored by the presence of a nonstandard shock vector (i.e., right-sided or abdominal pulse generator, congenital heart disease, unusual superior vena cava [SVC] coil position, or extreme left ventricular enlargement), clinical conditions that might have an increased risk of an elevated DFT or for which the overall ICD experience is relatively limited (hypertrophic cardiomyopathy, channelopathies, arrhythmogenic RV dysplasia), and a secondary prevention indication. Testing is not performed in patients with absolute contraindications (Table 1.2),[98] and it is less commonly performed in primary prevention cardiac resynchronization recipients, in whom the role of testing has been questioned and the perceived risks higher.[99]

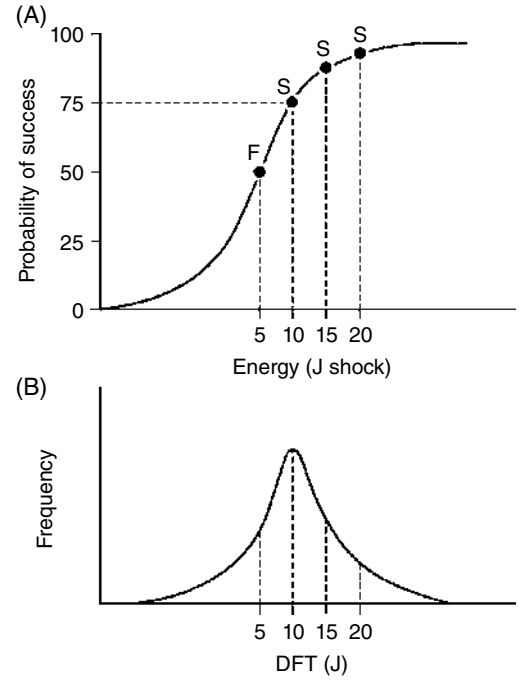


Fig. 1.27 Step-down to failure defibrillation threshold (DFT) testing. (A) Hypothetical example in which four shocks are required to define the DFT. The first shock is delivered at 20 J and is successful (S). The next shock, delivered at 15 J, also succeeds. A 10 J shock succeeds, and a 5 J shock fails (F), defining the DFT as 10 J (the lowest successful energy). Note from the curve that the likelihood of success at the DFT energy (10 J) is 70%. Now, if the DFT process were repeated, it is possible that the second shock might fail on one occasion (defining the DFT as 20 J) or that all four shocks might succeed on another occasion (and that a lower energy shock would fail to define the DFT), and so on. Thus, repeating the DFT determinations may result in different values for the DFT with each determination. However, if enough repetitions were performed, a population of DFTs, as shown in (B), would be created. The most commonly observed DFT in this example would be 10 J, which has a 70% likelihood of success. Further details in text.

Management of the patient who fails defibrillation testing

Before taking steps to manage defibrillation testing failure the diagnosis should be confirmed, because a single failed shock may occur by chance alone. If a test shock fails, but a maximum output rescue shock from the device succeeds, it is reasonable to repeat the test shock. If the maximum output shock also fails, or if the test shock fails twice, reliable defibrillation with a 10 J safety margin is likely absent and system modification is warranted. Options to address this are summarized in Table 1.3.

Defibrillation efficacy is modified by changing the waveform, altering the vector, or (at times) substituting the pulse generator for one with a higher output (Table 1.3). In general, the following steps are performed. First, if the implant procedure was prolonged, metabolic abnormalities may be present; if so, it may be reasonable to defer testing if they are not readily corrected. Screening for a pneumothorax also may also identify a treatable cause of an elevated DFT.

Second, it is important to insure an adequate vector, by assessing the position of the leads and can relative to the heart, and in particular the left ventricle. An anterior chest wall can to an RV lead coil may fail because both electrodes are relatively anterior. Insuring the RV lead is apical and that the SVC coil is in the high SVC or innominate vein optimizes vectors (Fig. 1.28). If the coil is low, it should be excluded (performed electronically in many devices). With a right-sided pulse generator, removing the can from the circuit may improve defibrillation. If the maximum output shock succeeded but the safety margin failed, reversing polarity (if the default polarity is not RV anodal for the first phase) or reprogramming shock pulse width (if an option for the pulse generator in use) may help.

If these approaches fail to result in adequate defibrillation, a subcutaneous lead is added. With current biphasic waveform systems, subcutaneous leads are required in only 3.7% of devices implanted.[100] An alternative to placing a subcutaneous lead (which may be associated with patient discomfort and increased fracture risk) is the addition of a defibrillation coil in the azygous vein (which lies directly behind the left ventricle) or in the branches of the coronary sinus (Fig. 1.29A).

Because the pulse generator shell serves as an electrode, its position can also affect defibrillation efficacy. Implantable defibrillators are most commonly placed in the left pectoral region, typically in the prepectoral (subcutaneous) plane. However, the site of pulse generator placement and vascular access is influenced by multiple factors, including patient and physician preference, anatomic anomalies, previous operations, integrity of the vascular system, and whether a preexisting permanent pacing system is present. In addition to factors specific to the patient, choice of the implantation site can affect ease of technical insertion, defibrillation effectiveness, and long-term rates of lead failure.

Right pectoral implantation may be considered in left-handed persons, hunters who place the rifle butt on the left shoulder, and patients with previous mastectomy, other surgical procedures, or anatomy that precludes left-sided insertion. In systems with both distal and proximal defibrillation coils, the proximal coil is either shifted toward the right hemithorax

Table 1.2 Contraindications to implantable cardioverter-defibrillator implant testing.

Absolute contraindication
Risk of thromboembolism
Left atrial thrombus
Left ventricular thrombus, not organized
Atrial fibrillation in the absence of anticoagulation
Inadequate anesthesia or anesthesia support
Known inadequate external defibrillation
Severe aortic stenosis
Critical, nonrevascularized coronary artery disease with jeopardized myocardium
Hemodynamic instability requiring inotropic support
Relative contraindication
Left ventricular mural thrombus with adequate systemic anticoagulation
Questionable external defibrillation (e.g., massive obesity)
Severe unrevascularized coronary artery disease
Recent coronary stent
Hemodynamic instability
Recent stroke or transient ischemic attack
Questionable stability of coronary venous lead

Source: Swerdlow CD, Russo AM, Degroot PJ. The dilemma of ICD implant testing. Pacing Clin Electrophysiol 2007; 30:675–700, by permission of Blackwell Publishing.

Table 1.3 Options in a patient with high energy requirements or an inadequate safety margin at defibrillation threshold testing.

Check for metabolic abnormalities or pneumothorax
Assess vector (insure right ventricular lead is apical, exclude superior vena cava coil if it is low in the right atrium or move it proximally, exclude the pulse generator with right-sided implants)
Reverse polarity (particularly if initial shock was not right ventricular anode for the first phase) or modify the waveform (if available)
Exchange the generator to a "high-output" device (if not already in use)
Add a subcutaneous array or patch, or add an azygous lead or coronary sinus coil to include more of the left ventricle in the defibrillation field
Move the generator to a left pectoral position if located on the right

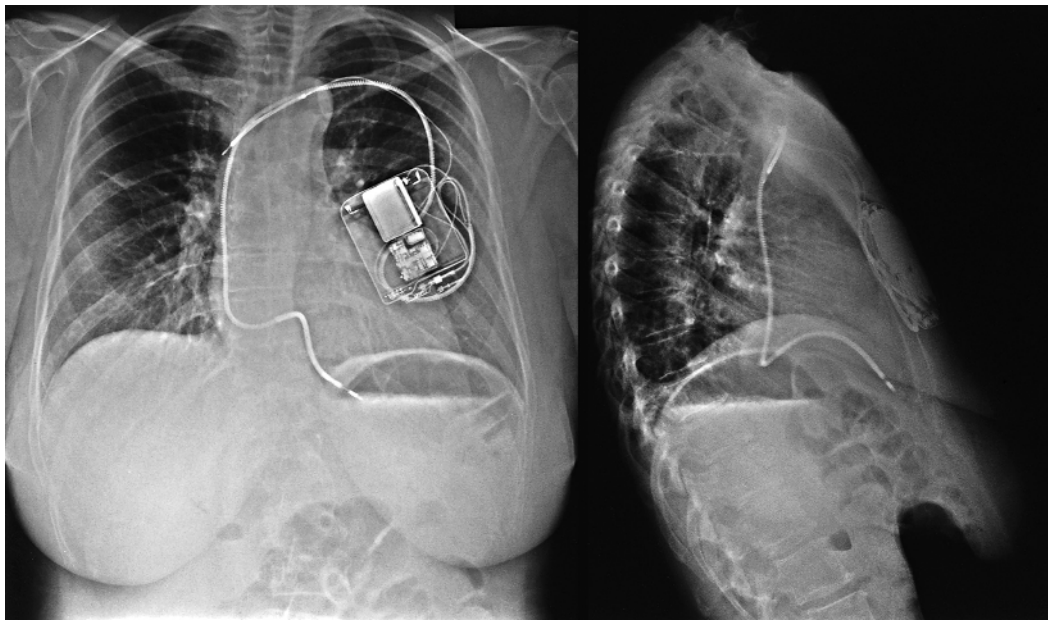


Fig. 1.28 Chest radiographs depict active pulse generator shell system with an added proximal defibrillation coil to optimize defibrillation threshold.

(if both coils are on the same lead) or, often, advanced to a lower SVC position for greater cardiac proximity (in two-lead systems) with right-sided placement. With active can pulse generators, the largest defibrillation lead surface, the device shell, is shifted away from the ventricular myocardium (Fig. 1.30). This unfavorable position decreases defibrillation effectiveness.[101,102] With biphasic waveforms, right-sided implantation results in a 6 J increase in DFT compared with left-sided placement (11.3 ± 5.3 J, left-sided; 17.0 ± 4.9 J, right-sided; $P < 0.0001$).[102] In general, however, left-sided insertion is superior to right-sided placement and is used if there are no compelling factors against it. Placement of a defibrillation coil in the azygous vein or coronary sinus branch is infrequently required, but it may result in a favorable vector and improved DFT (Fig. 1.31).

An alternative site for device placement is the abdomen, but this site is only rarely used. Although not as effective for defibrillation as the left pectoral position, the abdomen appears superior to the right pectoral location for active can placement.[103] However, abdominal insertion is technically more challenging, requiring two incisions, lead tunneling, abdominal dissection (often necessitating surgical assistance), and general anesthesia. Additionally, because of the greater risk of infection, threat of peritoneal erosion, and increased risk of lead fracture, even with totally transvenous systems this position is used only in rare circumstances.

There are many factors that may result in elevated DFT: drug therapy; underlying cardiac disease; the size, configuration, and number of defibrillating leads; the time that VF persists before shock delivery; ischemia; hypoxia; amplitude of the VF waveform; temperature; heart weight; body weight; direction of the delivered shock and waveform; and chronicity of lead implantation. In patients with inherited channelopathies, such as Brugada syndrome, high DFTs may be prevalent and problematic.[104] In one series of patients who received a high-output generator for an elevated DFT, the majority had underlying coronary artery disease, with reduced left ventricular function, and were on amiodarone. An important finding in this study was that, in patients with high DFTs who receive an ICD, arrhythmia death remained a significant long-term risk (42% of the deaths were arrhythmia related).

An interesting observation is that there is a circadian variation in the DFT. The DFT has a morning peak that is 16% higher than that measured after noon.[105] In addition, the first failed shock rate is more likely to occur in the morning than at other times during the day. This variability in DFT is clinically important in patients with high thresholds, in whom a 10 J safety margin becomes more difficult to achieve.

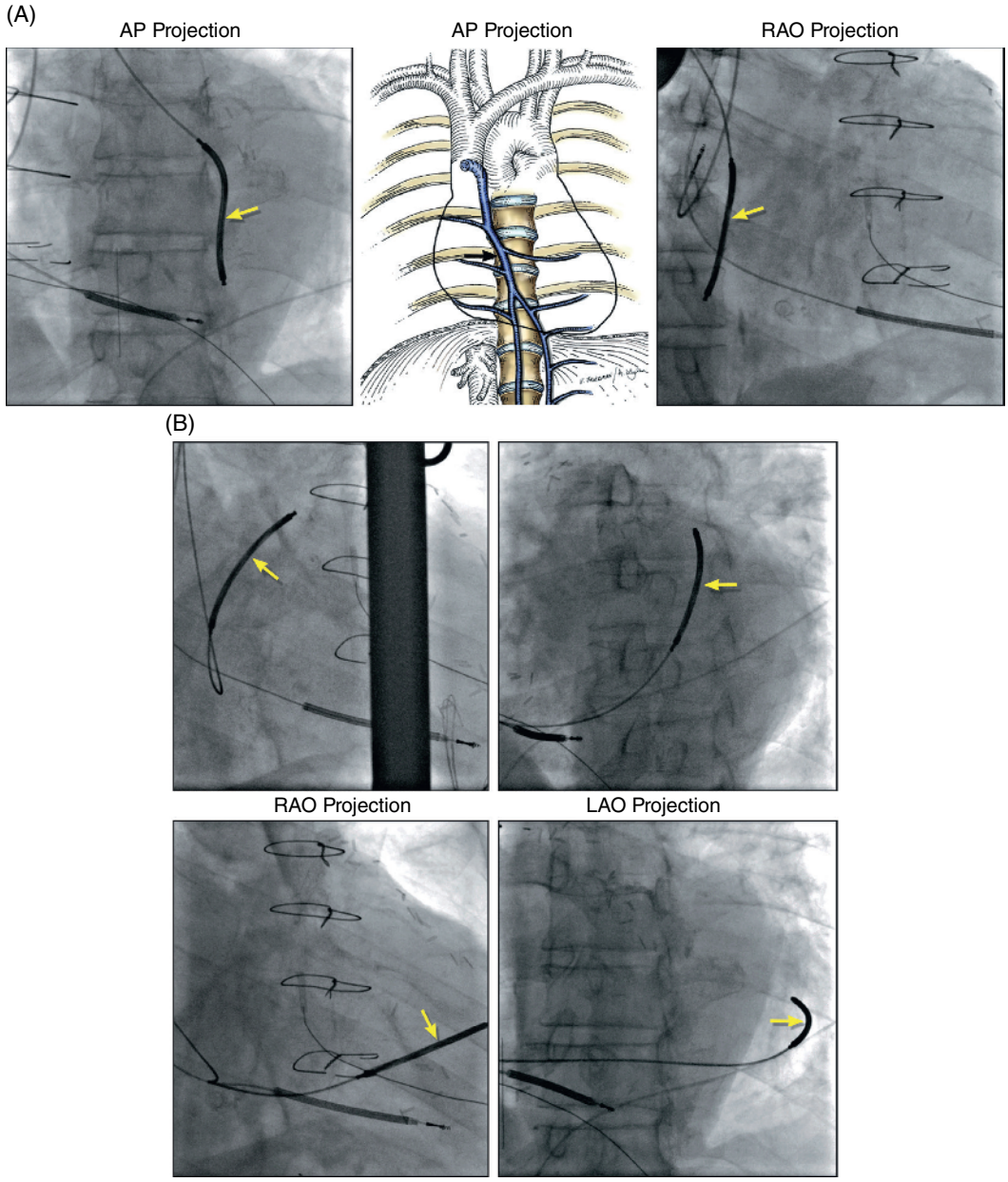


Fig. 1.29 (A) Placement of defibrillation coil in azygous vein to lower the defibrillation threshold by placing a coil behind the heart. Thus, current flows from behind the heart (azygous vein) to the anterior chest (pulse generator). The left panel shows a fluoroscopic anteroposterior (AP) projection. The middle panel shows a cartoon of the relevant anatomy (Source: Adapted from Cooper JA, Smith TW. How to implant a defibrillation coil in the azygous vein. Heart Rhythm J 2009; 6:1677–80). The right panel shows the right anterior oblique (RAO) projection. The arrow in each case points to the coil in the azygous vein. (B) Placement of a coil in the coronary sinus (CS). Top two panels: coil in main body of the CS. Bottom two panels: coil in the posterolateral branch of the CS in the same patient. This position resulted in effective defibrillation. Note that in the left anterior oblique (LAO) view the coil clearly encompasses the lateral aspects of the cardiac silhouette, suggesting the defibrillation vector effectively surrounds the heart.

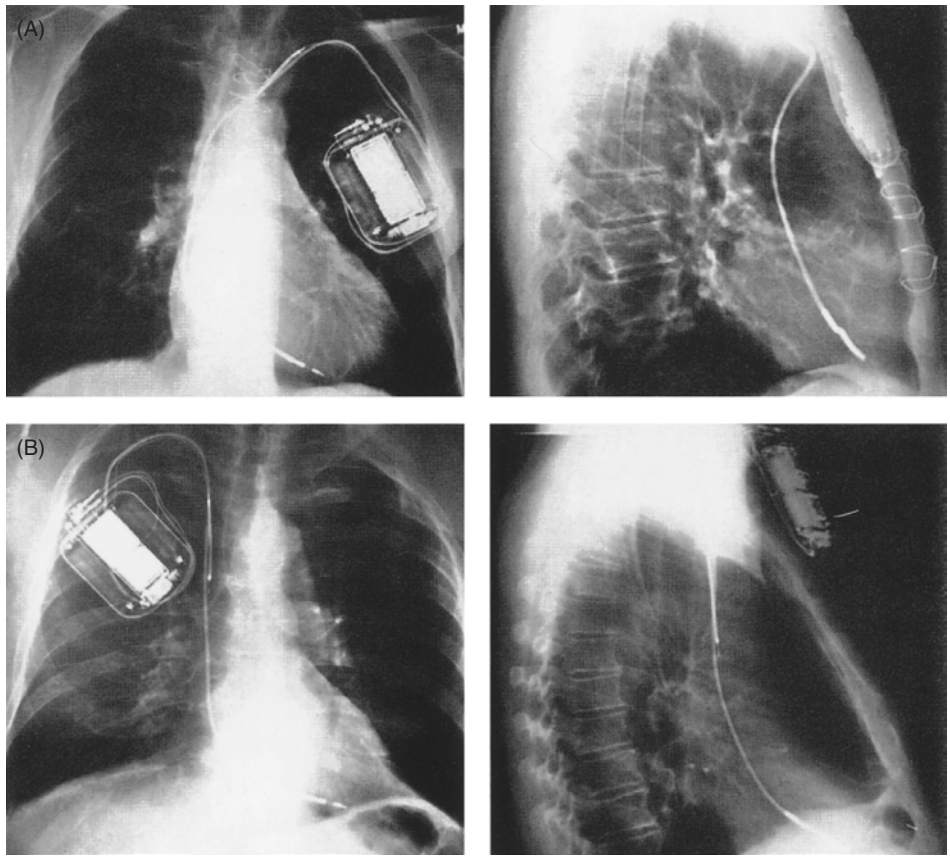


Fig. 1.30 (A) Posteroanterior and lateral chest radiographs from a patient with a left-sided defibrillator. Note that the proximal defibrillation lead is in the left subclavian vein. (B) Posteroanterior and lateral chest radiographs from a patient with right-sided defibrillator placement. Note that the proximal defibrillation lead is in the superior vena cava.

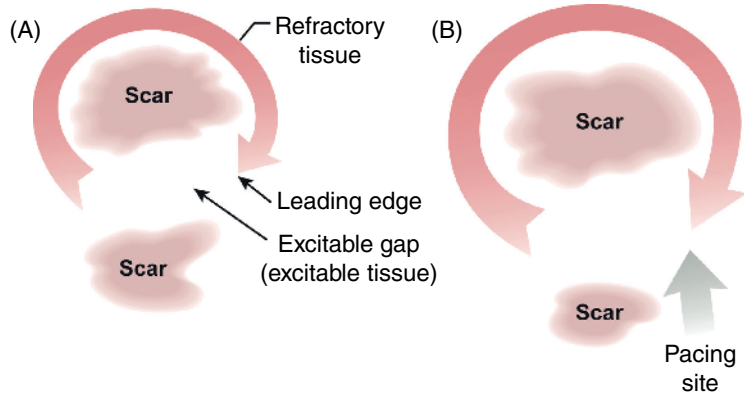


Fig. 1.31 Reentrant ventricular tachycardia circuit. (A) The circuit around a fixed scar is depicted by the arrow. The head of the arrow depicts the leading edge of the wave front, and the body of the arrow back to the tail consists of tissue that is still refractory (because the wave front has just propagated through it). The tissue between the tip and the tail of the arrow is excitable and is called the "excitable gap." For the arrowhead to continue its course around the scar, an excitable gap must be present; if the wave front encounters refractory tissue, it cannot proceed. (B) The wave front generated by an antitachycardia pacing impulse enters the excitable gap and terminates tachycardia. Tachycardias with a small excitable gap (i.e., the head of the arrow follows the tail very closely, so that only a small "moving rim" of excitable tissue is in the circuit) are more difficult to terminate with antitachycardia pacing.

Upper limit of vulnerability to assess safety margin

The ULV is the lowest energy above which shocks delivered during the vulnerable period do not induce fibrillation. Numerous studies have demonstrated that the DFT and ULV are strongly linked, with the ULV approximating the E90 (shock with 90% likelihood of success).[106–108] Because the DFT and ULV are correlated, delivery of a shock during the vulnerable period (T wave) that fails to induce VF indicates that the shock is of sufficient strength to terminate VF.[109] During sinus rhythm, test shocks are delivered at and around the peak of the T wave at a single energy (margin testing) or at progressively lower energies until VF is induced (patient-specific testing). Because the ULV may be dependent on the coupling interval, shocks are delivered at various intervals before the T-wave peak to "scan" repolarization. Shocks programmed 5 J above the ULV terminate spontaneous VF as reliably as shocks programmed using the DFT with a 10 J safety margin.[110,111] Because of the need to record 6–12 surface leads to insure proper shock timing, the lack of experience by many implanters with determining the timing of the test shocks, and the modest increase in time required to deliver three or four sinus rhythm shocks as opposed to a single VF induction, ULV testing has only been adopted as routine clinical practice in a few centers. Simplified approaches to ULV testing have been suggested, such as using a single electrogram-derived coupling interval to the latest peaking T wave on the surface ECG.[112]

Practical implications of defibrillator therapies

Though much has been published on the net mortality benefit of defibrillator therapies, recent studies have suggested that there can be incremental risk attributable to repeated defibrillation. For example, the TropShock trial demonstrated that the ICD shock, not VF, causes the increase in high sensitive troponin T after DFT testing.[113] In addition, the psychologic effects of repeated defibrillator therapies can be significant. Thus, though ensuring adequate programming of defibrillator therapy is critical to offer lifesaving benefits in patients at risk for ventricular arrhythmias, this needs to be balanced against other modalities to reduce shock burden. Given this, expert consensus statements on optimal programming parameters, in part to reduce the likelihood of need for a shock, have been published.[114] These approaches tend to favor ATP over ICD shock to try and terminate an arrhythmia when possible (more on ATP shortly).

Drugs and defibrillators

Antiarrhythmic drugs are frequently used in patients with ICDs to treat supraventricular arrhythmias (particularly atrial fibrillation), suppress ventricular tachyarrhythmias, and slow ventricular tachycardia (VT) to increase the responsiveness of antitachycardia pacing. In the implantable defibrillator trials, concomitant use of membrane-active agents (Vaughan-Williams class I or III drugs) has ranged from 11% to 31%.[115–118] Several important device–drug interactions must be considered.[119]

1. **Detection:** Most drugs slow VT. If slowed below the detection cut-off rate, VT is not detected by the device and remains untreated. Initiation of antiarrhythmic drugs in patients with VT is usually followed by device testing to assess detection of VT. This is the most important device–drug interaction with modern ICDs.
2. **Pacing thresholds:** Bradycardia and antitachycardia pacing thresholds may be affected by pharmacologic agents.
3. **Pacing requirements:** Drugs may exacerbate conduction defects or slow the sinus rate, necessitating pacing for bradycardia.
4. **Drug-induced proarrhythmia:** All antiarrhythmic drugs, in their effort to prevent arrhythmias, may alter normal cellular activation, thus leading to proarrhythmic potential.
5. **Changes in DFT:** Although it is well known that pharmacologic agents can modulate defibrillation effectiveness, drug–defibrillation interactions are complex. In general, agents that impede the fast inward sodium current (such as lidocaine) or calcium channel function (such as verapamil) increase the DFT, whereas agents that block repolarizing potassium currents (such as sotalol) lower the DFT. Long-term administration of amiodarone increases DFTs, whereas intravenous administration has little immediate effect on DFTs. In addition to antiarrhythmic agents, other drugs have been shown to increase the DFT, such as sildenafil,[120] venlafaxine,[121] and alcohol.[122]

Importantly, with the current of generation biphasic ICDs, the clinical effect of most drugs, including amiodarone, is modest.[123] In general, then, ICD evaluation should be performed when administration of membrane-active drugs that can increase the threshold (especially amiodarone) is initiated, particularly in patients with borderline DFTs. Drug effects on defibrillation are summarized in Table 1.4.

Antitachycardia pacing

In monomorphic VT, a reentrant circuit utilizing abnormal tissue (adjacent to an infarct in the postmyocardial infarction patient) is responsible for the arrhythmia (Fig. 1.31). For the reentrant circuit to perpetuate itself, the tissue immediately in front of the leading edge of the wave front must have recovered excitability so that it can

Table 1.4 Effects of drugs on defibrillation.

Drug	Class*	Effect on defibrillation threshold†
Quinidine	IA	Increase
Procainamide	IA	No change
N-Acetyl-procainamide	IA	Decrease
Disopyramide	IA	No change
Mexiletine	IB	Increase
Flecainide	IC	Increase
Moricizine	IC	Increase
Propafenone	IC	No change
Propranolol	II	Increase
Atenolol	II	No change
Isoproterenol		Decrease
Sotalol	III	Decrease
Ibutilide	III	Decrease
Dofetilide	III	Decrease
Amiodarone	III	
Oral		Increase
Intravenous		No change or decrease
Dronedarone	III	No change
Diltiazem	IV	Increase
Verapamil	IV	Increase

* Vaughan-Williams classification.

† If study results conflict, the most frequently reported effect is noted.

Source: Modified from Carnes CA, Mehdirad AA, Nelson SD. Drug and defibrillator interactions. Pharmacotherapy 1998; 18:516–25, by permission of Pharmacotherapy Publications.

be depolarized (Fig. 1.31). Thus, an excitable gap of tissue must be present in advance of the leading tachycardia wave front or the arrhythmia will terminate. ATP – delivered as a short burst of pacing impulses at a rate slightly greater than the tachycardia rate – can terminate VT by depolarizing the tissue in the excitable gap, so that the tissue in front of the advancing VT wave front becomes refractory, preventing further arrhythmia propagation. The ability of a train of impulses to travel to the site of the reentrant circuit and interrupt VT depends on several factors, including the site of pacing (the closer to the circuit entrance, the greater the likelihood of circuit penetration and termination), the length of the tachycardia cycle, and the size of the excitable gap. With delivery of ATP, faster and more remote circuits with smaller excitable gaps are generally more difficult to terminate and have a greater risk of degeneration to less organized tachyarrhythmias, including fibrillation.

ATP has been applied successfully to treat slow VT (<188–200 bpm, success rate 78–91%), and recently fast VT (200–250 bpm, success rate 50–81%).[124–126] Patients with ATP, rather than those programmed to defibrillation shocks only, report statistically higher quality-of-life scores. If ATP fails, or if the frequency of the VT is too high to apply ATP, the device diverts immediately to deliver a defibrillation shock. The use of ATP in the ventricle is important in limiting shocks. Most defibrillators offer ATP immediately before or during charging, because many tachyarrhythmias with cycle lengths in the VF zone are actually fast monomorphic VT. ATP is also available in the atrium in some dual-chamber ICDs, although its efficacy and role in clinical practice is far more limited.

When considering success of ATP, it is important to consider the mechanism underlying the arrhythmia. Prior work has suggested appropriately timed pacing cycles may even have the potential to regularize fibrillation in certain models.[127] Furthermore, the application of stimulatory potentials may have a virtual electrode effect that could alter defibrillatory potential.[128] When a patient has had failed ATP, a review of the device tracings may help facilitate appropriate reprogramming of ATP settings. As stated previously, defibrillatory shocks do hold the potential to cause injury to myocardium. Thus, preference toward ATP is critical to programming of implantable defibrillators. Recent data suggest that slower VT and lack of amiodarone use may be associated with higher likelihood of ATP success, though the reasoning for the latter is unclear.[129]

Summary

The basics of pacing involve an understanding of how stimulation occurs (application of energy between a cathode and anode), how interfacing with the myocardium plays a role in this via stimulation threshold and sensing, and the implications of lead design and material composition. In addition, the mechanisms underlying cardiac defibrillation require an intimate understanding of how electric fields interact with the heart through both virtual electrode and direct effects. Research into how to optimize defibrillation so as to achieve lower voltage conversion of arrhythmias is ongoing. However, efforts to reduce or eliminate the need for shocks are evolving.

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