Pacing and Defibrillation

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Abstract

Pacing and defibrillation systems monitor and treat inappropriate cardiac rhythms. In general, these inappropriate rhythms result in cardiac outputs that are inadequate to meet metabolic demands, and thus can be life-threatening. In order to best understand the function of such pacing and defibrillation systems, the underlying physiologic situations indicated for their use must also be defined and understood. Furthermore, as with the design of any biomedical device or system, a *first principles* understanding of the appropriate physiologic behavior is a prerequisite to the definition of the performance characteristics of the device. This chapter primarily aims to provide a basic understanding of the physiologic conditions that require intervention with pacing and/or defibrillation systems, as well as introduce technical information about these systems to provide the reader with a foundation for future research and reading on this topic.

Keywords

Cardiac pacing • Defibrillation • Cardiac arrhythmia • Electrical stimulation • Antiarrhythmic drugs • Drug interactions • Implantable pulse generator • Implantable cardioverter defibrillator • Leadless pacemaker

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30.1 Introduction

Pacing and defibrillation systems both monitor and treat inappropriate cardiac rhythms. In general, these inappropriate rhythms result in cardiac outputs that are inadequate to meet metabolic demands, and thus can be life-threatening. Currently, over 600,000 Americans have pacemakers and 150,000 have implantable cardioverter defibrillators (ICDs) [1].

In order to best understand the function of such pacing and defibrillation systems, the underlying physiologic situations indicated for their use must also be defined and understood. Furthermore, as with the design of any biomedical device or system, a *first principles* understanding of the appropriate physiologic behavior is a prerequisite to the definition of the performance characteristics of the device. This chapter primarily aims to provide a basic understanding of the physiologic conditions that require intervention with

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pacing and/or defibrillation systems, as well as introduce technical information on these systems to provide the reader with a foundation for future research and reading on this topic. The information provided in this chapter is by no means comprehensive and thus should not be used to make decisions relating to patient care.

30.2 Cardiac Rhythms and Arrhythmias

30.2.1 Cardiac Function and Rhythm

Cardiac output (CO) is defined as the heart rate (HR, beats per minute) multiplied by the stroke volume (SV, liters), or CO = HR × SV (L/min). Normally, the heart rate is determined by the rate at which the sinoatrial node (the *biologic pacemaker*) depolarizes. In healthy individuals, the sinoatrial node maintains the appropriate heart rate to meet variable metabolic demands (e.g., increasing with exercise). More specifically, the sinoatrial nodal rate is modulated by: (1) sympathetic and parasympathetic innervation; (2) local tissue metabolites and other molecules; (3) neurohormonal factors; and/or (4) the perfusion of the nodal tissues. Stroke volume is the quantity of blood ejected from the heart during each ventricular contraction. The instantaneous stroke volume is governed by a number of factors including: heart rate, degree of ventricular filling/atrial performance, atrial-ventricular synchrony, and/or myocardial contractility.

It is important to note that multiple physiologic and pathologic conditions exist that may result in an inappropriate cardiac output. These conditions need to be defined to understand the functional requirements of pacing and defibrillation systems, and to motivate the logic behind the system features and performance characteristics (Tables 30.1, 30.2, and 30.3) [2, 3].

30.2.2 Conditions of the Sinoatrial Node

Normal sinus rhythm	Sinoatrial nodal rate is appropriate for the current metabolic demand (see online Video 30.1).
Sinus bradycardia	A slow sinoatrial nodal rate, resulting in a slow heart rate which may or may not be functionally appropriate. HR↓ → CO↓
Sinus tachycardia	A fast sinoatrial nodal rate, resulting in a higher heart rate which may or may not be functionally appropriate. HR↑ → CO↑ (for excessive heart rates, CO↓ due to reduced filling time).
Sick sinus syndrome	Unpredictable sinoatrial nodal rate. The rate is not appropriately coordinated with physiologic demand. CO↑ or CO↓
Chronotropic incompetence	Inappropriate response of the sinoatrial node to exercise. CO is too low for metabolic demands.
Block	No sinoatrial nodal rhythm. The patient will have either no heart rate (asystole) or a rate defined by other regions within the heart. A rescue rhythm from the atrioventricular node normally occurs (40–60 beats per minute, a so-called *junctional rhythm*). HR = 0 → CO = 0 or HR↓ → CO↓

30.2.3 Conditions of the Atrioventricular Node

1st degree heart block	An atrioventricular interval >200 ms (normal atrioventricular interval is ~120 ms). SV↓ → CO↓
2nd degree heart block	Atrial and ventricular activity is not 1:1. Two types of 2nd degree block are defined: Mobitz types I and II.
	Mobitz type I: *Wenckebach phenomenon*. A ventricular beat is dropped after a progressive elongation of the atrioventricular interval. HR↓ (missed beat) → CO↓
	Mobitz type II: A ventricular beat is dropped without a progressive elongation of the atrioventricular interval. This is often an early indication of progressive disease of the conduction system. HR↓ (missed beat) → CO↓
3rd degree heart block	No atrioventricular nodal conduction (conduction from the atrium to the ventricles). The atria contract at the sinoatrial nodal rate, and the ventricles are either asystolic or contract at a ventricular rescue rate (40–60 beats per minute). HR↓ and SV↓ → CO↓

30.2.4 Arrhythmias

Atrial tachycardia/flutter	High atrial rate of non-sinoatrial nodal origin. Not a physiologic rate, therefore decoupled from metabolic demand (see online Video 30.2). HR↑ SV↓ → CO↑ or CO↓
Atrial fibrillation	Chaotic depolarization of the atrium. No atrial hemodynamic input to the ventricles and a nonphysiologic rate is conducted through the atrioventricular node to the ventricles. Ventricular output is decoupled from metabolic demand. Stasis of blood in the atria can result in clot formation and stroke (see online Video 30.3). HR↑ SV↓ → CO↑ or CO↓
Ventricular tachycardia	High ventricular rate decoupled from sinoatrial nodal and atrial activity. This commonly results from a reentrant conduction loop or an ectopic foci (spontaneously beating region of myocardium). Ventricular rate is nonphysiologic, therefore decoupled from metabolic demand (see online Video 30.4). HR↑ SV↓ → CO↑ or CO↓
Ventricular fibrillation	Chaotic depolarization of the ventricles. No organized heart rate (see online Video 30.5). CO = 0

Table 30.1 Recommendation for permanent pacing in acquired atrioventricular block in adults

Class I	Permanent pacemaker implantation is indicated for: 1. third-degree and advanced second-degree AV block at any anatomic level associated with bradycardia with symptoms (including heart failure) or ventricular arrhythmias presumed to be due to AV block (Level of Evidence: C) 2. third-degree and advanced second-degree AV block at any anatomic level associated with arrhythmias and other medical conditions that require drug therapy that results in symptomatic bradycardia (Level of Evidence: C) 3. third-degree and advanced second-degree AV block at any anatomic level in awake, symptom-free patients in sinus rhythm, with documented periods of asystole greater than or equal to 3.0 s or any escape rate less than 40 bpm, or with an escape rhythm that is below the AV node (Level of Evidence: C) 4. third-degree and advanced second-degree AV block at any anatomic level in awake, symptom-free patients with AF and bradycardia with 1 or more pauses of at least 5 s or longer (Level of Evidence: C) 5. third-degree and advanced second-degree AV block at any anatomic level after catheter ablation of the AV junction (Level of Evidence: C) 6. third-degree and advanced second-degree AV block at any anatomic level associated with postoperative AV block that is not expected to resolve after cardiac surgery (Level of Evidence: C) 7. third-degree and advanced second-degree AV block at any anatomic level associated with neuromuscular diseases with AV block, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy, with or without symptoms (Level of Evidence: B) 8. second-degree AV block with associated symptomatic bradycardia regardless of type or site of block (Level of Evidence: B) 9. asymptomatic persistent third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster if cardiomegaly or LV dysfunction is present or if the site of block is below the AV node (Level of Evidence: B) 10. second- or third-degree AV block during exercise in the absence of myocardial ischemia (Level of Evidence: C)
Class IIa	Permanent pacemaker implantation is reasonable for: 1. persistent third-degree AV block with an escape rate greater than 40 bpm in asymptomatic adult patients without cardiomegaly (Level of Evidence: C) 2. asymptomatic second-degree AV block at intra- or infra-His levels found at electrophysiological study (Level of Evidence: B) 3. first- or second-degree AV block with symptoms similar to those of pacemaker syndrome or hemodynamic compromise (Level of Evidence: B) 4. asymptomatic type II second-degree AV block with a narrow QRS. When type II second-degree AV block occurs with a wide QRS, including isolated right bundle branch block, pacing becomes a Class I recommendation (Level of Evidence: B)
Class IIb	Permanent pacemaker implantation may be considered for: 1. neuromuscular diseases such as myotonic muscular dystrophy, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy with any degree of AV block (including first-degree AV block), with or without symptoms, because there may be unpredictable progression of AV conduction disease (Level of Evidence: B) 2. AV block in the setting of drug use and/or drug toxicity when the block is expected to recur even after the drug is withdrawn (Level of Evidence: B)
Class III	Permanent pacemaker implantation is not indicated for: 1. asymptomatic first-degree AV block (Level of Evidence: B) 2. asymptomatic type I second-degree AV block at the supra-His (AV node) level or that which is not known to be intra- or infra-Hisian (Level of Evidence: C) 3. AV block that is expected to resolve and is unlikely to recur (e.g., drug toxicity, Lyme disease, or transient increases in vagal tone or during hypoxia in sleep apnea syndrome in the absence of symptoms) (Level of Evidence: B)

AV atrioventricular, *AF* atrial fibrillation, *LV* left ventricle
Adapted from ACCF/AHA/HRS Guidelines [2]

Table 30.2 NASPE/BPEG classifications for pacing and defibrillation systems

I	II	III	IV
Chamber(s) paced	Chamber(s) sensed	Response to sensing	Programmability/rate modulation
O = none	O = none	O = none	O = none
A = atrium	A = atrium	T = triggered	P = simple programmability
V = ventricle	V = ventricle	I = inhibited	M = multiparameter programmability
D = dual (A + V)	D = dual (A + V)	D = dual (T + I)	C = communication with programmer R = rate modulation

Roman numerals I–IV indicate the position in the coding
NASPE North American Society of Pacing and Electrophysiology, *BPEG* British Pacing and Electrophysiology Group
Adapted from Bernstein et al. [3]

Table 30.3 Pacing and timing abbreviations

AP = Atrial pace
VP = Ventricular pace
AS = Atrial sense
VS = Ventricular sense
AR = Atrial refractory event
VR = Ventricular refractory event
AEI = Atrial escape interval—longest allowable interval between ventricular and atrial event (also called VA interval)
ARP = Atrial refractory period
AV = Atrioventricular
AV interval = Longest allowable interval between atrial and ventricular event
LR = Lower rate—slowest pacing rate allowed
LR interval = Longest period of time allowed before delivery of a pacing stimulus
MS = Mode switch
PAV = Paced atrioventricular interval—longest allowable interval between paced atrial beat and paced or sensed ventricular beat
PMT = Pacemaker-mediated tachycardia
PVAB = Postventricular atrial blanking period
PVARP = Postventricular atrial refractory period
SAV = Sensed atrioventricular interval—longest allowable interval between sensed atrial beat and paced or sensed ventricular beat
TARP = Total atrial refractory period (AV + PVARP)
UAR = Upper activity rate (also called maximum sensor-indicated rate)
UR = Upper rate—fastest pacing rate allowed
UR interval = Shortest allowable interval between paced beats or a sensed and paced beat
UTR = Upper tracking rate—fastest rate the ventricles may be paced in 1:1 synchrony with the sensed atrial rate (also called maximum tracking rate)
VA interval = Time between ventricular and atrial event
VRP = Ventricular refractory period
VSP = Ventricular safety pacing

30.3 Introduction to Implantable Pacing and Defibrillation Systems

For proper function and programming, implantable pacing and defibrillation systems require multiple components as well as external instruments. The implantable portion of the system is typically comprised of the implantable pulse generator (IPG, or pacemaker) or an implantable cardioverter defibrillator (ICD, or defibrillator) and the pacing and/or defibrillation leads. The IPG or ICD is most commonly implanted in a subcutaneous location in the left pectoral region. Depending on handedness, the condition of the upper venous system, the presence of other devices, and/or physician/patient preference, the device may also be placed in the right pectoral region. The device may be placed in a submuscular location in situations where the physician is concerned about either erosion of the IPG or ICD through the skin (most common in thin, elderly, or very young patients) or for cosmetic reasons (to reduce the obvious nature of the device). Another variation is to place the device in an abdominal location. This is commonly done in small children to avoid discomfort and/or interference with the motion of the arm and is of course dependent upon device size. This may also be a more practical device location in association with epicardial leads.

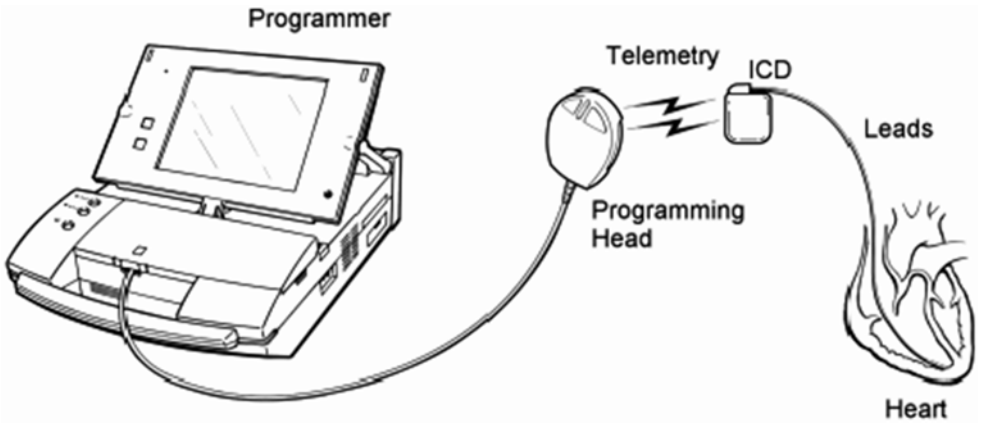

Fig. 30.1 Schematic of a typical implantable defibrillation system and the associated programmer. *ICD* implantable defibrillation device

In support of the implanted hardware, an external programmer is used to noninvasively telemeter information to and from the programmable IPG. This allows the physician to set/reset parameters within the device and download information relating to the status of the patient and the device. A complete defibrillation system is shown schematically in Fig. 30.1 (pacing systems use a similar configuration).

Pacing and defibrillation systems can be implanted using several methods. Early systems used leads attached to the epicardial surface of the heart, with the IPG or ICD placed in the abdomen of the patient (due to their larger sizes). Although this technique is still used in certain clinical situations (i.e., neonates), a transvenous approach for attaching the leads to the heart and a pectoral placement of the IPG or ICD is far more common. The implantation technique for implantable pacing and defibrillation will be described to provide a more thorough understanding of the system requirements.

Following anesthesia and a sterile preparation of the incision site, typically one of two techniques is used to access the venous system for the implantation of transvenous leads. Accordingly, venous access is achieved through either a surgical *cutdown* to the cephalic vein (the jugular vein is also used, but this is rare) or a transcutaneous needle puncture into the subclavian vein. The cutdown involves a careful surgical dissection down to the vessel, placement of a cut through the vessel wall, and direct insertion of the lead into the vessel lumen. The subclavian puncture uses a needle to puncture the vessel, followed by passage of a guidewire through the needle. Subsequently, an introduction catheter (percutaneous lead introducer) with an internal dilator is forced over the wire and into the vein. The dilator is removed, leaving the catheter behind. The lead is then inserted through the catheter (this *Seldinger Technique* can be viewed in online Video 30.6).

Following insertion into the vein, the lead(s) are advanced through the superior vena cava and into the right atrium for final placement in the right atrium, right ventricle, and/or the coronary sinus/cardiac veins (providing access to the left atrium and ventricle). Once positioned properly, the leads are secured in the desired location within the heart using either a passive or active means of fixation (see the section on leads). Next, an anchoring sleeve is used at the venous entry site to secure the lead into the vein and the surrounding tissue. This isolates the lead from mechanical forces outside of the vein, ensuring that adequate lead length remains within the heart to accommodate motion due to activity, respiration, and/or heart motion. Following lead implantation, the proximal terminal ends are connected to the IPG or ICD, which is then placed in a subcutaneous or submuscular pocket formed in the tissue. The implant site is then sutured closed, thus completing the implantation. Chest X-rays of a dual-chamber endocardial pacing system are shown in Fig. 30.2, and additional radiographic images of several pacing configurations are found in online JPGs 30.7–30.12.

30.4 Cardiac Pacing

30.4.1 History

Discoveries relating to the identification of the electrophysiological properties of the heart and the ability to induce cardiac depolarization through artificial electrical stimulation are relatively recent. Gaskell, an electrophysiologist, coined the phrase *heart block* in 1882 and Purkinje first described the ventricular conduction system in 1845. Importantly, Gaskell also related the presence of a slow ventricular rate to disassociation with the atria [4]. The discovery of the bundle of His is attributed to its namesake, Wilhelm His Jr. [5]. He described the presence in the heart of a conduction pathway from the atrioventricular node through the cardiac skeleton that eventually connected to the ventricles. Tawara later verified the existence of the bundle of His in 1906 [6]. He is also credited with being the first to clearly identify the specialized conduction tissues (modified myocytes) that span from the

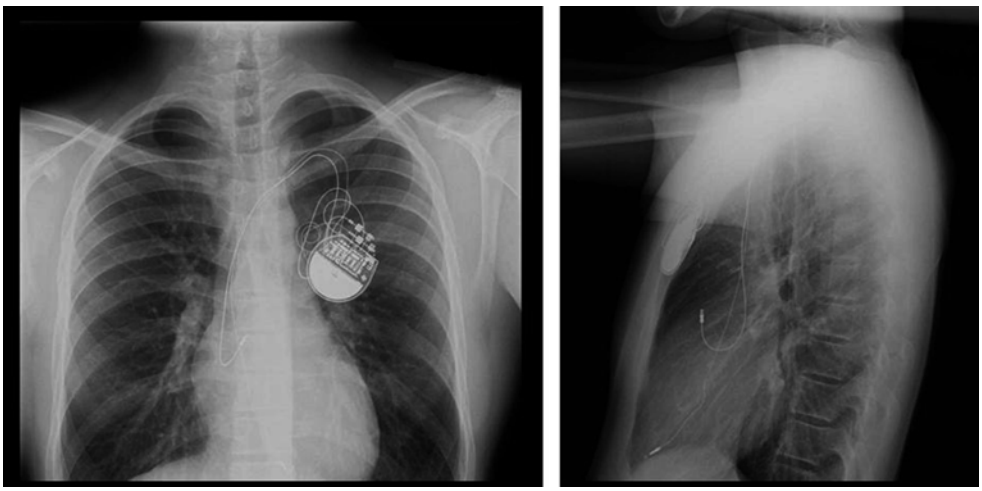

Fig. 30.2 Chest X-rays of an endocardial, dual-chamber pacing system in a young patient (anterior view on the *left*; lateral view on the *right*; Sainte-Justine Hospital, Montreal, QC, Canada used with permission). The implantable pulse generator (IPG or pacemaker) is implanted in the left pectoral region. The superior lead is implanted in the right atrial appendage and the inferior lead is in the right ventricular apex

Fig. 30.3 Dr. C. Walton Lillehei with the first battery-powered, wearable pacemaker

atrial septum to the ventricular apex, including the right and left bundle branches and Purkinje fibers.

The first known instance of electrical resuscitation of the heart was by Lidwell in 1929. Further, Hyman produced the first device for emergency treatment of the heart in 1932. Paul Zoll performed the first clinical transcutaneous pacing in 1952. Importantly for the pacing industry, the first battery-powered pacemaker was developed by Earl Bakken and used in postsurgical pediatric patients by C. Walton Lillehei in 1957 at the University of Minnesota (Fig. 30.3) [4, 7, 8]. Also see Chap. 25.

30.4.2 Artificial Electrical Stimulation

In addition to the spontaneous contraction that occurs within the heart, artificial electrical stimulus (cardiac pacing) can be used to initiate myocardial contraction. This stimulation, in the form of cardiac pacing, is routinely performed as a means to manage patients with cardiac arrhythmias and conduction abnormalities [9, 10]. Pacing induces myocardial contraction through the delivery of an electrical pulse to the patient's heart using an IPG and a cardiac pacing lead. The cardiac pacing lead acts as the electrical conduit for both stimulation and sensing, thus interfacing with the myocardial tissue. The electrical pulse is delivered either in a bipolar mode (involving cathodal and anodal electrodes on the lead) or in a unipolar mode (with a cathode on the lead and the metallic housing of the IPG serving as the anode).

To initiate depolarization, an action potential must be created on a given volume of myocardium. As was described in previous chapters, a normal myocardial cell has a resting membrane potential of approximately –90 mV. The resting membrane potential is dominated by the concentration of potassium (K). A cellular action potential occurs when the resting membrane potential is shifted towards a more positive value (i.e., less negative value) to approximately –60 to –70 mV. At this threshold potential, the cell's voltage-gated Na channels open and begin a cascade of events. In artificial electrical stimulation (pacing), this shift in the resting potential and subsequent depolarization is produced by the pacing system.

Two theories describe the mechanism by which artificial electrical stimulation initiates myocardial depolarization. The *Current Density Theory* states that a minimum current density (amps/cm^3) is required for stimulation of an excitable tissue. The *Electric Field Theory* requires that a minimum voltage gradient (volts/cm) be produced within the myocardium to initiate depolarization [11]. These two theories can, in part, be considered related, since the passage of current through the tissue (Current Density Theory) will induce a potential difference across the cell membranes due to the limited conductivity of the tissue. Similarly, the creation of a potential within the tissue (Electric Field Theory) will also induce a current. Regardless of the theoretical position taken

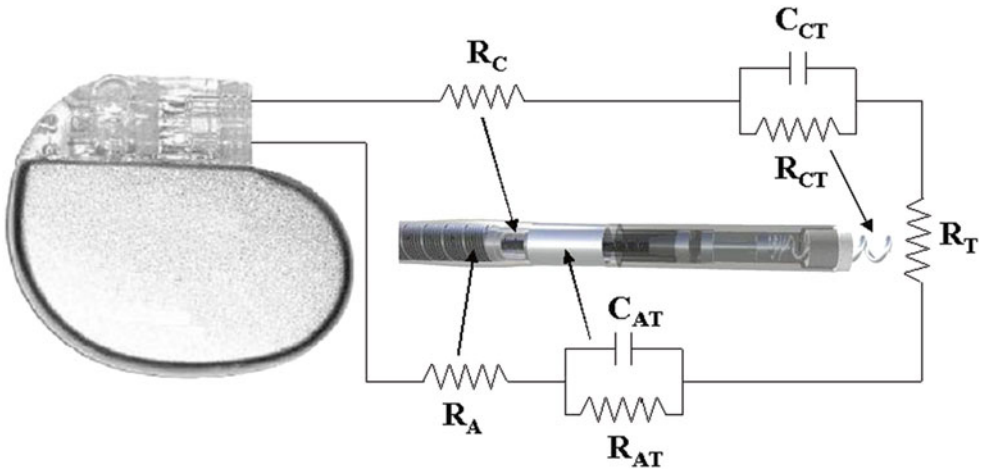

Fig. 30.4 Bipolar pacing circuit, including an implantable pulse generator and a pacing lead. Resistances: *RC* cathodic lead conductor, *RCT* cathode–tissue interface, *RT* tissue, *RAT* anode–tissue interface, *RA* anodic lead conductor. Capacitances: *CCT* cathode–tissue interface, *CAT* anode–tissue interface

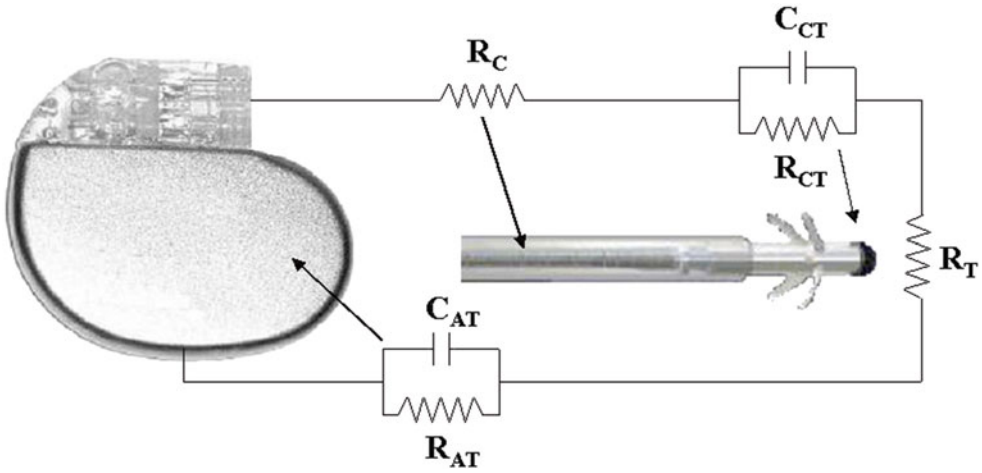

Fig. 30.5 A pacing circuit (unipolar type) which includes an implantable pulse generator and a pacing lead. Resistances: *RC* cathodic lead conductor, *RCT* cathode–tissue interface, *RT* tissue, *RAT* anode–tissue interface. Capacitances: *CCT* cathode–tissue interface, *CAT* anode–tissue interface

regarding stimulation, the requirement for artificial stimulation is the shifting of the resting membrane potential from its normal value (typically –90 mV) towards a more positive value, until the depolarization threshold is reached.

The impedance associated with charge transfer from an IPG to the cardiac tissue is comprised of resistive (R) and reactive components (X_C = capacitive; X_L = inductive):

$$Z^2 = R^2 + \left(X_C + X_L\right)^2$$

The resistive term (R) includes the DC resistance associated with the conductors internal to the lead (R_C = cathodic conductor; R_A = anodic conductor), the cathode–tissue interface (R_{CT}), the anode–tissue interface (R_{AT}), and the tissue itself (R_T):

$$R = R_C + R_{CT} + R_T + R_{AT}$$

The capacitive term (X_C) is the sum of the capacitance of the cathode–tissue interface (C_{CT}) and the anode–tissue interface (C_{AT}).

$$X_C = C_{CT} + C_{AT}$$

The inductance within the conductors and circuit is extremely small and this term is typically neglected. Ignoring inductance, the resulting equation for lead impedance is:

$$Z^2 = R^2 + X^2\mathrm{C}$$

Schematic representations of the circuitry for bipolar and unipolar pacing systems are shown in Figs. 30.4 and 30.5. In these figures, the electric circuit for the delivery of energy to the myocardium is described as a simple RC circuit in which the IPG acts as the voltage/charge source and the lead conductors, electrodes, and cardiac tissue act as the load. Figure 30.4 depicts a bipolar pacing circuit in which the cathode and anode both reside on the pacing lead. Figure 30.5 represents the circuitry associated with a unipolar pacing system. In this case, the circuit is still bipolar but the anode is the metallic housing of the IPG; the term *unipolar* refers to the polarity of the lead.

Typical pacing circuit impedances range from 400 to 1500 Ω. Approximately 80 % of the total impedance is at the tissue interface; as an example, this will result in a 0.8 V drop at the tissue interface when a 1.0 V pacing pulse amplitude is used. Using the aforementioned impedances (400–1500 Ω),

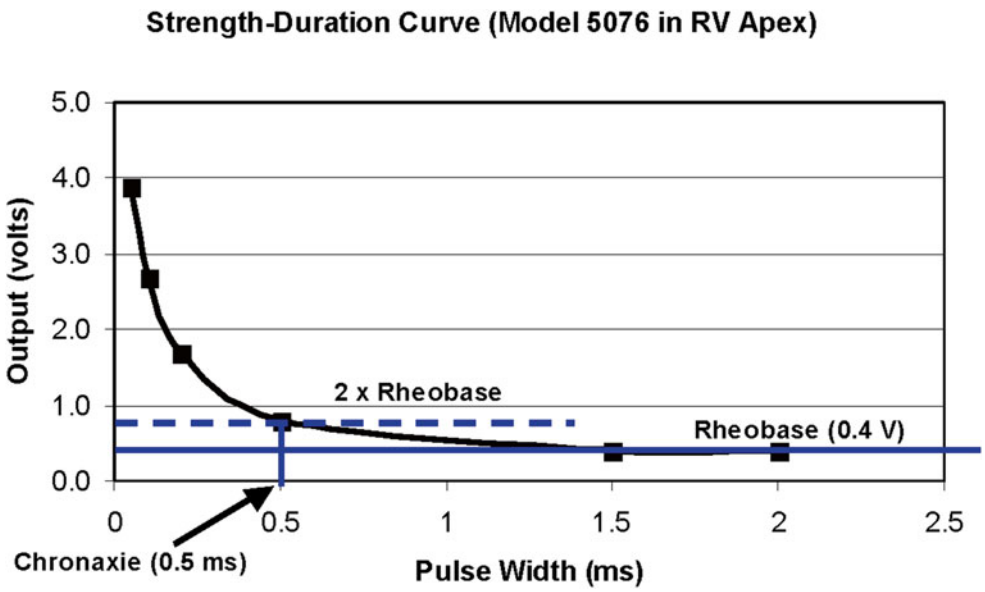

Fig. 30.6 A typical strength-duration curve for cardiac pacing. This particular curve was obtained using a Medtronic model 5076 bipolar pacing lead positioned in the right ventricular (*RV*) apex of a canine. In this plot, chronaxie and rheobase were 0.5 ms and 0.4 V, respectively

a pacing output of 1.0 V produces currents of 2.5 mA and 0.67 mA, respectively.

To date, the most common pacing stimulation waveform used to electrically activate the myocardial tissue is an exponentially decaying square wave. An active recharge is also commonly included at the trailing edge of the stimulation pulse to reduce the post-pace polarization on the electrodes by balancing the charge delivered. The stimulating portion of the waveform is characterized by its amplitude (volts) and width (milliseconds). A relationship exists between the amplitude and pulse width that will be required for depolarization (*pacing*) of the tissue. This relationship, termed a *strength-duration curve* is most commonly plotted as shown in Fig. 30.6.

Terminology relating to the strength-duration curve includes the rheobase and chronaxie values. Rheobase is the threshold voltage at an infinitely long pulse width, and chronaxie is the threshold pulse width at two times the rheobase voltage. The output of a clinical IPG is commonly set at twice the voltage threshold corresponding at the chronaxie pulse width, thus insuring a safety margin [11].

30.4.3 Indications for Pacing

Pacing and defibrillation systems are designed to maintain appropriate cardiac rhythms to maximize the patient's safety and quality of life. With the exception of cases of sudden cardiac death where an external defibrillator is clearly required, the determination of when to use a pacing or implantable defibrillation system can be complex. This section will describe the current classification of indications for pacing and provide a few practical examples of these indications and the decision process associated with choosing the appropriate system for a given patient's condition.

The indications for either pacing or defibrillation therapies are commonly classified in the standard ACCF/AHA/HRS (American College of Cardiology Foundation/American Heart Association/Heart Rhythm Society) format as follows [2]:

Class I	Benefit >>> Risk. Procedure/treatment should be performed/administered.
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.
Class IIa	Benefit >> Risk. Additional studies with focused objectives needed. It is reasonable to perform procedure/administer treatment.
Class IIb	Benefit ≥ Risk. Additional studies with broad objectives needed; additional registry data would be helpful. Procedure/treatment may be considered.
Class III	Classified as "No benefit" (Procedure/test is not helpful with no proven treatment benefit) or "Harm" (Procedure/test is associated with excessive cost without benefit or is harmful and the treatment is harmful to the patient).

For each classification listed above, a Level of Evidence is also referenced: Level A—multiple populations evaluated with data derived from multiple randomized clinical trials or meta-analyses; Level B—limited populations evaluated with data derived from a single randomized trial or nonrandomized studies; and Level C—very limited populations evaluated and only consensus opinions of experts, case studies, or standards of care.

Cardiac pacing can be used for both temporary and permanent management of heart rhythm and function. Although the permanent pacing systems are the most well known, there are numerous indications for temporary pacing. The most common

Table 30.4 Recommendations for permanent pacing in sinus node dysfunction

Class I	Permanent pacemaker implantation is indicated for: 1. SND with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms (Level of Evidence: C) 2. symptomatic chronotropic incompetence (Level of Evidence: C) 3. symptomatic sinus bradycardia that results from required drug therapy for medical conditions (Level of Evidence: C)
Class IIa	Permanent pacemaker implantation is reasonable for: 1. SND with heart rate less than 40 bpm when a clear association between significant symptoms consistent with bradycardia and the actual presence of bradycardia has not been documented (Level of Evidence: C) 2. syncope of unexplained origin when clinically significant abnormalities of sinus node function are discovered or provoked in electrophysiological studies (Level of Evidence: C)
Class IIb	Permanent pacemaker implantation may be considered in minimally symptomatic patients with chronic heart rate less than 40 bpm while awake (Level of Evidence: C)
Class III	Permanent pacemaker implantation is not indicated for: 1. SND in asymptomatic patients (Level of Evidence: C) 2. SND in patients for whom the symptoms suggestive of bradycardia have been clearly documented to occur in the absence of bradycardia (Level of Evidence: C) 3. SND with symptomatic bradycardia due to nonessential drug therapy (Level of Evidence: C)

SND sinus node dysfunction
Adapted from ACCF/AHA/HRS Guidelines [2]

temporary pacing systems utilize transcutaneous wires that are stitched directly into the myocardium and connected to an external stimulator. The stimulator is usually a small portable unit, but it can be a console. Common indications for temporary pacing include: postsurgical heart block, heart block following an acute myocardial infarction, pacing for post- or intra-operative cardiac support, pacing prior to implantation of a permanent pacemaker, and/or pacing during a pulse generator exchange.

The primary indication for the implantation of a permanent pacing system (pacemaker and leads) is to chronically eliminate the symptoms associated with the inadequate cardiac output due to bradyarrhythmias. Typical causes of these bradyarrhythmia are: (1) sinus node dysfunction; (2) acquired permanent or temporary atrioventricular block; (3) chronic bifascicular or trifascicular block; (4) hypersensitive carotid sinus syndrome; (5) neurocardiogenic in origin; and/or (6) a side effect due to a drug therapy. The type of pacing system to be employed is dependent on the nature and location of the arrhythmia, the patient's age, previous medical/surgical history, as well as additional medical conditions.

For conditions related to dysfunction of the sinoatrial node, an IPG with atrial features is commonly used in combination with a lead placed in (or on) the atrium. When management of the ventricular rate is required, a device with ventricular functionality and a ventricular lead are used. When management of the rhythms of both the upper and lower chambers of the heart is required, a dual-chamber system is implanted.

Two clinical situations are outlined below to illustrate common indications for pacing, as well as the decision tree that is often used to determine the type of pacing system for the particular indication. The indications for pacing in a patient with sinus node dysfunction are found in Table 30.4 [2] and the decision tree in Fig. 30.7 [12]. The indications for pacing in an adult with acquired atrioventricular block are found in Table 30.1 and the decision tree in Fig. 30.8 [2]. As an example, a patient with symptomatic chronotropic incompetence would have a Class I indication for pacing (Table 30.4). Since this is related to dysfunction of the sinus node, Fig. 30.7 would then be used to determine the type of pacing system required. In this situation, a rate response system (a pacing system that responds to patient activity/exercise) would clearly be desired. If atrioventricular synchrony were also required, a rate-responsive ventricular pacemaker would be implanted (mostly commonly a DDDR system; see the next section on the standard coding system and Table 30.2).

30.4.4 NASPE/BPEG Codes

In order to describe the function of a pacing system in a standardized manner, the North American Society of Pacing and Electrophysiology (NASPE) and British Pacing and Electrophysiology Group (BPEG) had developed a standard coding system [3]. This code describes the pacing system's functionality using a multi-letter designation. The first four letters are typically used, although this practice is evolving as new pacing features and indications are being developed. In the four letter code system, the first letter indicates the pacing activity (A = atrial pacing, V = ventricular pacing, D = dual-chamber pacing, O = no pacing), the second letter

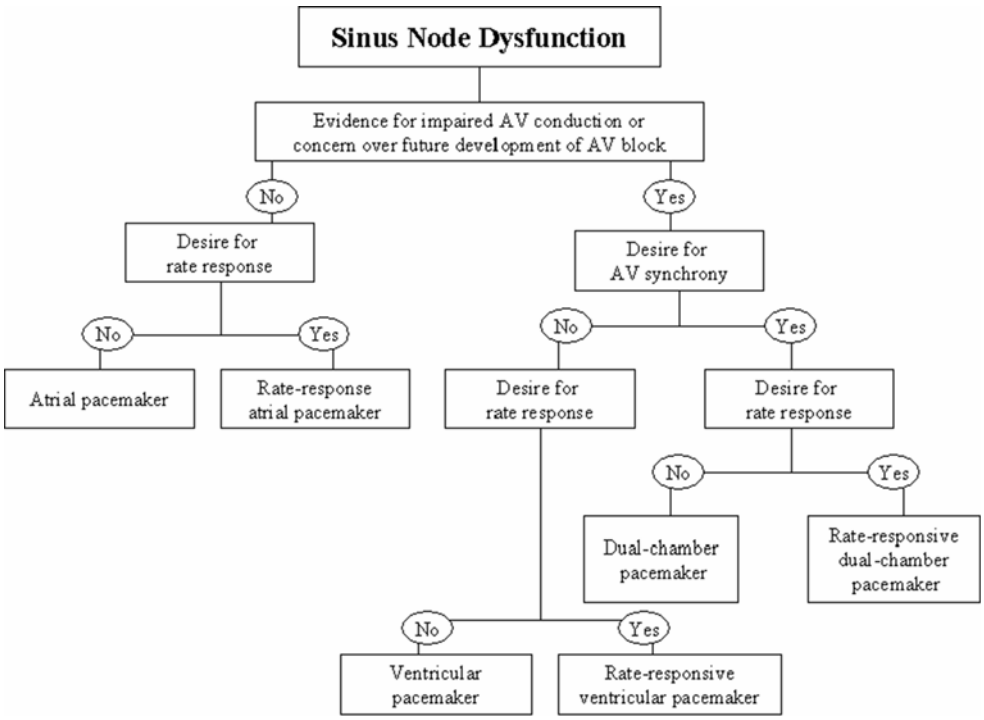

Fig. 30.7 A typical decision tree employed for determining proper therapy when the implantation of a pacemaker for sinus node dysfunction is being considered. *AV* atrioventricular. Adapted from ACC/AHA/HRS Guidelines [12]

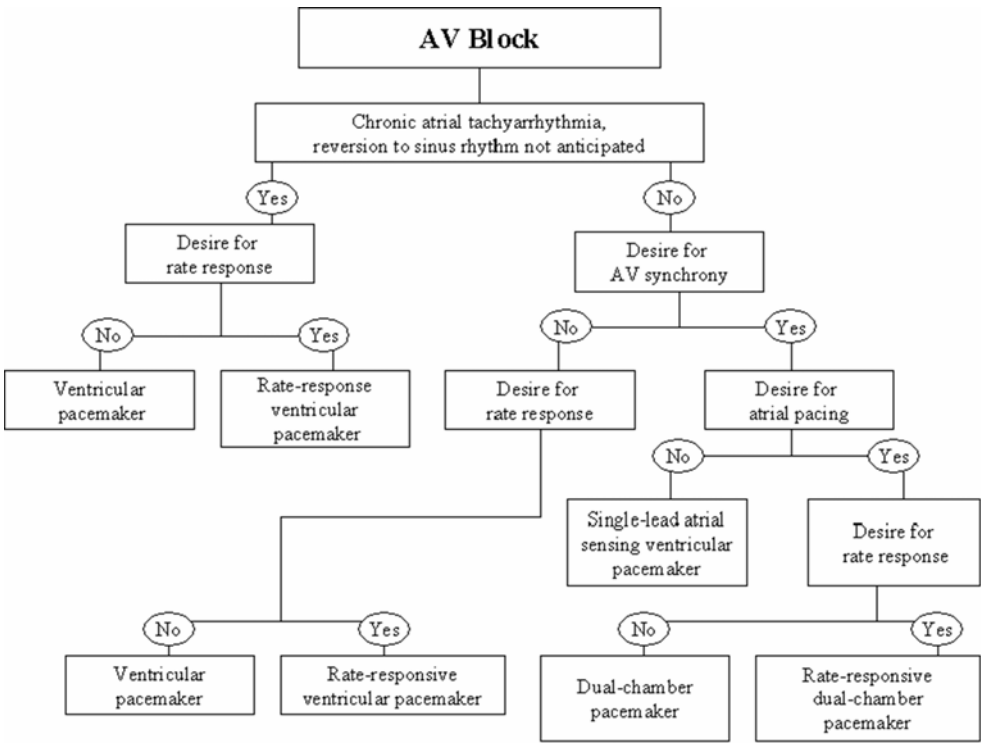

Fig. 30.8 A typical decision tree employed for determining proper therapy when the implantation of a pacemaker for atrioventricular (*AV*) block is being considered. Adapted from ACC/AHA/HRS Guidelines [12]

indicates sensing (A = atrial sensing, V = ventricular sensing, D = dual-chamber sensing, O = no sensing), the third letter indicates the reaction to a sensed event (I = inhibit pacing, T = trigger pacing, D = inhibit and trigger, O = no reaction to sensing), and the fourth letter is used to describe unique device functionality (R = rate responsive, for example). Thus, a VVIR system would pace the ventricles (V---), sense ventricular activity (-V--), inhibit or withhold pacing upon detection of a sensed event in the ventricle (--I-), and provide rate response to manage chronotropic incompetence (---R). See Table 30.2 for a more complete explanation of the coding system.

30.4.5 Implantable Pulse Generators

The IPG is an implantable computer with an integral pulse generator and battery. The componentry is typically encased within a hermetically sealed stamped titanium housing with the battery taking up approximately half of the device volume. The most common battery chemistry used in modern pacemakers is lithium iodide. Device longevity is typically 8–10 years, but may vary significantly depending on system utilization (Fig. 30.9). Electrically insulated feedthroughs connect the internal circuitry to an external connector block, which acts as the interface between the internal circuitry of the IPG and the leads. Typically today, the connector block consists of a molded polyurethane superstructure which houses metallic contacts. The contacts may be simple machined blocks or "spring-type" metallic beams. Most connector blocks employ set screws to ensure permanent retention of the leads and these may also enhance electrical contact. A cutaway view of an IPG can be found in Fig. 30.10, and the scheme for connection between the IPG and the leads is shown in Fig. 30.11 and online Video 30.13. In addition to the standard IS-1 and DF-1 connectors shown in Fig. 30.11, a new standard connection scheme is now available. The so-called *DF-4* connector is an in-line quadripolar connector that includes electrical connections for both the pacing and defibrillation electrode circuits. The new connector is documented in ISO 30186:2010 (Active implantable medical devices—Four-pole connector system for implantable cardiac rhythm management devices—Dimensional and test requirements).

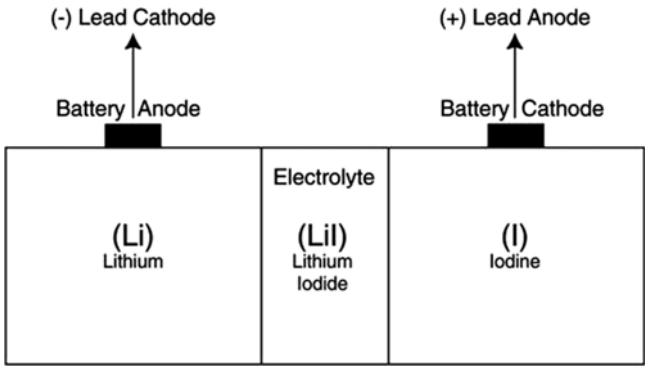

Fig. 30.9 Schematic of a lithium iodide battery. This is the most common chemistry used in modern pacemakers

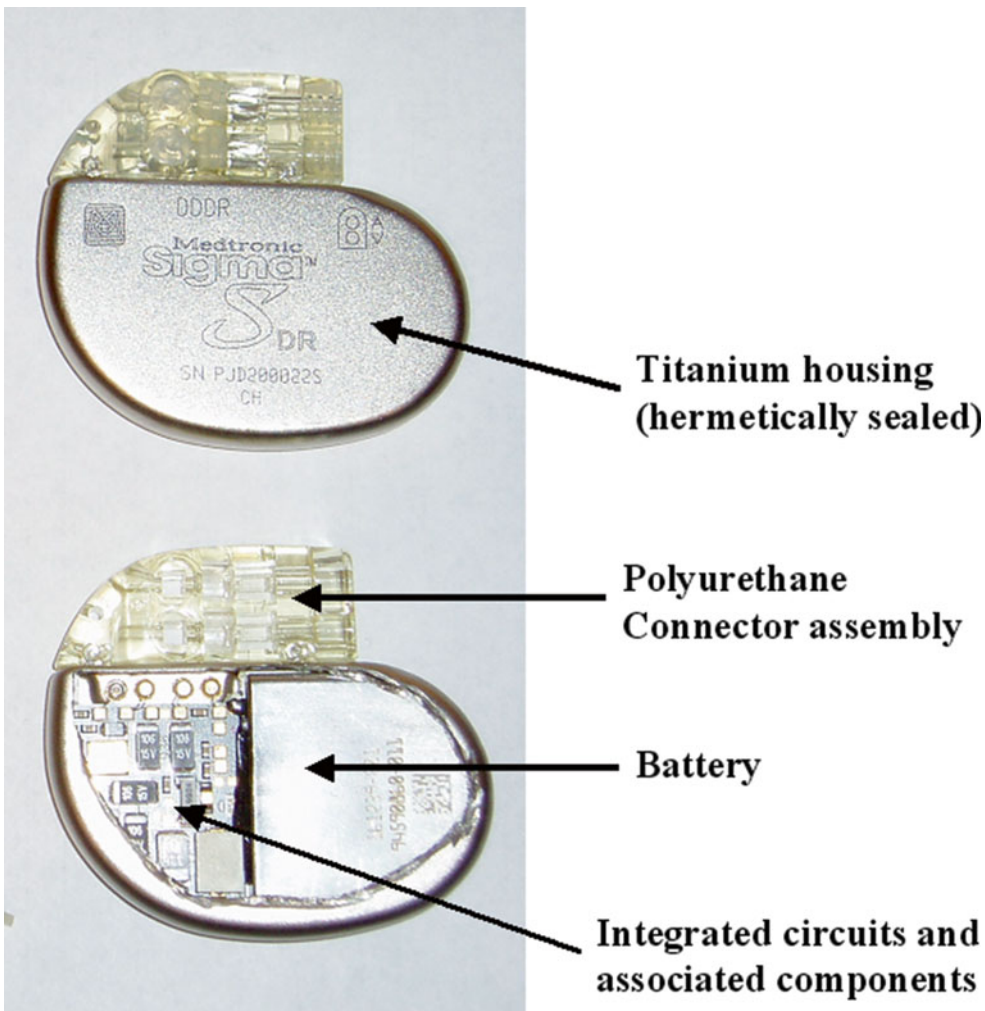

Fig. 30.10 Cutaway view of an implantable pulse generator (IPG or "pacemaker")

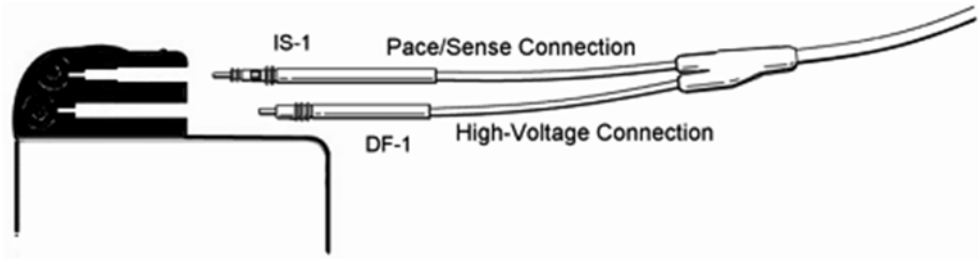

Fig. 30.11 Schematic of the implantable pulse generator-to-lead interface. The IS-1 connector is the standard configuration for pacing. The DF-1 connector is the standard configuration for high-voltage defibrillation (see Video 30.13)

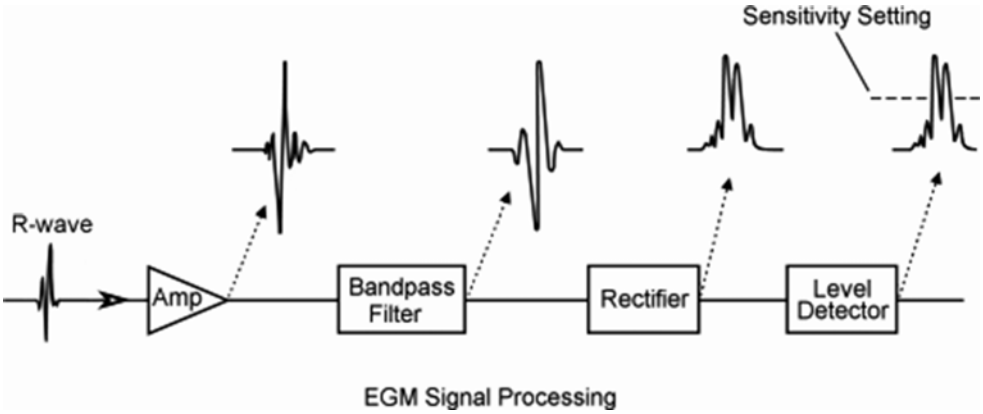

Fig. 30.12 The electrogram amplification and rectification scheme that is used in most modern implantable pacing and defibrillation systems. *EGM* electrogram

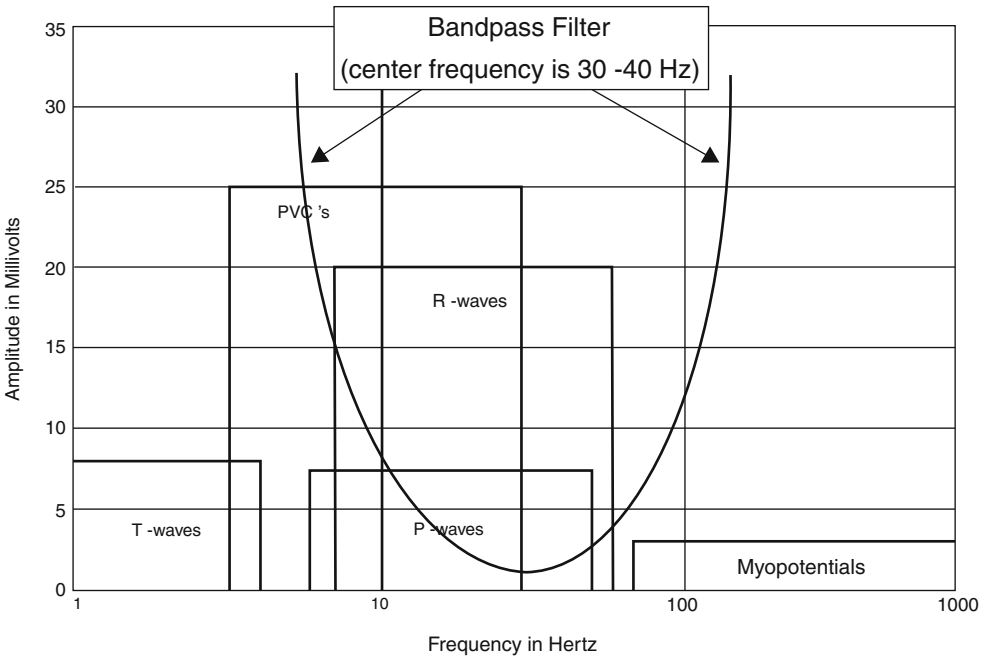

Fig. 30.13 Plot of electrical signals (amplitude and frequency) frequently encountered by pacing and defibrillation sensing algorithms. A bandpass filter for preferential detection of P-waves and R-waves is shown (*parabolic line*). This filter is designed to "reject" myopotentials and T-waves

30.4.6 Sensing Algorithms

In order to assess the need for therapeutic intervention, the pacing system must be able to accurately detect and interpret the various electrical activities of the heart. The instantaneous electrical activity of the heart, or electrogram (EGM), is recorded as a differential voltage measured between the bipolar electrode pair on the lead (bipolar lead) or between the cathode on the lead and the housing of the IPG (unipolar lead). This signal is then processed within the IPG and analyzed by the sensing algorithms. Typically, such signals are amplified, filtered, and rectified prior to undergoing analyses by the device (Figs. 30.12 and 30.13). The resulting signals are then passed through a level detector to determine if they exceed the minimum threshold for detection that was preprogrammed into the device by the clinician. The sensitivity

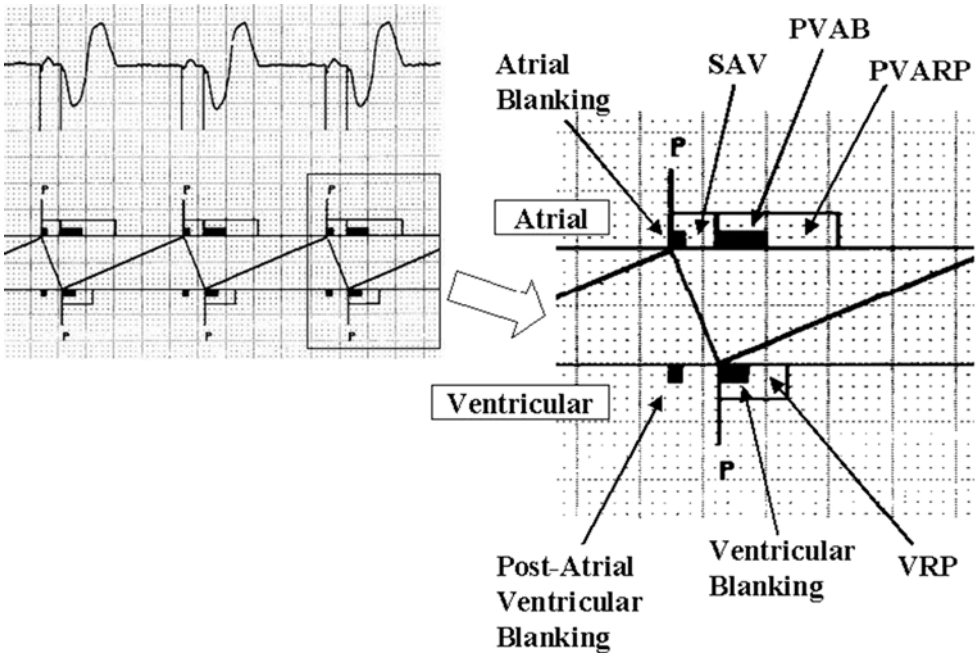

Fig. 30.14 A typical dual-chamber timing diagram, including subdiagrams for the atrial and ventricular channels. The sequence of events begins with a paced atrial beat (P). This paced beat occurs when the maximum allowable interval between sensed atrial events is exceeded. For example, if the minimum rate is programmed to 60 beats per minute, an atrial pace will occur when a 1000 ms interval between sensed events is exceeded. Immediately following this pacing pulse, both the atrial and ventricular sensing algorithms are blanked. This means that the threshold detector ignores all sensed activity. The system is blanked to avoid sensing the resultant atrial depolarization on the atrial channel, and the atrial pacing spike and the atrial depolarization on the ventricular channel. Concurrently in the atrium, a sensed atrioventricular (SAV) interval occurs. This is the longest interval that will be allowed by the device without a paced ventricular beat. The SAV is commonly programmed to 150 ms, and is set to optimize filling of the ventricle due to the atrial contraction. During a cardiac cycle, if the SAV value is reached (meaning an intrinsic ventricular beat does not occur within the programmed interval following the intrinsic or paced atrial beat), a ventricular pacing pulse is then delivered. This pacing pulse is again accompanied by blanking in both channels to avoid oversensing of the pacing pulse and the resultant ventricular depolarization. This interval is referred to as the postventricular atrial blanking (*PVAB*) period on the atrial channel. Concurrently, the postventricular atrial refractory period (*PVARP*) occurs on the atrial channel in which the device attempts to avoid sensing of retrograde P-waves (i.e., atrial contractions conducted through the atrioventricular node in a retrograde manner) and the ventricular refractory period (*VRP*) occurs on the ventricular channel to avoid oversensing of T-waves. Following these intervals, the timing is repeated. If the atrial rate stays above the minimum programmed rate (the lower rate) and the SAV is never reached, the device will never pace unless inappropriate sensing occurs

setting (in mV) determines what is discarded as noise by the algorithm and which signals will be detected. An ideal sensitivity setting is one that will reliably detect the depolarization spike of the chamber (P-wave in the atrium; R-wave in the ventricle) while ignoring repolarization and other physiologic and nonphysiologic signals.

Most rhythm management decisions are based on the heart rate detected. The modern IPG continuously measures the time from one sensed event to the next, and compares the interval to the rates and intervals programmed by the clinician. For example, if two atrial events occur with a separation of 1500 ms (1.5 s), the heart rate is 40 beats per minute (HR = 60/measured beat-to-beat interval; 60/1.5 = 40 beats per minute). In order to understand the logic behind sensing algorithms and pacing timing diagrams, the terminology needs to be introduced. Table 30.3 includes the most commonly used terms and abbreviations. These terms will be freely used in further discussions of the logic behind pacing and defibrillation sensing and therapies without further explanation. This table will also provide the reader with the vocabulary required for interpreting and understanding current literature and publications on the topic.

The decision processes and behaviors of the typical pacing algorithm are usually described using a timing diagram (Fig. 30.14). An understanding of this diagram will provide the basis for analysis of the behavior of pacing systems and will communicate the various parameters that the clinician and device manufacturer must be concerned with. The concepts associated with pacemaker timing are shown in Fig. 30.14; the information is presented in an alternate form in Fig. 30.15.

The actual behaviors of pacing systems can deviate from the ideal for a number of reasons. For example, the pacing pulse can be of an inadequate energy to pace the chamber, losing capture on one or more beats (Fig. 30.16). Another undesired situation that commonly arises is oversensing. In this case, the device inappropriately identifies electrical activities as an atrial or ventricular event (Fig. 30.17). Clinically, oversensing is resolved by reprogramming the

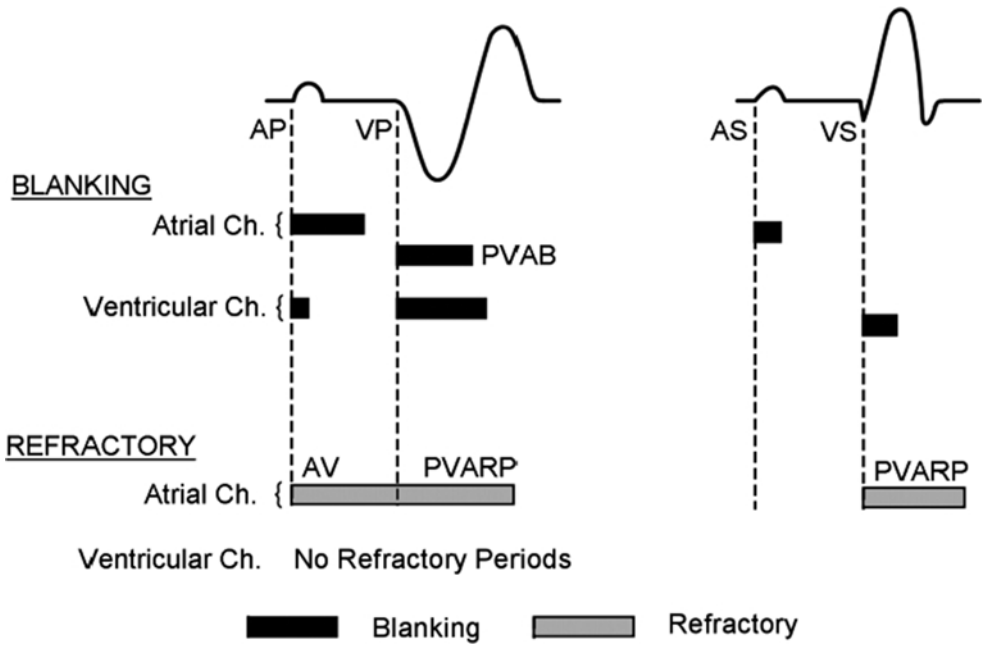

Fig. 30.15 Blanking and refractory periods. The *top trace* represents the electrocardiogram. The portion of the diagram on the left is a situation in which both the atrial and ventricular leads are pacing. The portion on the right is a situation where the system is sensing intrinsic atrial and ventricular activity (i.e., no pacing is occurring). *AP* atrial pace, *AS* atrial sense, *AV* atrioventricular, *PVAB* postventricular atrial blanking period, *PVARP* postventricular atrial refractory period, *VP* ventricular pace, *VS* ventricular sense

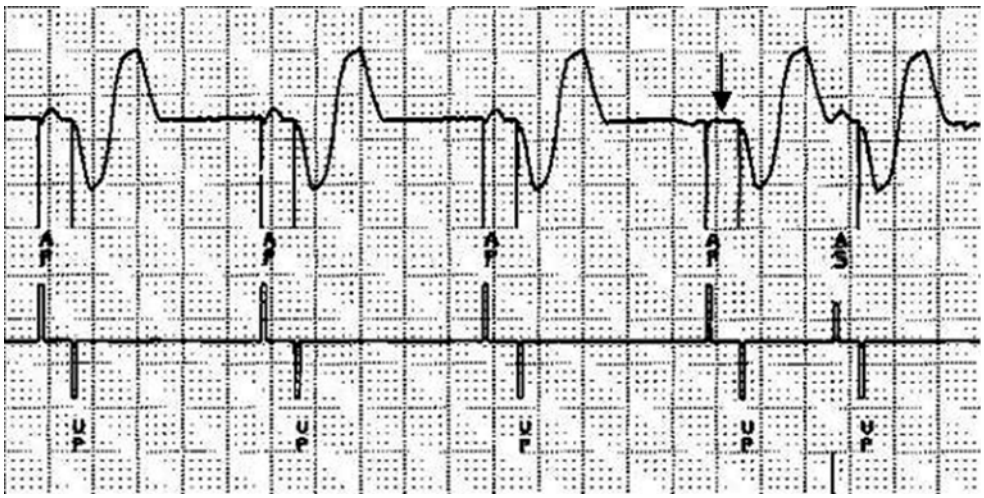

Fig. 30.16 An electrocardiogram (*above*) and pacemaker marker channel (*below*) printed from a programmer. Note the loss of capture on the atrial channel (indicated by the *arrow*); notice that no P-wave follows the pacing pulse

device to a lower sensitivity. Conversely, if a system is undersensing, the sensitivity is increased. Assessment of the behavior of the pacing system is vastly simplified through the use of marker channels. These are shown below the electrograms in both Figs. 30.16 and 30.17. The marker channel is used to report the overall behavior of the pacing system (i.e., documenting how the pacing system interprets the signals transmitted by the device and/or is sensed), allowing a quick assessment of the performance of the algorithms and device output levels.

30.4.7 Drug Interactions with Pacing Systems

It is important to note that certain drug therapies have been reported to impact pacing system performance. Although it is rare for antiarrhythmic drugs to significantly affect pacing thresholds, they have been found to alter stimulation thresholds by inducing changes in the lead-myocardial interfacial conductivity and excitability. Additionally, they can slow the intrinsic sinus rate or atrioventricular rate, which then necessitates pacing of the resultant bradycardia or heart block, respectively. In general, Class Ia antiarrhythmic drugs can increase pacing thresholds at toxic dosages and sotalol and amiodarone, Class III antiarrhythmic drugs can increase pacing thresholds while at therapeutic levels; however, due to the advent of steroid-coated leads and efficient pacing systems, these potential interactions are rarely clinically significant [13, 14]. Rate controlling agents such as beta-blockers, calcium channel blockers, and digoxin decrease the sinoatrial node and atrioventricular rates thereby decreasing heart rate and increasing the PR interval, respectively, which may increase the need for pacing. A summary of the more commonly administered drugs and their impact on

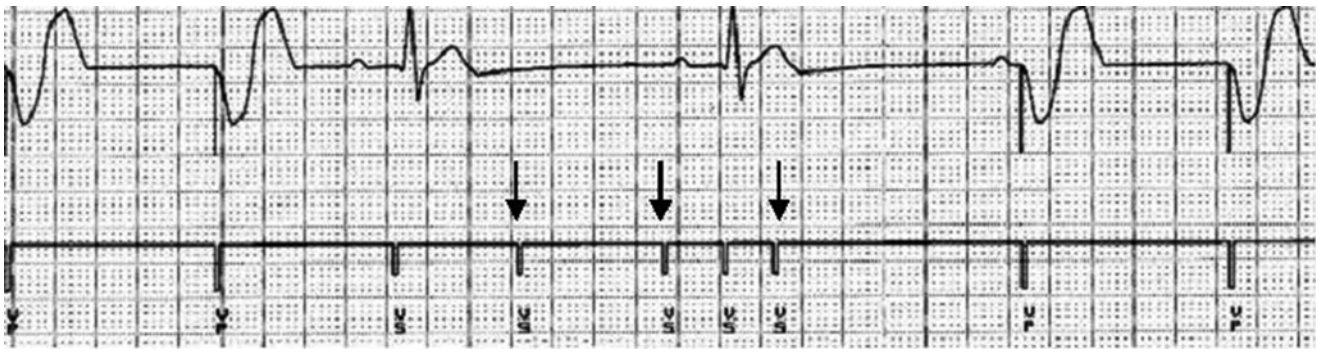

Fig. 30.17 An electrocardiogram (*above*) and pacemaker marker channel (*below*) printed from a programmer. Note the ventricular oversensing (indicated by the *arrow*); notice that no QRS complex is associated with the detected event

Table 30.5 Effect of antiarrhythmic drugs on pacing thresholds

Increase at normal drug levels	Increase at toxic drug levels	No increase
Flecainide	Quinidine	Lidocaine
Propafenone	Procainamide[a]	
Amiodarone	Disopyramide	
Sotalol		

[a]Procainamide, a Class Ia antiarrhythmic drug, is metabolized to *N*-aceyl-procainamide (NAPA) which has Class III activity

pacing thresholds, action potentials, and the physiologic consequences of their action is found in Tables 30.5 and 30.6.

30.4.8 New Indications/Recent Clinical Trials

Today, single- and dual-chamber pacing systems have become the standard method of treating many bradyarrhythmias. Recent clinical evidence has raised interest in the selection of the frequency at which patients are paced and the optimal site of stimulation [15]. It has long been known that pacing produces a nonphysiologic contraction pattern, but recent research has also indicated that potentially detrimental effects may result from long-term pacing [16–19]. Currently, alternate choices in ventricular stimulation sites are of particular interest due to the presumed physiologic and hemodynamic benefits. For example, pacing of the bundle of His is thought to produce a more physiologic contraction pattern, while additional evidence exists that there may also be hemodynamic benefits associated with right ventricular septal and outflow tract pacing [16, 20–25]. In patients with heart failure and associated wide QRS complexes, biventricular pacing has been adopted [26, 27] (see online Video 30.14). Finally, research in atrial pacing has focused on reducing atrial fibrillation, improving methods of pace terminating atrial tachycardias, and/or improving ventricular filling and atrial hemodynamics [28–30]. Recent research is even investigating the possibility of genetically engineering a biologic pacemaker [31].

30.5 Cardiac Defibrillation

Today, sudden cardiac arrest is one of the most common causes of death in developed countries. During 2013, the incidence of in-hospital and out-of-hospital cardiac arrest in the USA was 209,000 and 359,400, respectively. Sudden cardiac arrest claims more lives in the USA each year than the combination of deaths from Alzheimer's disease, assault with firearms, breast cancer, cervical cancer, colorectal cancer, diabetes, HIV, house fires, motor vehicle accidents, prostate cancer, and suicides [32].

Several studies have identified multiple risk factors for sudden cardiac arrest, which include: (1) coronary artery disease; (2) heart failure and/or decreased left ventricular ejection fraction; (3) previous events of sudden cardiac arrest; (4) prior episodes of ventricular tachycardia; (5) hypertrophic cardiomyopathies; and/or (6) long QT syndrome [33]. The combination of any three of these factors significantly increases the risk for sudden cardiac arrest. Ninety percent of sudden deaths occur in patients with two or more occlusions in their major coronary arteries [34].

30.5.1 History

The first documentation of ventricular fibrillation was noted in 1850 [35]. A little over a century later, in 1962, the first direct current defibrillator was developed. Ventricular fibrillation began to be recognized as a possible cause of sudden death in the 1970s and the first transvenous ICD was implanted in the 1990s. Since then, the medical device industry has provided dramatic reductions in ICD size, while

Table 30.6 Antiarrhythmic drugs, action potential phases, and physiologic consequences

Class	Drug	Action potential phase	Physiologic consequence
Ia	Quinidine Procainamide Disopyramide	0	Decreases automaticity of the sodium channel Slows conduction velocity Prolongs refractory period
Ib	Lidocaine Mexiletine Tocainide Phenytoin	0	Decreases automaticity of the sodium channel May or may not slow conduction velocity Decreases refractory period
Ic	Flecainide Propafenone Encainide Moricizine	0	Decreases automaticity of the sodium channel Slows conduction velocity No effect on refractory period
II	Propranolol Atenolol Metoprolol	SA node	Decreases automaticity of nodal tissue Decreases conduction velocity Increases refractory period
III	Bretylium Sotalol Amiodarone Ibutilide Dofetilide Dronedarone	3	Increases refractory period No effect on conduction velocity No effect on automaticity
IV	Verapamil Diltiazem	SA node	Decreases automaticity of nodal tissue Decreases conduction velocity Increases refractory period

SA sinoatrial

simultaneously increasing safety, efficacy, battery longevity, diagnostics, and memory capability. Figure 30.18 shows the evolution in the size of one manufacturer's ICD model.

30.5.2 Tachyarrhythmias

The commonly recognized mechanisms that lead to tachyarrhythmias (tachycardias and fibrillation) include reentry circuits, triggered activities, and automaticity. Reentry is considered as the most common tachyarrhythmia mechanism. It can be described as an electrical loop within the myocardium that has a circular, continuous series of depolarizations and repolarizations (Fig. 30.19). In general, there are three requirements for reentry to occur: (1) the presence of a substrate, for example, an area of ischemia or scar tissue; (2) two parallel pathways which encircle the substrate; and (3) one pathway that conducts slowly and one that exhibits unidirectional block. An impulse reaching the substrate is slowed by the unidirectional block and is allowed to slowly conduct down the slow pathway. As the impulse continues to move around the substrate, it conducts in a retrograde manner up the fast pathway and the impulse continues to conduct in a circular fashion.

Inappropriate atrial or ventricular tachycardias can be further classified as either hemodynamically stable or unstable. The level of hemodynamic compromise that occurs is typically considered to depend on both the rate and the pathway of the arrhythmia. In general, atrial tachycardias usually result in higher ventricular rates due to conduction through the atrioventricular node. As atrial rates increase, the rate conducted to the ventricles may or may not be 1:1, since the atrioventricular node has inherent limitations in its ability to conduct depolarizations. If, however, an abnormal pathway exists from the atria to the ventricles, then 1:1 conduction may be possible even at very high rates. Nevertheless, a patient's clinical risks are related to the level of hemodynamic compromise, with the most extreme case being ventricular fibrillation which, if not immediately reversed, most often results in death.

Triggered activity, or hyperautomaticity, is typically not consistently spontaneous and is a less common mechanism. Early and delayed after depolarizations seen in phase 3 and 4 of the action potential are associated with triggered activity. Automaticity is defined as the ability of the cell to depolarize spontaneously at regular intervals. However, in a diseased heart, often cells will exhibit abnormal automaticity that causes them to depolarize at rates faster than the intrinsic nodal rates.

Common symptoms observed in patients with tachyarrhythmias may include syncopal episodes, palpitations, fatigue, and/or dyspnea. Both invasive and noninvasive diagnostic tools are available for diagnosing tachyarrhythmias. The typical noninvasive procedures include: (1) a thorough patient interview; (2) blood work; (3) a 12-lead ECG; (4) tilt table testing; (5) holter monitoring; (6) exercise stress

Fig. 30.18 The evolution of the implantable cardioverter defibrillator (*ICD*). Dramatic reductions in size have occurred, with simultaneous improvements in longevity, diagnostics, functionality, and memory

Fig. 30.19 Reentrant circuits. *Panel A* = unidirectional block, *Panel B* = slow conduction, *Panel C* = reentry circuit

test; (7) echocardiography; (8) signal average ECG; and/or (9) SPECT/MuGA. Currently, an electrophysiological study using cardiac catheterization and/or insertable cardiac monitors is the most commonly used invasive diagnostic procedure.

Therapeutic interventions to manage tachyarrhythmias have a common objective of affecting the behavior of myocardial cells or the conduction of the electrical impulse in the diseased tissue. They include attempts to correct the underlying complication such as coronary reperfusion in the presence of a myocardial infarction, restoration and maintenance of normal sinus rhythm with antiarrhythmic drugs, use of electrical therapies such as antitachycardia pacing, cardioversion, defibrillation, and lastly, ablation performed surgically or with the assistance of a catheter. The role of medical devices in the management of these arrhythmias will become clear as device function is described in subsequent text.

30.5.3 ICD Indications

As was the case for the pacing indications previously discussed, the indications for an ICD are also complex. The indications by class are shown in Table 30.7 [2].

Table 30.7 Recommendations for implantable cardioverter defibrillators

Class I	ICD therapy is indicated in: 1. patients who are survivors of cardiac arrest due to VF or hemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude any completely reversible causes (Level of Evidence: A) 2. patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable (Level of Evidence: B) 3. patients with syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study (Level of Evidence: B) 4. patients with LVEF less than 35 % due to prior MI who are at least 40 days post-MI and are in NYHA functional Class II or III (Level of Evidence: A) 5. patients with nonischemic DCM who have an LVEF less than or equal to 35 % and who are in NYHA functional Class II or III (Level of Evidence: B) 6. patients with LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF less than 30 %, and are in NYHA functional Class I (Level of Evidence: A) 7. patients with nonsustained VT due to prior MI, LVEF less than 40 %, and inducible VF or sustained VT at electrophysiological study (Level of Evidence: B)
Class IIa	ICD implantation is reasonable for: 1. patients with unexplained syncope, significant LV dysfunction, and nonischemic DCM (Level of Evidence: C) 2. patients with sustained VT and normal or near-normal ventricular function (Level of Evidence: C) 3. patients with HCM who have one or more major risk factors for SCD (Level of Evidence: C) 4. the prevention of SCD in patients with ARVD/C who have one or more risk factors for SCD (Level of Evidence: C) 5. reducing SCD in patients with long-QT syndrome who are experiencing syncope and/or VT while receiving beta-blockers (Level of Evidence: B) 6. nonhospitalized patients awaiting transplantation (Level of Evidence: C) 7. patients with Brugada syndrome who have had syncope (Level of Evidence: C) 8. patients with Brugada syndrome who have documented VT that has not resulted in cardiac arrest (Level of Evidence: C) 9. patients with catecholaminergic polymorphic VT who have syncope and/or documented sustained VT while receiving beta-blockers (Level of Evidence: C) 10. patients with cardiac sarcoidosis, giant cell myocarditis, or Chagas disease (Level of Evidence: C)
Class IIb	ICD therapy may be considered in patients with: 1. nonischemic heart disease who have an LVEF of less than or equal to 35 % and who are in NYHA functional Class I (Level of Evidence: C) 2. long-QT syndrome and risk factors for SCD (Level of Evidence: B) 3. syncope and advanced structural heart disease in whom thorough invasive and noninvasive investigations have failed to define a cause (Level of Evidence: C) 4. a familial cardiomyopathy associated with sudden death (Level of Evidence: C) 5. LV noncompaction (Level of Evidence: C)
Class III	ICD therapy is not indicated: 1. for patients who do not have a reasonable expectation of survival with an acceptable functional status for at least 1 year, even if they meet ICD implantation criteria specified in the Class I, IIa, and IIb recommendations above (Level of Evidence: C) 2. for patients with incessant VT or VF (Level of Evidence: C) 3. in patients with significant psychiatric illnesses that may be aggravated by device implantation or that may preclude systematic follow-up (Level of Evidence: C) 4. for NYHA Class IV patients with drug-refractory congestive heart failure who are not candidates for cardiac transplantation or CRT-D (Level of Evidence: C) 5. for syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias and without structural heart disease (Level of Evidence: C) 6. when VF or VT is amenable to surgical or catheter ablation (e.g., atrial arrhythmias associated with the Wolff-Parkinson-White syndrome, RV or LV outflow tract VT, idiopathic VT, or fascicular VT in the absence of structural heart disease) (Level of Evidence: C) 7. for patients with ventricular tachyarrhythmias due to a completely reversible disorder in the absence of structural heart disease (e.g., electrolyte imbalance, drugs, or trauma) (Level of Evidence: B)

ARVD/C arrhythmogenic right ventricular dysplasia/cardiopathy, *CRT-D* cardiac resynchronization therapy device and defibrillator, *DCM* dilated cardiomyopathy, *HCM* hypertrophic cardiomyopathy, *ICD* implantable cardioverter defibrillator, *LV* left ventricle, *LVEF* left ventricular ejection fraction, *MI* myocardial infarction, *RV* right ventricle, *SCD* sudden cardiac death, *VF* ventricular fibrillation, *VT* ventricular tachycardia

Adapted from Adapted from ACCF/AHA/HRS Guidelines [2]

30.5.4 External Cardiac Defibrillators

External defibrillators have become ubiquitous in most countries worldwide. In addition to traditional use in hospitals and by paramedics, these systems are now commonly found in many schools, public buildings, airplanes, and even in homes. As with ICDs, these systems are used to treat sudden cardiac death. These systems deliver high-voltage shocks (up to 360 joules) directly to the chest of the patient, using either patches or paddles. One electrode is typically placed in the right pectoral region and the second in the left axilla for delivery of this energy (Fig. 30.20).

30.5.5 Implantable Cardioverter Defibrillators

Similar to a pacemaker, an ICD is a self-contained, implantable computer with an integral pulse generator and battery. In addition to providing pacing therapies for bradyarrhythmia and tachyarrhythmia, ICDs also deliver high-energy discharges. The major components of an ICD include: (1) a battery; (2) electronic circuitry and associated components; (3) high-voltage capacitors; (4) high-voltage transformers; (5) a telemetry antenna; (6) a reed switch triggered upon application of a magnetic field; and (7) a connector block. To date, this componentry is most commonly housed within a hermetically sealed stamped titanium case. Feedthroughs connect the internal circuitry to an external connector block, which acts as the interface between the internal circuitry of the ICD and the leads. The connector block is commonly fabricated from a molded polyurethane superstructure, which houses metallic contacts for interconnection with the leads. The contacts may be simple machined blocks or "spring-type" metallic beams. Most connector blocks used today have set screws to ensure permanent retention of the leads. A cutaway view of an ICD can be found in Fig. 30.21.

Fig. 30.20 An external cardiac defibrillator (LIFEPAK® 1000, Medtronic, Inc.)

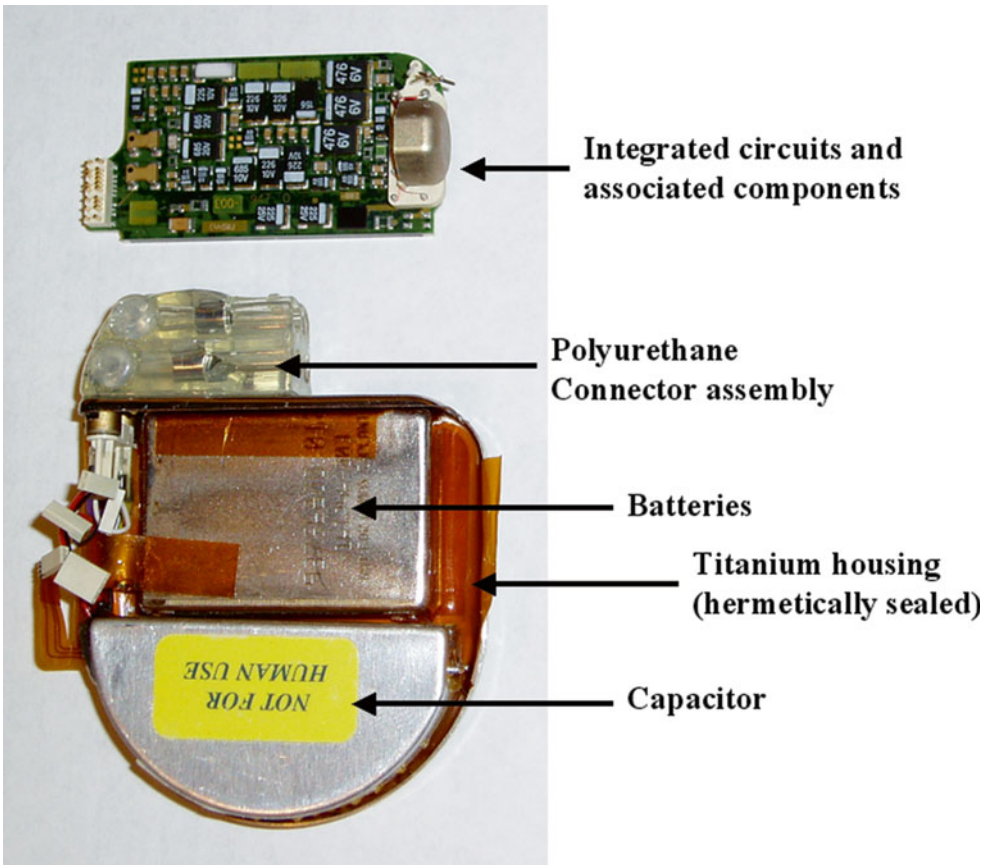

Fig. 30.21 The inner workings of a modern implantable cardioverter defibrillator (*ICD*). A portion of the titanium housing has been removed to expose the typical internal components

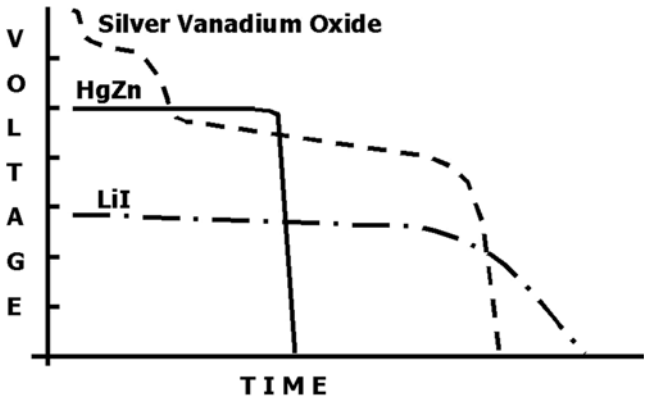

Fig. 30.22 A typical example of an implantable cardioverter defibrillator (*ICD*) depletion curve for a silver vanadium oxide battery. Lithium iodide and mercury zinc batteries included for comparative purposes

Today, most ICDs will use one or two batteries with silver lithium vanadium oxide chemistry. A typical full charge of this type of battery is 3.2 V. As the ICD battery energy starts to deplete, the voltage will follow the path shown in Fig. 30.22, where there are two characteristic plateaus. The voltage is provided to the clinician upon device interrogation to determine if the battery is at the beginning of life, middle of life, elective replacement indicator, or end of life [36]. Each manufacturer and each device will have its own elective replacement indicator voltage. This value is representative of the voltage and current drain from the circuitry, and is also related to the characteristics of the capacitor used. All devices should be replaced before end of life is reached. The longevity of such devices depends on the number of therapies delivered, but is typically between 6 and 8 years.

The primary function of the ICD's capacitor is the accumulation and storage of an adequate amount of energy to shock-terminate a fibrillating heart. As previously mentioned, a typical voltage of an ICD battery is 3.2 V, whereas the capacitor can store up to 800 V (delivering energy of 30–35 joules). Periodic conditioning of each capacitor is required to maintain charge efficiency and therefore guarantee short charge times to allow rapid conversion of the arrhythmia [37]. The materials currently used in the capacitors slowly lose efficiency, especially when they are not used for a period of time due to a chemical decay process. This process (termed *deformation*) is mitigated by conditioning the capacitors (termed *reformation*). Reforming of the capacitor should be performed regularly by charging the capacitor to its maximum capacity and leaving the charge on it until it gradually discharges the energy. Fortunately, reformation can be easily programmed at regular intervals in most modern devices (e.g., every 6 months) without affecting the patient.

30.5.6 Sensing and Detection

It is desirable for an ICD to be able to accurately sense ventricular rhythms that vary in amplitude, rate, and/or regularity, in order to distinguish between normal sinus rhythm, ventricular tachycardia, ventricular flutter, ventricular fibrillation, and/or supraventricular (atrial) arrhythmias (see examples in Fig. 30.23). Current devices adjust their sensitivity on a beat-to-beat basis in order to sense fine waves of ventricular fibrillation and to avoid oversensing of intrinsic T-waves. If an ICD undersenses (misses cardiac activity that it was intended to detect), the device may fail to treat a ventricular tachycardia, which subsequently may accelerate into ventricular fibrillation. If an ICD oversenses, overestimating the cardiac rate, it may deliver inappropriate therapy which will lead to patient discomfort or, more seriously, it may even induce a tachyarrhythmia.

The steps involved in sensing and detection are similar to those discussed previously for the pacemakers. In fact, almost all ICDs on the market today include the pacing algorithms described previously, with additional functionality/logic for detection and management of tachyarrhythmias. Arrhythmia detection typically occurs via the following steps: (1) sense the R-wave or P-waves; (2) measure the interval or cycle length between consecutive beats; and (3) compare the cycle length to prescribed detection zone intervals to classify the arrhythmia (Fig. 30.24). For the sake of simplicity, this chapter will focus on only two detection zones—the ventricular fibrillation and ventricular tachycardia zones. A fibrillation zone is commonly programmed to detect any interval faster than the interval prescribed by the clinician (e.g., 320 ms = 187.5 beats per minute). If a minimum number/percentage of beats is sensed within this interval, the rhythm will be detected as ventricular fibrillation and the device will treat the rhythm using the high-energy shock amplitudes preprogrammed by the clinician.

During the process of arrhythmia detection, the device counts the number of events in each of the detection zones and compares them to prescribed rules in order to classify the arrhythmia. Most ICD designs employ two different counters when classifying whether an arrhythmia is ventricular fibrillation or a ventricular tachycardia. The ventricular fibrillation counter uses a probabilistic approach. Since ventricular fibrillation waves are chaotic and vary in amplitude and cycle length, the device will look for a programmed percentage of cycle lengths to fall within the fibrillation detection zone (e.g., 75 %, Fig. 30.25); if that criterion is met, the device will detect a ventricular fibrillation and deliver the appropriate therapy. Ventricular tachycardias, on the other hand, usually have regular cycle lengths. A consecutive event counter is used which states that a programmed number of

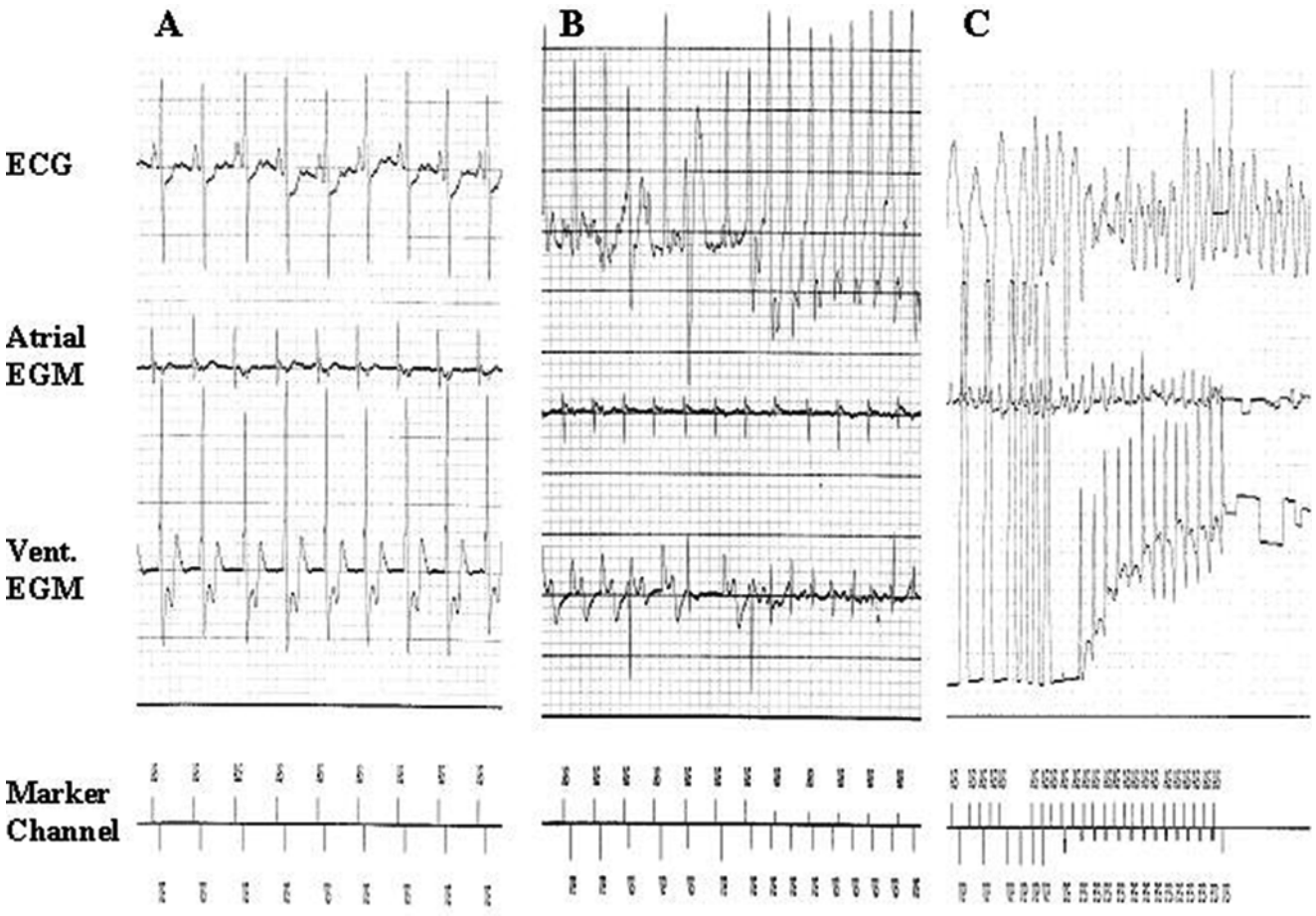

Fig. 30.23 Examples of recorded tachyarrhythmias and the associated device response (refer to the marker channel). *Panel A* = sinus rhythm; *Panel B* = spontaneous ventricular tachycardia; *Panel C* = atrial fibrillation resulting in ventricular fibrillation. *ECG* electrocardiogram, *EGM* electrogram

cycle lengths (e.g., 18 out of 18) needs to be within the tachycardia detection zone in order to classify the rhythm as a ventricular tachycardia. If one cycle length falls out of the tachycardia detection zone, the consecutive counter is reset to zero and the count begins again. Each ICD has the capability to redetect the same arrhythmia if the initial therapy was not successful. Redetection criteria will often be more aggressive (fewer number of beats sampled) than the initial detection criteria to ensure that subsequent therapies can be delivered quickly. An example of when the redetection criteria may not be as aggressive is in cases where the patient has a long QT interval and is prone to developing Torsades de Points which may spontaneously terminate.

Typical devices available today have the option of programming an additional detection zone, which is referred to as a *fast ventricular tachycardia* zone. This is a zone that can be programmed for those patients with a fast ventricular tachycardia who may benefit from antitachycardia pacing. Treating a fast ventricular tachycardia with antitachycardia pacing may decrease the number of high-voltage shocks delivered, increase the patient's quality of life, and prolong device longevity [38]. Evidence of the benefit of this therapeutic approach was seen in the PainFREE Rx trial which concluded that fast ventricular tachycardias with ventricular cycle length less than 320 ms could be terminated by antitachycardia pacing 3 out of 4 times with a low incidence of acceleration into ventricular fibrillation and syncopal episodes [38]. If a fast ventricular tachycardia zone is programmed, the device will always ensure that the most aggressive therapy is being delivered. For example, if a fast ventricular tachycardia is detected, the device will verify that no ventricular fibrillation intervals falls within that fast ventricular tachycardia zone before delivering antitachycardia pacing.

When targeting treatment of ventricular arrhythmias, it is important to verify that the arrhythmia is of a ventricular origin. Therefore, it is common that each manufacturer will have a unique algorithm for distinguishing a supraventricular tachycardia from a ventricular tachycardia. This is very important in order to avoid inappropriate shocking of a

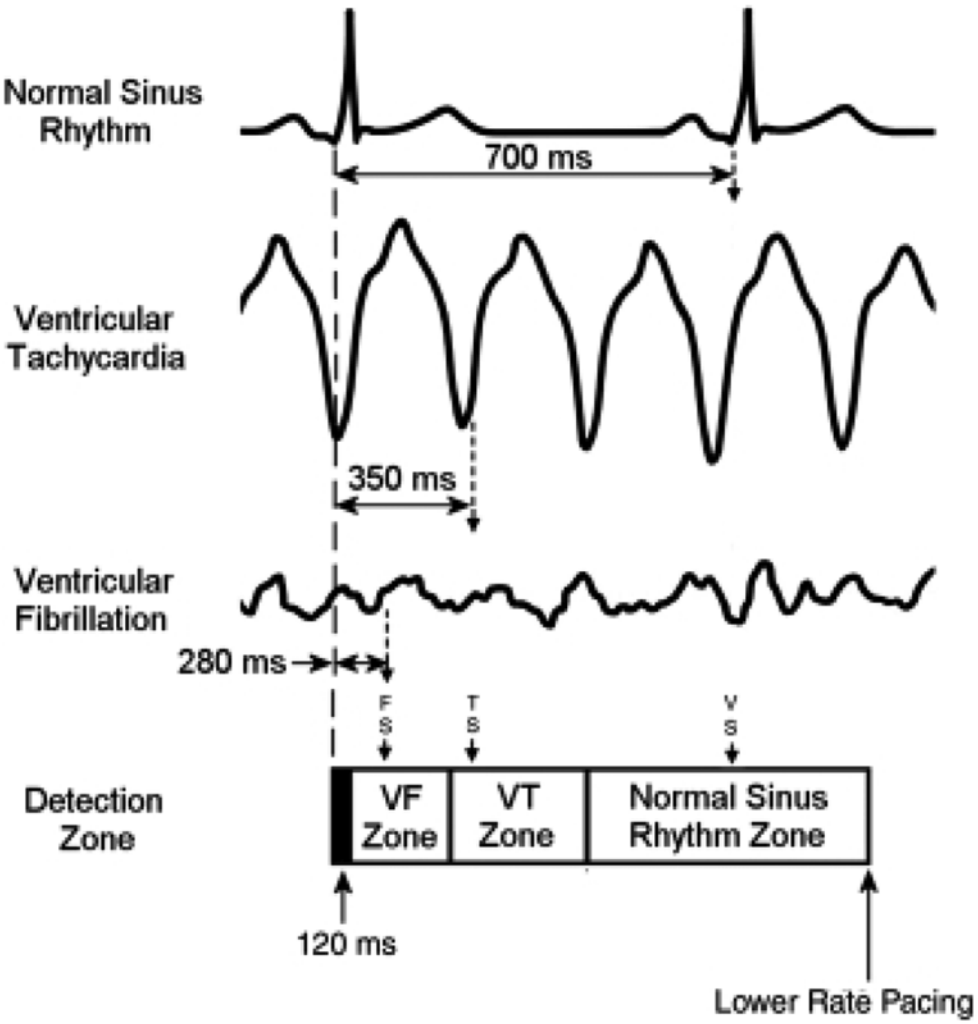

Fig. 30.24 Tachyarrhythmia detection intervals. The *top three traces* represent typical electrocardiograms that might be encountered by the device. The detection zones for ventricular fibrillation, ventricular tachycardia, and sinus rhythm are shown at the *bottom*. Note that an event with a cycle length of 700 ms is categorized as sinus rhythm, 350 ms as a ventricular tachycardia, and 280 ms as ventricular fibrillation. *VF* ventricular fibrillation, *VT* ventricular tachycardia

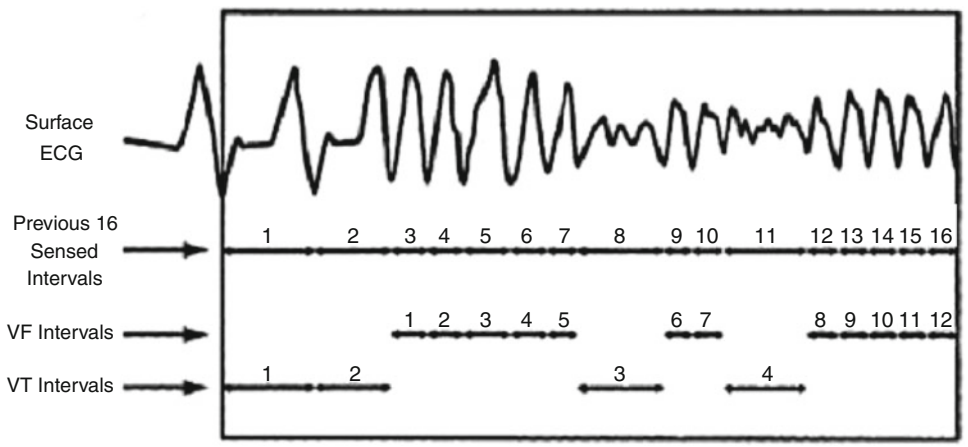

Fig. 30.25 An example of an implantable cardioverter defibrillator (*ICD*) device record including 16 consecutive beats and their classification. Since 12 of 16 events (75 %) were within the ventricular fibrillation detection zone, the arrhythmia would be classified as ventricular fibrillation and high-voltage shocks would be delivered. *ECG* electrocardiogram, *VF* ventricular fibrillation, *VT* ventricular tachycardia

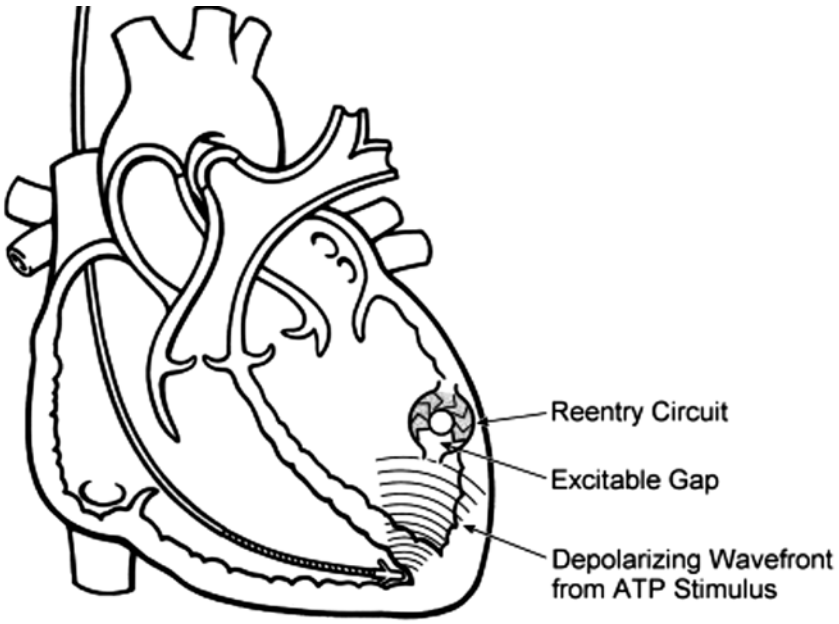

Fig. 30.26 Antitachycardia pacing therapy. A pacing stimulus is applied to entrain an excitable gap in the reentrant circuit. This disrupts the reentrant circuit and terminates the tachycardia. *ATP* antitachycardia pacing

patient with sinus tachycardia due to exercise or an atrial arrhythmia (atrial fibrillation or atrial flutter).

30.5.7 ICD Therapies

ICD therapies are programmed to ensure maximum patient safety, while attempting to deliver the lowest energy therapies (least painful and least impact on device longevity) that will terminate the arrhythmia. ICD therapies can be tiered, such that the device initially delivers low energy which is subsequently increased until the desired treatment is obtained. A typical delivery order is as follows: antitachycardia pacing (delivering the least amount of energy), followed by cardioversion, and finally defibrillation. Nevertheless, each of these therapies can be programmed to the physician's preference.

Antitachycardia pacing is typically used in a clinical situation where one reentrant circuit is repeatedly activating the ventricles and causing a rapid, but regular, ventricular tachycardia. The goal of the antitachycardia pacing therapy is to deliver, via a pacing stimulus, a depolarization wave into the area of the excitable gap (an area of repolarized tissue) of the reentry circuit. Recall that a reentrant circuit causes the majority of tachyarrhythmias. Thus, if a pacing pulse reaches the excitable gap before a new wavefront of the reentrant circuit, the reentrant activity is terminated (Fig. 30.26).

Cardioversion and defibrillation shocks are high-energy shocks that are delivered between two or three high-voltage electrodes, one of which is typically the ICD itself (i.e., the titanium housing acts as an electrode). The goal of these shocks is to defibrillate a critical mass of the myocardial cells

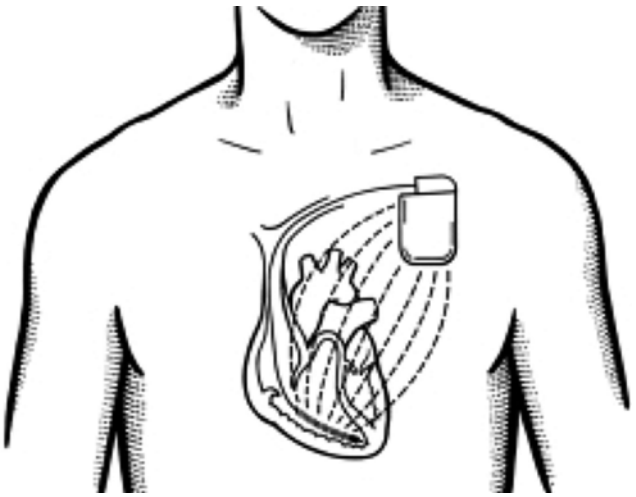

Fig. 30.27 Electric field between high-voltage electrodes during a shock; note that the implantable cardioverter defibrillator (*ICD*) is functioning as one of the electrodes

that are depolarizing at a rapid and irregular rate (Figs. 30.27 and 30.28), thus returning the heart to a normal rhythm.

Cardioversion can be described as a synchronized high-voltage shock because the shock needs to be synchronized to an R-wave or the shock will not be delivered. Cardioversion shocks are used to treat ventricular tachycardias or regular fast ventricular tachycardias. Therefore, the shock is delivered on an R-wave that has been detected in the tachycardia detection zone. If the shock would happen to be delivered on a T-wave, the underlying arrhythmia could be dangerously accelerated into ventricular fibrillation, which is why a cardioversion shock will be aborted if it is not synchronized

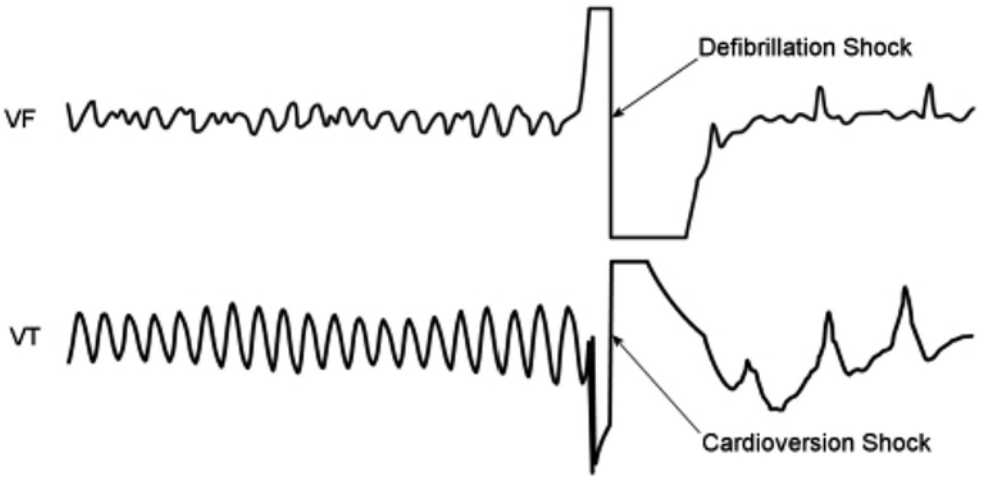

Fig. 30.28 Examples of successful defibrillation (*top electrogram*) and cardioversion (*lower electrogram*) therapies. *VF* ventricular fibrillation, *VT* ventricular tachycardia

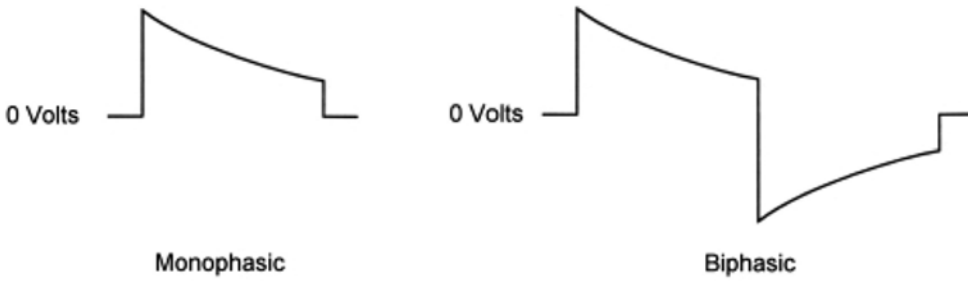

Fig. 30.29 Monophasic and biphasic shock waveforms

to an R-wave. The chaotic nature of ventricular fibrillation is treated by delivering an asynchronous shock. Both cardioversion and defibrillation gain their energy from the discharge of the ICD's high-voltage capacitor.

Depending on the manufacturer, each device will offer a number of programmable therapies per detection zone, again all of which can be programmed to physician preferences. Yet, the programming of the defibrillation therapy is typically based on a specific patient's defibrillation threshold. This threshold is defined as the minimum amount of energy needed to rescue the heart from the fibrillating state (various algorithms exist for determining this energy). The physician commonly will set the first defibrillation therapy at an energy output that is greater than the defibrillation threshold, to provide a margin of safety for the patient. A safety margin of at least 10 joules greater than the defibrillation threshold is common. For example, if a patient's defibrillation threshold has been determined to be 15 joules, the device will be programmed to deliver its first therapy at 25 joules. Therefore, the maximum output of the device needs to be considered when assessing an appropriate safety margin. If a device has a maximum output of 35 joules and the patient's defibrillation threshold is 30 joules, there would only be an 5-joule safety margin.

The relative shape of shock waveforms delivered by the ICD has evolved over time. Early systems used a monophasic waveform delivered between a dedicated set of electrodes (i.e., delivered with a constant direction of current flow or polarity). Later, sequential monophasic shocks between selected pairs of electrodes were employed, since they were found to produce lower defibrillation thresholds in certain patients. Modern devices typically use a biphasic shock that reverses polarity during the discharge of the capacitors (Fig. 30.29).

The development of biphasic waveforms was considered as a significant improvement in ICD technology, and they have been almost exclusively used since the mid-1980s [39]. The percentage of the drop in voltage, prior to termination of the waveform, is the current polarity, also known as *tilt.* Tilt is measured from the instant the current starts to flow in one direction (leading edge) to the time that it ends its flow in that same direction (trailing edge). A tilt value can be measured for each direction that the current is flowing; typical tilts are between 50 and 65 % (Fig. 30.30).

As mentioned previously, most modern ICDs also include pacemaker functionality. As a final summary of the similarities and differences between IPGs and ICDs, Table 30.8 is provided.

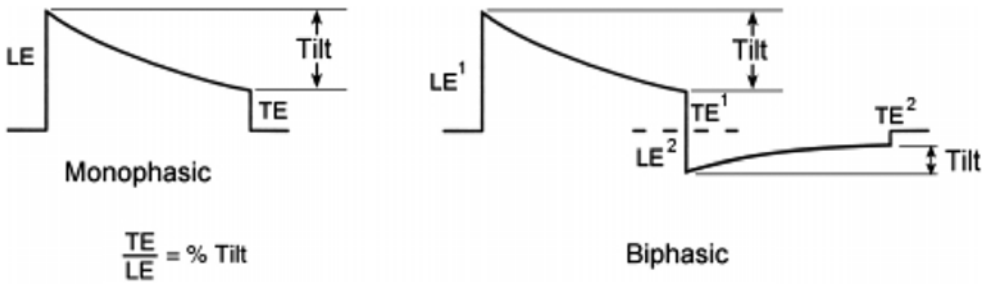

Fig. 30.30 Determination of the percent tilt of a defibrillation waveform. *LE* leading edge, *TE* trailing edge

Table 30.8 Comparison of the principal differences between implantable pulse generator (IPG) and implantable cardioverter defibrillator (ICD)

ICD	IPG
Senses intrinsic rhythms, ventricular tachycardia/ ventricular fibrillation, and prefers to oversense	Senses intrinsic rhythms, and prefers to undersense
Paces and shocks when appropriate	Paces when appropriate
Saves episode data	Rejects signals that occur at high rates
Battery requires high current capability for shocking	Battery optimized for long-term, low current use

30.5.8 Pharmacologic Considerations in the Management of Tachyarrhythmias

In contrast to the relatively small effect that antiarrhythmic drugs typically have on pacing thresholds, the defibrillator threshold of an ICD may be significantly altered when used in conjunction with antiarrhythmic drug therapies. Nevertheless, there are several positive benefits that have been considered useful in the concomitant use of ICDs and antiarrhythmic drugs. For example, antiarrhythmic drugs may act to decrease the frequency and duration of sustained and nonsustained ventricular tachycardia events that would otherwise require a shock from an ICD. In addition, they may also slow the rate of the ventricular tachycardia to increase the efficacy of antitachycardia pacing, decreasing the need for shock therapy. Lastly and importantly, antiarrhythmic agents may lower defibrillation thresholds. Therefore, the use of antiarrhythmic drugs with ICDs can decrease the frequency and/or amplitude of therapeutic shocks, thereby improving patient comfort and prolonging battery longevity [40].

As opposed to the benefits explained earlier, there are also potentially undesired consequences associated with the concurrent use of ICDs and antiarrhythmic drugs [13]. Specifically, antiarrhythmic drugs may: (1) alter the detection of the arrhythmia leading to an increase in the duration of a tachyarrhythmia; (2) increase defibrillation thresholds, making it more difficult to successfully defibrillate the heart; (3) slow the rate of the tachyarrhythmia so much that it no longer falls within the detection zone for both antitachycardia pacing and shock; and/or (4) increase the width of the QRS complex on the EKG, thus causing double counting and inappropriate shocks. The typical antiarrhythmic drugs that may affect defibrillation thresholds are: (1) Type I agents, those with sodium channel blocking activities and the membrane stabilization effects; (2) beta-blockers and calcium channel antagonists due to their effect on the nodal tissues; and (3) Type III agents which may either increase or decrease defibrillation thresholds after long-term therapy (Table 30.9) [14]. Studies have also revealed that the use of illicit drugs, such as cocaine, may increase defibrillation thresholds.

Table 30.9 Impact of select antiarrhythmic drugs on defibrillation thresholds

Increase	Mixed effect	Decrease
Flecainide	Quinidine	Sotalol
Propafenone	Procainamide[a]	Bretylium
Lidocaine	Amiodarone[b]	Dofetilide

[a]Procainamide, a Class Ia antiarrhythmic drug, is metabolized to *N*-aceylprocainamide (NAPA) which has Class III activity
[b]Amiodarone decreases defibrillation thresholds initially but increases defibrillation thresholds with chronic utilization

Antiarrhythmic agents can also be proarrhythmic, which may even lead to an increased requirement for ICD therapies. Predisposing factors to proarrhythmias are: (1) prolonged ventricular repolarization (i.e., prolonged QT wave); (2) electrolyte imbalances such as hypomagnesemia or hypokalemia; (3) underlying ventricular arrhythmias; (4) ischemic heart disease; and/or (5) poor left ventricular function. One of the most dangerous forms of proarrhythmia is considered to be Torsades de Pointes or "twisting of the points." Specifically, Torsades is a rapid form of polymorphic ventricular tachycardia that is associated with delayed ventricular repolarization. It should be noted that both inherited conditions such as long QT syndrome and exposure to Type Ia or Type III

antiarrhythmic drugs that prolong the refractory period on the cardiac action potential put patients at an increased risk of Torsades de Pointes.

30.5.9 New Indications/Recent Clinical Trials

This section will focus on some of the recent clinic trials assessing the value of ICD therapy. Clinical trials serve the important role of assessing therapeutic safety and efficacy for: (1) determining the validity of current clinical indications; (2) discovering new indications for use; and/or (3) driving reimbursement through identification of clinical value. Properly run clinical studies continue to play an important role in continuous improvement of patient outcomes. Yet, an important distinction to make here is that there are major differences between primary and secondary studies. Specifically, primary studies seek to find morbidity and mortality benefit in those patients who have not experienced an event. These studies identify a patient population that is considered "at risk" and attempt to determine means to treat such patients before they experience an event such as myocardial infarction or sudden cardiac arrest. In contrast, secondary studies evaluate post-treatment morbidity and mortality benefits to patient populations that have already suffered from an event (e.g., postmyocardial infarction patients or patients who have survived sudden cardiac arrest).

An example of an important clinical trial associated with the identification of the indications for ICD therapy is the Multicenter Automatic Defibrillator Implantation Trial (MADIT). This trial was instrumental in providing clinical evidence for identifying patients who would benefit from an ICD therapy. The clinical hypothesis stated "in patients with previous myocardial infarction and left ventricular dysfunction, prophylactic therapy with an ICD improves survival versus treatment with conventional medical therapy" [41]. The primary end point of the study was a reduction in total patient mortality, and the secondary end points evaluated mortality-associated with arrhythmias as well as cost-effectiveness. Of 196 patients included in the study, there were 39 deaths in the conventional therapy arm and 15 deaths in the ICD group. The stated conclusions were that, in postmyocardial infarction patients at a high risk for ventricular tachycardia, prophylactic therapy with an ICD reduced overall mortality by 54 % and arrhythmic mortality by 75 % when compared with conventional therapy.

A follow-up to MADIT was the Multicenter Automatic Defibrillator Implantation Trial-II (MADIT-II). The purpose of this study was to investigate the effects of prophylactic implantation of an ICD on the survival of patients postinfarction who presented with significant left ventricular dysfunction (left ventricular ejection fraction $\leq$30 %). The primary conclusion of this study was that prophylactic implantation of an ICD in such patients resulted in improved survival and decreased mortality by 28 % after 3 years. Importantly, the noted benefits of this study have changed practice in that physicians now routinely implant an ICD in postmyocardial infarction patients with left ventricular dysfunction [42].

30.5.10 Pacing and Defibrillation Leads

Cardiac pacing and defibrillation leads are the electrical conduit between the IPG or ICD and the heart. Specifically, they transmit therapeutic energy to the cardiac tissue and return sensed information to the IPG or ICD for diagnostic and monitoring purposes. It is noteworthy that such leads must: (1) withstand the extremely harsh environment of the internal human body and its intense foreign body responses; (2) permanently span multiple anatomic and physiologic features, e.g., the moving body and heart (Fig. 30.31); and (3) undergo approximately 400 million heartbeat-induced deformations over each 10-year period within the heart (see online Video 30.15).

Leads can be placed either endocardially or epicardially, depending on the patient's indication, physician preference, and/or anatomic considerations. In the case of the endocardial pacing systems (those implanted through the venous system to the endocardial surface of the cardiac chambers), the lead travels from subcutaneous tissue including muscle and fat into the blood stream. These leads then pass through the upper vasculature and finally are permanently placed within the beating heart. Today, the vast majority of pacing and defibrillation systems utilize endocardial leads (this lead placement technique can be viewed in online Video 30.6). In contrast, epicardial leads are attached directly to the surface of the heart and are routed through the subcutaneous tissue to the ICD or IPG. Epicardial leads are most commonly used in pediatric patients and in adults with compromised venous accesses to their hearts. Typical implanted configurations for endocardial single- and dual-chamber pacing systems are shown in Fig. 30.32, endocardial defibrillation systems in Fig. 30.33 and online Video 30.16, and an epicardial defibrillation system with epicardial pacing leads in Fig. 30.34.

Modern leads are generally constructed of highly biostable and biocompatible polymers and metals. Configurations for the body of the leads (i.e., the portion traveling from the IPG or ICD to the distal electrodes) are chosen based on the number of circuits required, as well as considerations relating to size, handling, and manufacturer preferences (Fig. 30.35). The electrodes for stimulation and sensing are designed to provide stable electrical performance acutely and chronically.

In order to provide stability at the cardiac–tissue interface, leads often use a mechanism for fixation to cardiac tissue and structures. Passive mechanisms for fixation include

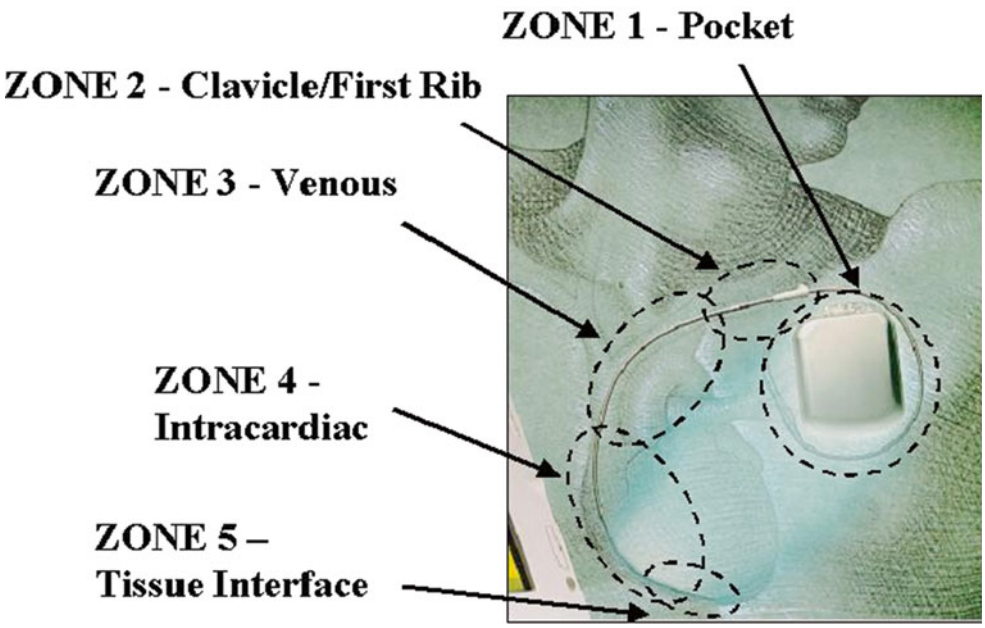

Fig. 30.31 The anatomic regions commonly spanned by transvenous endocardial pacing leads

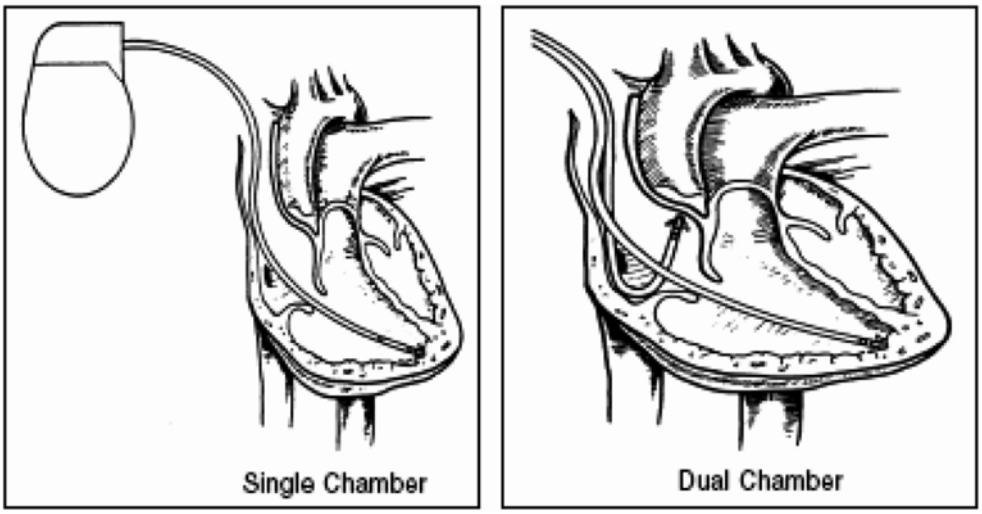

Fig. 30.32 Examples of single- and dual-chamber endocardial lead configurations

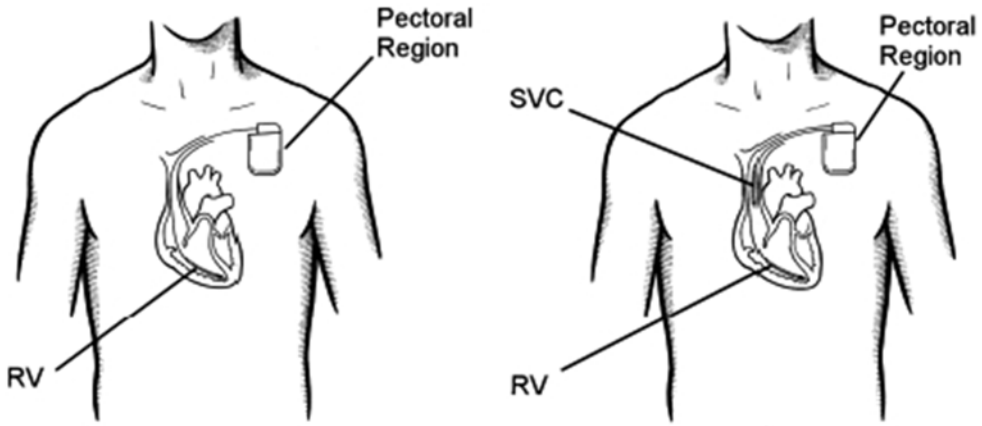

Fig. 30.33 Implanted configurations for two endocardial defibrillation systems with pectoral ICD placements. The single coil system (*left*) delivers the shock energy from the right ventricular coil to the ICD. The dual coil system (*right*) can deliver the energy from right ventricular coil to the ICD, or from the right ventricular coil to a superior vena cava coil and/or the ICD (see Video 30.16). *RV* right ventricle, *SVC* superior vena cava

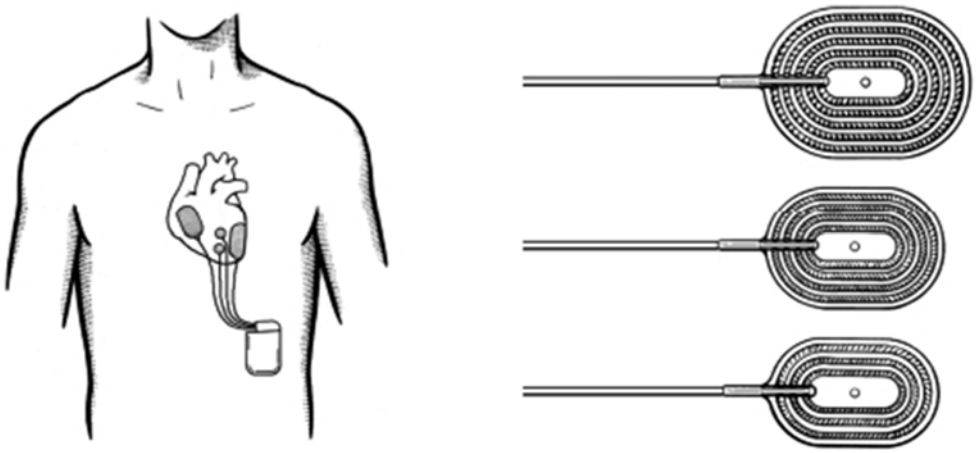

Fig. 30.34 Implanted configuration of an epicardial defibrillation system with an abdominal ICD placement. The system shown includes two unipolar epicardial pacing leads for stimulation and sensing as well as a pair of epicardial defibrillation patches

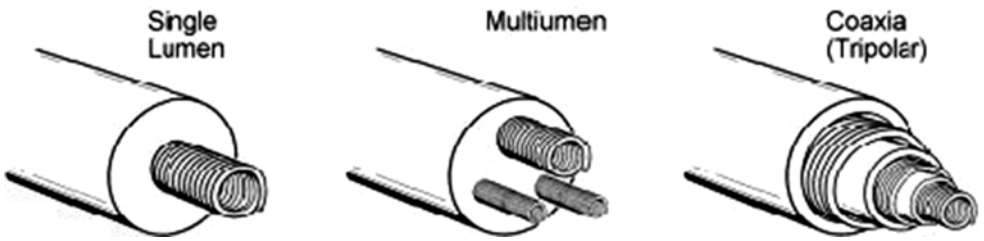

Fig. 30.35 Typical constructions used for cardiac pacing and defibrillation leads: (1) the single lumen design (*left*) has a central conductor surrounded by a polymeric insulation; (2) the multilumen design (*center*) uses an extruded polymer to insulate the conductors from one another and from the implanted environment; and (3) the coaxial design has conductors embedded within concentric layers of insulation. Today, the most commonly used insulation materials are silicones and polyurethanes and the conductors are usually coiled or cabled wires. Modern lead body diameters range from approximately 4–10 French (one French = 1/3 of a millimeter)

polymeric tines and shaped segments along the length of the lead. They are termed *passive* because they do not require an active deployment by the clinician. Common active means of fixation include helices, hooks, or barbs. Additionally, some epicardial leads require sutures to maintain a stable position. Finally, some leads have no fixation means whatsoever and count solely on lead stiffness to maintain locational stability (Figs. 30.36, 30.37, 30.38, and 30.39). To view examples of leads placed within the Visible Heart® preparation, see the following online material: Video 30.17, Video 30.18, Video 30.19, Video 30.20, and Video 30.21.

Various electrode configurations have been utilized on a variety of commercially available leads. As described previously, unipolar pacing circuits use a lead with a single cathodal electrode, with the IPG serving as the anode. Bipolar pacing systems use electrodes placed distally on the lead as both the cathode and anode. Pacing leads commonly use a cylindrical electrode placed along the lead body (ring electrode) as the anode, while defibrillation leads may use a dedicated ring (the so-called *true bipolar* leads) or a defibrillation coil as the anode (an *integrated bipolar* lead). Defibrillation leads utilize electrodes with large surface areas, which allow for the delivery of high-energy shocks within and around the heart. Defibrillation leads may be unipolar (defibrillation electrode only) or they may have a combination of defibrillation electrodes and pacing electrodes. The most common defibrillation lead configurations used today

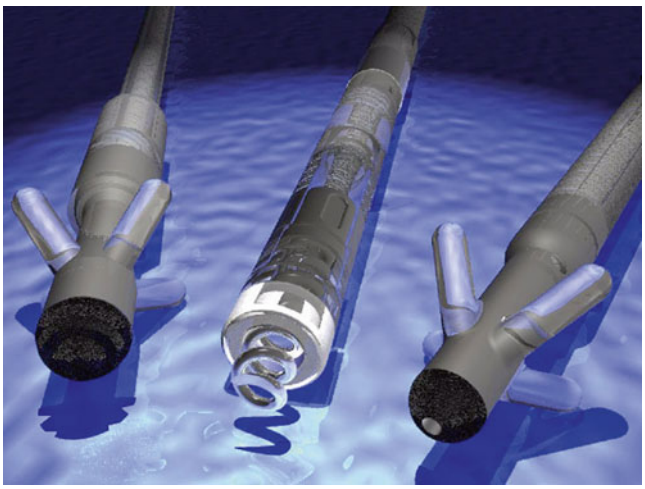

Fig. 30.36 Endocardial pacing leads: passive fixation leads (tined) are shown on the *left* and *right*. An active fixation lead (extendable, retractable helix) is shown in the *center*

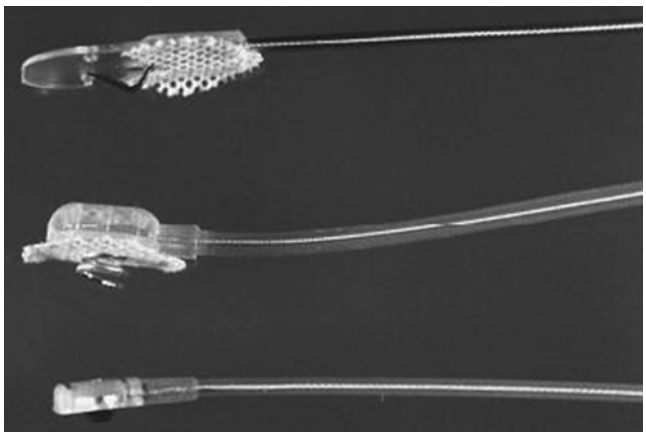

Fig. 30.37 Epicardial pacing leads: stab-in active fixation lead (*top*), active fixation lead with helical fixation (*middle*), and a hemispherical electrode secured by sutures (*bottom*)

are shown in Figs. 30.38, 30.39, and 30.40. For examples of leads placed within the Visible Heart® preparation, see the following online material: Video 30.22 and Video 30.23.

Typically, the portion of the lead that interfaces with the cardiac tissue has been designed to: (1) minimize inflammatory responses; (2) provide low polarizations; (3) provide high capacitances and impedances; and/or (4) act as a fixation mechanism. This distal electrode is most commonly used as the cathode but, in some cases, a similar electrode is used as the anode on a separate unipolar lead. To suppress inflammation, most modern electrodes incorporate a system for the elution of an anti-inflammatory agent (e.g., dexamethasone sodium phosphate); this helps to manage acute changes in the local tissue which will then aid in stabilizing pacing and sensing performance. Coatings are also applied to many pacing electrodes to produce a large surface area that is

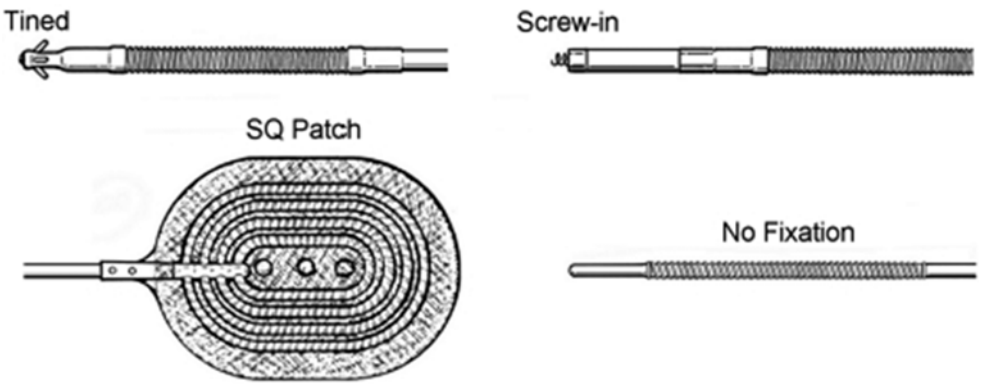

Fig. 30.38 Cardiac defibrillation leads. Clockwise from upper left: a passive fixation endocardial lead ("integrated bipolar"), an active fixation endocardial lead ("true bipolar"), an endocardial lead with no fixation, and an epicardial patch (commonly sewn to the pericardium)

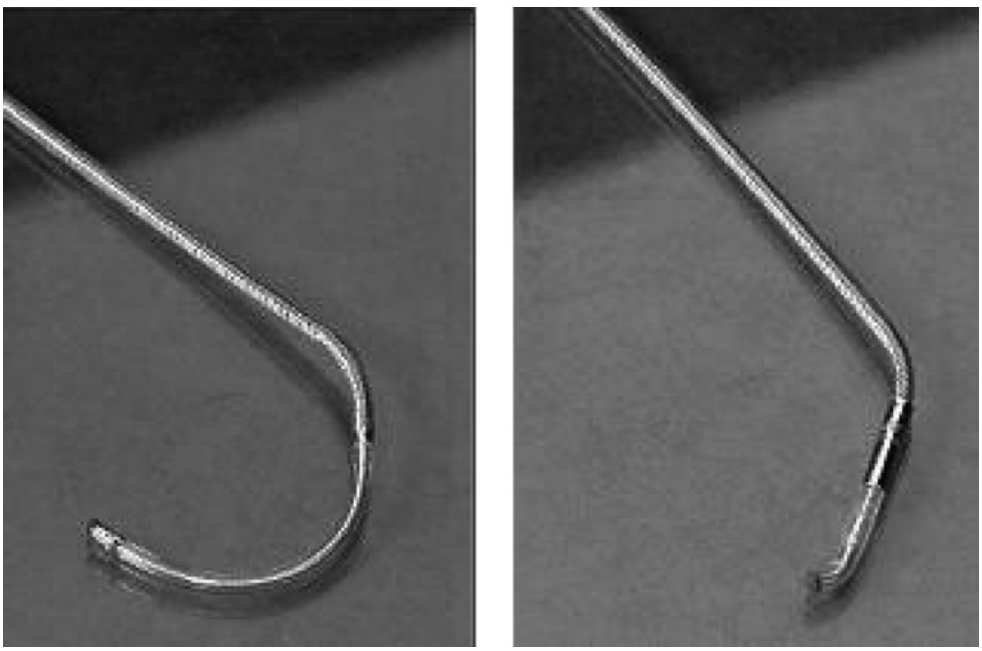

Fig. 30.39 Pacing leads designed for placement in the cardiac veins; they are shaped to enhance stability. The leads shown are primarily used in biventricular pacing systems for the management of heart failure patients with the appropriate clinical indications

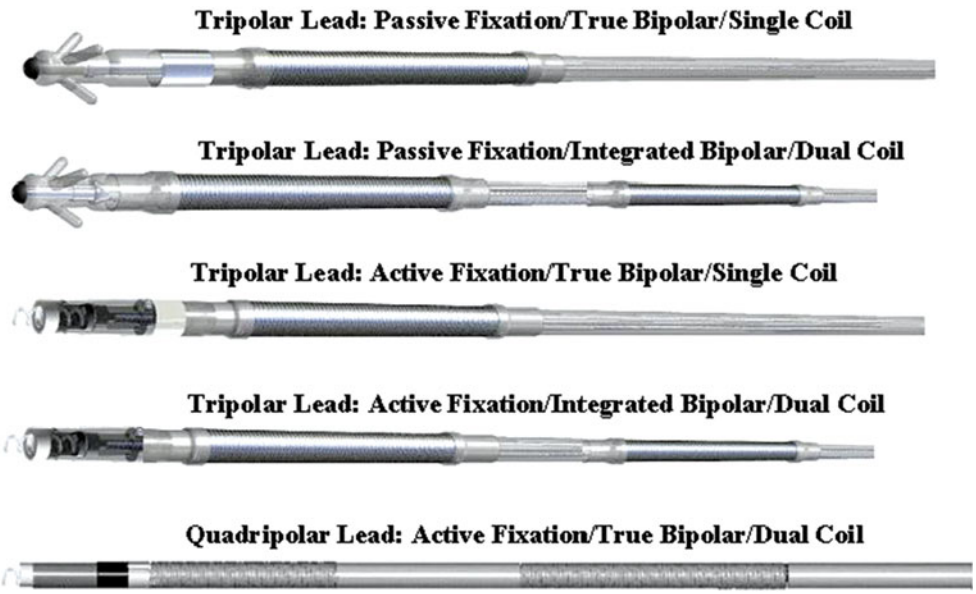

Fig. 30.40 Endocardial defibrillation leads. Various configurations are shown, including leads with active and passive fixation mechanisms, true and integrated bipolar pace/sense circuits, and/or single and dual defibrillation electrodes. The designs shown are typically placed in the right ventricle with the distal defibrillation coil within the right ventricular chamber and the proximal coil located in the superior vena cava

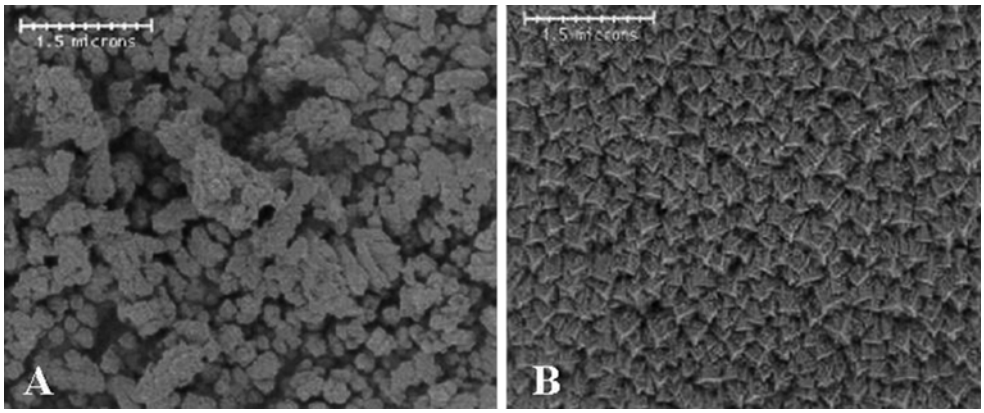

Fig. 30.41 Common electrode coatings for high capacitance and low polarization. The left panel (**A**) shows a platinized surface at 20,000× and the right panel (**B**) a titanium nitride (TiN) surface at 20,000×

highly capacitive (i.e., to reduce battery drain), and have a low level of polarization following a pacing pulse (to avoid undersensing; Fig. 30.41). Interestingly, the size of the pacing cathode has decreased over time, as a means to increase the cathode-tissue impedance and increase system efficiency by reducing current drain (Figs. 30.42 and 30.43) [43].

30.5.11 Leadless Pacing

Although pacing systems with leads have been utilized since the inception of cardiac pacing, recent advances in miniaturization technology and battery chemistry have made it possible to develop a self-contained pacemaker small enough to be implanted entirely within the heart, i.e., while still aiming to provide similar battery longevity as in conventional pacemakers. In general, leadless pacemakers (or transcatheter-delivered pacemakers) are self-contained devices designed to be implanted within the chambers of the heart directly at the site of desired pacing. Further, by eliminating the need for a subcutaneous device pocket and insertion of permanent leads within the vasculature, some of the complications associated with traditional pacing systems can be avoided, including pocket infection/erosion/hematoma and lead dislodgement/fracture/infection.

To date, leadless pacemakers have been developed for both bradycardia and CRT patients. For example, the Micra™ Transcatheter Pacing System (Medtronic, Inc., Minneapolis, MN, USA; not available for sale, but currently under clinical investigation) is a self-contained, percutaneously delivered transcatheter pacemaker (VVIR) that is designed to be implanted in the right ventricle via femoral vein access [44, 45]. The pacemaker is 0.8 cc, 1.76 g, 25.9 mm long, and 6.7 mm in diameter and contains a 3-axis accelerometer used for rate response pacing (Fig. 30.44). In addition, the fixation mechanism consists of four self-expanding nitinol tines which are used to anchor the system within the right ventricle and to stabilize the pacing electrode

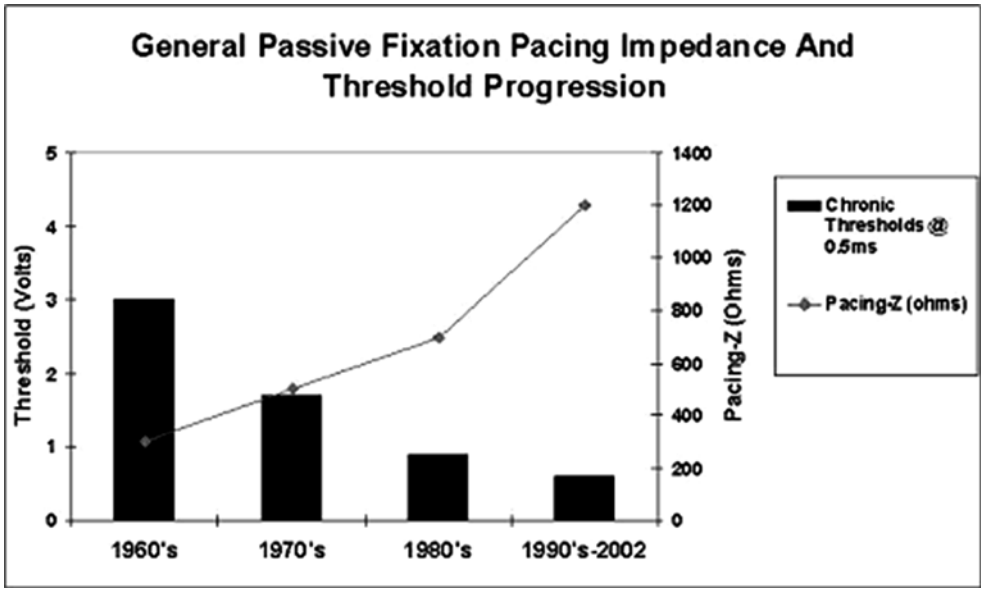

Fig. 30.42 Evolution of pacing lead impedances and pacing thresholds. Modified from Brabec and Laske [43]

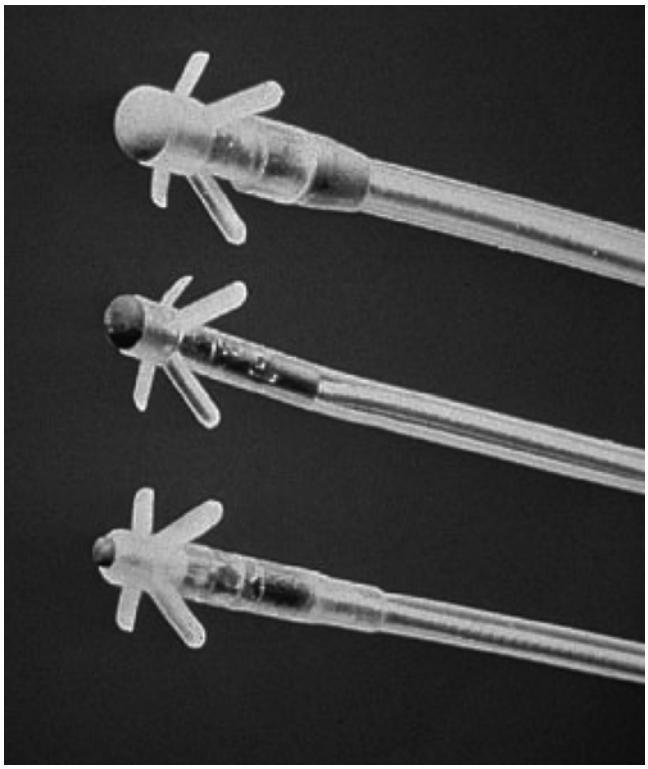

Fig. 30.43 Passive fixation leads with low (*top*; ~400–600 Ω), medium (*middle*; ~600–800 Ω), and high (*bottom*; ~800–1200 Ω) impedance pacing cathodes

Fig. 30.44 Micra™ Transcatheter Pacing System (Medtronic, Inc., Minneapolis, MN, USA; not available for sale, but currently under clinical investigation)

against viable myocardium. This system can be seen implanted in an isolated human heart using direct visualization in online Video 30.24, as published in Eggen et al. [44]. In another recent example, a VVIR leadless pacemaker is implanted in the right ventricle and attached to the myocardium using a helix mechanism (Nanostim, St. Jude Medical, St. Paul, MN, USA); this device has shown promise in the LEADLESS clinical trial [46] and in animal studies [47]. These leadless pacemakers are currently restricted to clinical investigation in the USA. Lastly, a leadless ultrasound-based endocardial left ventricular resynchronization system (WiCSw-LV system, EBR Systems Inc., Sunnyvale, CA, USA) has been developed for heart failure patients [48].

The WiCSw-LV system is a hybrid system which consists of a traditional lead system that stimulates the right atrial and right ventricular chambers, and a transmitter/receiver combination that stimulates the left ventricular endocardium. As such, after activation of the right ventricle by the traditional system, an ultrasound wave is emitted by a subcutaneous transmitter, and the ultrasound energy is converted into pacing energy by a receiver (containing a pacing electrode) implanted in the left ventricle which results in left ventricular stimulation. With these recent developments in pacemaker technology, we can expect the leadless pacemaker to partially eclipse the use of the traditional pacing systems in the near future.

30.6 Summary

This chapter has reviewed the basic methodologies and devices employed to provide pacing and/or defibrillation therapy to the patient with specific needs. A brief history was provided on the use of external electricity to deliver lifesaving therapy to the heart. Although significant progress has been made, future developments in materials, electronics, and communication systems (e.g., wireless) will allow ever-increasing utility and patient value.

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