Implantable Devices for the Treatment of Cardiac Arrhythmia

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The availability of catheter techniques for recording intracardiac electrograms and ablating portions of the myocardium and specialized conducting tissue to modify or terminate cardiac arrhythmia has led to the development of a specialized field of catheterization known as electrophysiology. Although this field was reviewed in previous editions of *Cardiac Catheterization, Angiography, and Intervention*, it has become too complex and specialized for review here. The reader seeking more information about electrophysiology is referred to one of the specialized textbooks in this area (1–3). One area that has been retained, however, concerns the implantation of “devices” for control of the cardiac rhythm. Pacemakers may be implanted in the cardiac catheterization laboratory by cardiologists who perform other invasive procedures and who may have undergone some additional electrophysiologic training. Because of the complexity of programming and testing, implantable cardioverter defibrillators (ICDs) should be implanted only by full-time electrophysiologists.

With increasing sophistication and miniaturization, implanted devices that can monitor and diagnose the cardiac rhythm, pace the atrium or ventricle, and/or deliver a countershock have become first-line (and potentially life-saving) therapy for many arrhythmic disorders. Both pacemakers and ICDs are now small enough to be implanted in the pectoral region through a small incision. In addition to their clear therapeutic potential, the ability of these devices to record, store, and transmit data has made them powerful diagnostic tools.

### DEVICES FOR THE TREATMENT OF BRADYCARDIA: PACEMAKERS

The first electronic pacemaker was a transcutaneous device developed by Zoll in 1952 for the treatment of life-threatening bradycardia (4). With miniaturization of the pulse generator and direct myocardial leads, implantation of the first internal pacemaker was performed in 1958 (5). Compared with these early fixed-rate devices, modern pacemakers have highly sophisticated microprocessors to apply diagnostic and therapeutic algorithms that have dramatically increased their versatility. The relative ease and low risk of implantation make them an attractive choice of therapy in a variety of bradyarrhythmic conditions.

The American College of Cardiology/American Heart Association Task Force on Practice Guidelines, Committee on Pacemaker Implantation periodically publishes guidelines for the implantation of pacemakers (6). The guidelines also address implantable cardioverter-defibrillators (ICDs) and the use of pacing to terminate tachyarrhythmias (discussed in another section).

### Temporary Pacing

**Indications**

Temporary pacing is indicated in any patient with profound bradycardia and hemodynamic compromise and occasionally for prophylaxis when the risk of such an occurrence is high.

The ACC/AHA Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction) has published guidelines for temporary pacing in the setting of acute myocardial infarction (MI) (7). They are
summarized in Table 22.1. In inferior infarction, atrioventricular (AV) block usually occurs in the AV node and is second degree, type I. It usually is transient, though it may last for several days. If third-degree AV block occurs, it usually develops after progressing from prolonged AV conduction (first-degree AV block) and then second-degree AV block. Because of hemodynamically effective escape rhythms originating below the AV node, this type of heart block is often asymptomatic and resolves within a week or less (8). In anterior infarctions, on the other hand, AV block usually occurs below the AV node, may be quite sudden in onset, and often lacks an effective escape rhythm resulting in cardiovascular collapse. Moreover, heart block in anterior infarction is usually a sign of extensive infarction and carries a poor prognosis (9).

A temporary pacemaker may be placed prophylactically during right heart catheterization in the patient with preexisting left bundle branch block, where catheter contact with the right bundle branch may produce complete heart block. Although less common, the same outcome may develop when left heart catheterization is performed in a patient with preexisting right bundle branch block (see Chapter 3).

Other indications for temporary pacemaker placement include the prevention of pause- or bradycardia-dependent tachycardias, or the pace termination of tachyarrhythmias.

Venous Access

Temporary pacemakers can be inserted using any of the right heart catheterization sites discussed in Chapter 4. Careful selection of the access site is indicated, based on the advantages and disadvantages shown in Table 22.2. The femoral site is frequently used in the electrophysiology laboratory or catheterization laboratory, since the need for the temporary pacemaker is usually short-term, sterile technique is easily observed, the patient remains essentially immobile, and fluoroscopy is readily available. In the intensive care unit, however, the internal jugular and subclavian veins are used more commonly, for ease of placement, stability, and longer dwell time without thrombosis or infection. On the other hand, use of a vein that will be needed for a subsequent permanent pacemaker should be avoided (most often the left subclavian vein). Another option is one of the antecubital veins, particularly the basilic vein (the more medial of the antecubital veins), which takes a more direct course to the central vasculature than the cephalic (the more lateral antecubital vein) (see Chapter 5). Antecubital access reduces the risk of bleeding in patients who are anticoagulated or have received thrombolytic therapy, but the patient must keep the arm nearly immobile to minimize the risk of perforation or dislodgment.

Lead Placement

Fluoroscopy is invaluable to guide a standard bipolar pacing catheter into the right ventricular apex, and its use is recommended for temporary lead placement whenever possible. Care must be taken to avoid excess forward pressure on the catheter, which may lead to perforation, especially in the setting of inferior MI with right ventricular involvement. The best location for the catheter tip is along the inferior or septal surface of the right ventricle, about two-thirds of the way toward the apex. Free wall or outflow tract placements carry more risk of dislodgment, perforation, or ectopy, and should be avoided. Once placed in a suitable anatomic location, the capture threshold of the lead should be determined. Good placement should have a threshold of less than 1 mA; if the threshold is higher, consideration should be given to repositioning the lead. The integrity of all the components and connections of the system should be ensured to avoid repeated manipulation of the lead in an attempt to improve capture when the fault is in a connector wire. After adequate pacing has been ensured, the lead and sheath should be secured as well as possible with suture to prevent dislodgment.

If fluoroscopy is not available, placement of a pacing catheter from the femoral or antecubital veins should be avoided. Instead a balloon-tipped catheter pacing lead can be placed via internal jugular or subclavian vein access. The distal tip electrode is connected to the precordial (V) lead of an ECG to allow monitoring of the (unipolar) electrogram as the catheter is advanced (Fig. 22.1). When the distal tip of the catheter is in the atrium, there will be a large atrial electrogram (P wave) with a smaller ventricular electrogram (QRS). When the catheter tip enters the ventricle, however, the ventricular electrogram will grow larger than the atrial electrogram. When the catheter tip touches the endocardium, a current of injury can be recorded, and the catheter should be advanced no further. Alternatively, the electrodes can be connected to the temporary pacemaker pulse generator, which is then activated. As the catheter is advanced into the ventricle, paced QRS complexes appear when contact is made with the
endocardium. This may be particularly helpful in the setting of severe bradycardia or asystole, where observing spontaneous electrical activity may be difficult.

A 12-lead ECG of the paced complexes should always be examined to ensure correlation with presumed position of the catheter. Pacing from the typical right ventricular apical position should produce a wide left bundle branch block/superior axis pattern. Pacing from the left ventricle (via a septal defect, for example), in the cardiac veins, or in the pericardial space will yield a different QRS morphology.

**FIG. 22.1.**

Unipolar electrograms recorded during placement of temporary pacing catheter by clipping lead V₁ to the distal electrode of the catheter. The catheter is being placed via a right internal jugular vein introducer. Lead II is also shown. A: The catheter tip is in the right atrium. The P wave is as large as the QRS complex. B: The catheter tip is in the right ventricle. The amplitude of the QRS complex is markedly increased, and the P-wave amplitude has decreased. C: The catheter tip is in contact with the right ventricular endocardium. The massive ST segment elevation represents a current of injury.

**Temporary AV Sequential Pacing**

Occasionally, patients who need temporary pacing may benefit from preserving AV synchrony, particularly in the setting of right ventricular infarction with heart block. If the only conduction system abnormality is sinus node dysfunction, this may be achieved by atrial pacing alone. If there is concurrent AV node dysfunction, however, maintaining an optimal AV temporal relationship requires placement of both atrial and ventricular pacing catheters. Unfortunately, stable atrial lead positioning is difficult with a passive fixation electrode. Options include the use of a temporary active fixation lead or placement of the catheter in the coronary sinus to allow left atrial pacing. Capture thresholds in coronary sinus pacing are typically higher than those seen pacing a catheter in direct contact with the endocardium.

After placement of the temporary pacemaker is completed, a chest x-ray is indicated to document placement of the lead. Pneumothorax must also be excluded after internal jugular or subclavian vein puncture. The duration of temporary pacing should be minimized (usually < 72 hours) to reduce risks of infection, dislodgment, and perforation. Although in place, the capture threshold of temporary pacemakers should be checked at least twice a day to avoid sudden, unexpected loss of capture. When checking thresholds, the rate of pacing should be gradually reduced to allow for the emergence of an escape rhythm.

**Permanent Pacemaker Implantation**

The guidelines for placement of permanent pacemakers are summarized in Tables 22.3 to 22.11. The guidelines are best understood by considering what benefit may be afforded the patient by implantation of a permanent pacemaker.

1. Alleviation of symptoms
2. Prevention of future morbidity or mortality

Some of the indications are discussed later.

**Alleviation of Symptoms**

*Symptomatic bradycardia* is a general term that encompasses several disorders of the sinus node and the atrioventricular (AV) node. The symptoms of bradycardia are caused by poor organ perfusion in the setting of inadequate cardiac output. They include fatigue, lightheadedness or dizziness, and frank syncope. Bradycardia may also exacerbate myocardial ischemia, causing angina or precipitating congestive heart failure.

Sinus node dysfunction is addressed in Table 22.6 and is one of the most common causes of profound symptomatic bradycardia. The sick sinus syndrome, in which the sinus node fails to generate an adequate heart rate, is an example. In the tachy-brady variant of sick sinus syndrome, profound bradycardia or long pauses usually occur after the termination of a paroxysm of tachycardia (such as atrial fibrillation or atrial tachycardia), due to a prolonged recovery time of adequate sinus node function. This may result in severe lightheadedness or syncope at the time of conversion from the rapid rhythm. Other syndromes include “chronotropic incompetence,” in which an adequate heart rate at rest fails to elevate with exertion or physiologic stress, often leading to decreased exercise tolerance and dyspnea on exertion. This may be due either to dysfunction of the sinus node or to dysfunction of the autonomic nervous system. Inadequacy of the chronotropic response should be confirmed after discontinuation of any unnecessary bradycardic drugs (beta- or calcium channel blockers, for example) prior to implantation of a pacemaker.
Sinus bradycardia itself is rarely an indication for pacing in the absence of symptoms. Highly trained athletes often have baseline heart rates in the 40s or even 30s. Less highly trained individuals may have similarly low heart rates during times of high vagal activity, particularly while sleeping. Before deciding on pacemaker implantation, the bradycardia should be shown to be associated with symptoms. Ambulatory monitoring, especially patient-triggered “cardiac event” monitoring is a useful tool in this regard. The patient whose episodic bradycardia does not correlate with symptoms does not have a clear indication for a pacemaker.

Disorders of AV conduction frequently cause symptoms and are shown in Table 22.3. Prolongation of the AV conduction time is rarely symptomatic but may be so if the PR interval becomes so long that atrial activation occurs during ventricular systole, with the atria contracting against closed AV valves. Patients often feel this as an uncomfortable throbbing in the neck or head. This situation is closely related to “pacemaker syndrome,” which occurs when ventricular pacing is not synchronized to atrial contraction or there is retrograde (ventricular–atrial) conduction that causes the atria to contract against closed AV valves (12)(13). Occasionally, patients develop symptoms when proper timing of atrial and ventricular contraction is disturbed, and loss of the atrial contribution to ventricular filling reduces cardiac output. Second- or third-degree AV block may also cause symptoms, depending on the rate of the infranodal escape mechanism. Higher degrees of heart block, even when asymptomatic, may be indications for pacing to reduce morbidity and mortality (see later discussion).

Prevention of Morbidity and Mortality

Some conditions may produce no immediate symptoms but herald the possibility of severe events.

Type II second-degree AV block and third-degree AV block are usually caused by disease in the His-Purkinje system, and type II second-degree AV block may progress unpredictably to third-degree block (14). Since third-degree AV block is likely to have an unreliable ventricular escape mechanism, there is a risk of profound bradycardia as well as pause-dependent tachyarrhythmias. Therefore type II second- and third-degree heart block, even in the absence of symptomatic bradycardia, is considered an indication for pacing (Table 22.3). On the other hand, type I second-degree (Wenckebach) AV block usually occurs in the AV node, does not progress to higher-degree AV block, and is therefore not considered an indication for pacing in the absence of symptoms.

Table 22.4 shows indications for pacing as related to the manifestations of a diseased His-Purkinje system on ECG. Block of a single fascicle without symptoms should not be considered an indication for pacemaker implantation. Bifascicular and trifascicular block refers to evidence of impairment of two or three of the three fascicles below the His bundle. There may not be complete conduction block in all three fascicles at all times (equivalent to third-degree AV block), but there may be conduction delay or intermittent block. Chronic bifascicular block progresses only slowly to third-degree AV block and is not usually an indication for permanent pacing unless it is accompanied by additional evidence of compromise (a markedly prolonged His-to-Ventricle (HV) interval, abnormal infra-His block during electrophysiology study, or syncope likely to be due to intermittent third-degree AV block; that is, it cannot be attributed to any other cause).

The need for permanent pacing after acute MI is related to the site of the MI, its extent, and the type of block. The need for temporary pacing during the acute phase of a MI is not in itself an indication for permanent pacing (Table 22.5) (6)(7). This is particularly true in inferior MI, where AV block is more likely to be related to intranodal block and is usually transient, lasting a few days (15–18). In anterior MI, AV block is usually related to damage to the conduction system in the setting of a large infarct. The AV block is a marker for the extent of infarct and is rarely a cause of death in itself (19).

Permanent pacing may also be indicated to prevent the morbidity caused by pause-dependent tachyarrhythmias (Table 22.7) (20),(21). Pacing at rates faster than the intrinsic sinus rate shortens the action potential duration and reduces after-depolarizations, possibly decreasing the risk of arrhythmia induction (22),(23).

Neurally Mediated Syncope

Neurally mediated syncope is a particularly difficult problem, since the symptoms may be the result of bradycardia or vasodilation. Such patients frequently have a cardioinhibitory component, and bradycardia is common (24). Since vasodilation is also a part of the syndrome, however, pacing may not prevent syncope. Pacemaker manufacturers have devised algorithms that attempt to avoid sudden decreases in the heart rate and overcome any vasodepressor activity by pacing at fast rates (see the following section entitled “Hysteresis”). Carotid sinus hypersensitivity in patients with syncope is generally considered an indication for pacing (Table 22.8). Head-up tilt-table testing may also be helpful, but its use is severely limited by poor reproducibility and lack of sensitivity and specificity (25),(26).

Other Indications and Potential Indications

Some novel indications, not always associated directly with bradycardia, have received recent attention.
One group of patients who may find symptomatic benefit from pacing in the absence of bradycardia is those with hypertrophic obstructive cardiomyopathy (HOCM). It is thought that the abnormal ventricular activation associated with right ventricular pacing may relieve outflow tract obstruction and increase cardiac output (27). Some trials have shown benefit of pacing in terms of both symptoms and reduction of outflow tract gradients. It is not clear whether these benefits related directly to altered ventricular contraction pattern with pacing or to the ability to use higher doses of negatively inotropic and/or chronotropic agents, or perhaps to placebo effect alone (28–32).

It has been suggested that AV synchronous pacing can benefit patients with dilated cardiomyopathy. Some of the benefit may result from minimizing mitral and tricuspid regurgitation, which may be seen in late diastole in the dilated heart, particularly if there is intraventricular conduction delay. Preliminary data have not been conclusive (33–36).

Some have suggested that atrial pacing may help prevent recurrence of atrial fibrillation; dual-site atrial pacing may be even more beneficial (37),(38). At present, atrial pacing cannot be considered first-line therapy for prevention of recurrence of atrial fibrillation.

**Pacemaker Systems and Function**

Transvenous permanent pacemakers typically are made up of a pulse generator implanted in a subcutaneous or submuscular pocket connected to one or two leads whose distal tips are placed against the right atrial and/or ventricular wall. The pulse generator is set to sense and pace one or both of the chambers in any one of a variety of programmable modes.

Pacemaker function is described by a code established by the North American Society for Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG). The pacer mode is identified by at least three letters signifying (in order): (a) the chamber paced, (b) the chamber sensed, and (c) the response to sensed events. Additional letters may be appended to signify: (d) programmability or rate modulation, and (e) antitachycardia functions. The entire code is shown in Table 22.12. The code is very useful in the discussion of the many potential pacing modalities that follow. Most pacing modes can be adequately identified using the first three positions of the code. Each of the typical single- and dual-chamber modes can exist with or without rate responsiveness (an “R” in the fourth position of the code), which is discussed later.

**The Basics of Pacing**

Reduced to the most essential level, pacemakers perform three basic functions: pacing, sensing, and timing, which are discussed in the following sections.

**Pacing**

If an electrical impulse is introduced by an electrode pair and depolarizes enough myocardium to initiate propagation of an activation wave front throughout the myocardium, the chamber can be said to have been paced. Sometimes the term capture is used to denote successful pacing. The minimum total energy required to pace successfully is the stimulation threshold.

The amplitude of the depolarization stimulus (in volts for constant voltage stimulation or in milliamps for constant current stimulation) that is required for capture varies with the duration of the stimulus (referred to as pulse width, in milliseconds). Minimizing the energy of the pacing impulse is important to minimize battery depletion, but a safety margin is always included to allow for inevitable variations in the stimulation threshold. In the stable situation, there should be either a threefold safety margin in pulse width or a twofold margin in amplitude. Some newer pacemakers can detect changes in capture threshold and modulate their output accordingly. This allows energy drainage to be minimized to prolong battery life.

**Sensing**

For a pacemaker to coordinate pacing with any intrinsic activity of the heart, there must be a means of sensing electrograms and differentiating the true electrogram of depolarization from extrinsic signals and the repolarization electrogram.

As the wave of depolarization approaches a recording electrode in contact with the endocardium, a deflection is recorded due to the potential difference between the recording electrode and the indifferent electrode. As the wave front passes under the recording electrode and proceeds away from it, a deflection in the opposite direction is recorded. The transition from the approaching to receding deflection is called the intrinsic deflection. The slope of the intrinsic deflection (the rate of change of potential, dV/dt) is called the slew rate. Lower-frequency deflections before and after the intrinsic deflection represent depolarization of surrounding myocardium. Finally, there is also a deflection as the wave of repolarization approaches and then passes by the recording electrode.

The pacemaker system has an amplifier to increase the signal from the recording electrode and a bandpass filter to remove signals with frequencies too high or low to be the intrinsic deflection. Whenever the amplitude of the filtered signal exceeds the sensing threshold (which may be programmed), the electrogram is “sensed” and the appropriate timing circuits of the pulse generator are reset.
Timing Cycles

The pacemaker's timing circuits allow complicated interactions between sensed and paced events and, in dual-chamber pacemakers, between atrial and ventricular events. The nature of these interactions is determined by the programmed mode of the pacemaker, discussed later. The pacemaker's program makes use of a series of timers that are reset by specific sensed or paced events. The programmed mode of the pacemaker is the combination of timers and their reaction to sensed and paced events.

Cycle Lengths and Rates

Intervals and cycle lengths are expressed in units of time, usually milliseconds (ms), with rates expressed in the inverse of time, such as beats per minute (bpm) or pulses per minute (ppm). For practical purposes, it is a simple matter arithmetically to convert between cycle length and rate. Rate in bpm or ppm can be found by dividing 60,000 by the cycle length in milliseconds; conversely, cycle length in milliseconds can be found by dividing 60,000 by the rate in beats per minute or pulses per minute.

Single-Chamber Pacing

The timing cycles of single-chamber pacemakers are simpler and will be considered first. The simplest case is a single-chamber asynchronous mode (AOO or VOO). No intrinsic electrograms are sensed, and pacing stimuli are delivered regardless of any intrinsic activity. The only timer is that of the basic cycle length or lower rate limit. The timer is reset when the pacing stimulus is delivered. When the timer reaches its programmed time, another pacing stimulus is delivered, resetting the timer again. Asynchronous pacing like this is now only used in temporary situations in which the pacemaker may be inappropriately inhibited (e.g., by surgical electrocautery) or when sensing is unreliable (as in the case of lead dislodgment or lead fracture). VOO pacing is shown schematically in Fig. 22.2A.

Schematic representation of asynchronous (A) and inhibited (B) ventricular pacing. The bars indicate the duration of the indicated timing circuit. (For simplicity, no atrial activity is shown.) A: V00 pacing. Pacing stimuli occur when the lower rate limit timer completes its cycle and the timer is reset. There is no sensing of intrinsic electrical events; therefore the timer completes its cycle regardless of the native QRS complexes. B: VVI pacing. The lower rate limit and the ventricular refractory period timers are shown. The lower rate limit timer is reset by a pacing stimulus or by a sensed intrinsic event that does not fall within the refractory period. An intrinsic QRS that is detected within the refractory period does not reset the lower rate limit timer.

VVI pacing is shown schematically in Fig. 22.2B.

The so-called demand or inhibited modes (in which the pacing only occurs in the absence of intrinsic activity) require sensing of intrinsic events. The basic cycle length timer can now be reset by either a paced or a sensed event. However, it would be undesirable for the timer to be reset by an early premature beat, which may not generate an effective stroke volume. Therefore an additional timer is introduced, the ventricular (or atrial) refractory period (VRP or ARP). The refractory period also prevents inappropriate sensing of the repolarization wave as an electrogram (e.g., the T wave). During the refractory period, intrinsic events are not sensed and the lower rate limit timer is not reset. The refractory period occurs during the first part of the basic cycle, starting when an electrogram is sensed or when a pacing stimulus is delivered. VVI pacing is shown schematically in Fig. 22.2B.

Practically speaking, the only modes commonly used with single-chamber pacemakers are AAI and VVI. Rate responsiveness can be added in either case (AAIR or VVIR). In these inhibited modes, the pacer output is inhibited when it senses an impulse within the appropriate time period after the last paced or sensed event.

In triggered modes, a sensed event causes the delivery of a pacing stimulus. Triggered pacing is intended to prevent inappropriate (and possibly dangerous) inhibition by incorrectly sensed events, such as skeletal myopotentials. Though triggered pacing is now uncommonly used clinically, it may be used temporarily to assist in pacemaker interrogation to test sensing. In triggered mode, a pacing stimulus indicates that an electrogram has been sensed. As the sensitivity is decremented, disappearance of pacing artifacts indicates loss of sensing.

Dual-Chamber Pacing

Dual-chamber pacing dramatically increases the complexity of the programming that is possible. Interaction of paced and sensed events in two chambers allows for multiple pacing modes and multiple possible responses to intrinsic cardiac activity. It follows therefore that the number and complexity of the timings circuits also increase.

DOO Mode

In the DOO mode (asynchronous dual-chamber pacing), the atrium and ventricle are paced sequentially; no intrinsic electrical activity is sensed. Therefore pacing stimuli are delivered regardless of any intrinsic activity. The atrial and ventricular pacing stimuli are separated by the AV interval (AVI), which is reset each time an atrial pacing stimulus is delivered.
Modes in which sensing of electrograms is necessary (for inhibition or triggering) require additional timing circuits to coordinate the paced and sensed events in the two chambers. First among these is the \textit{blanking period}, which is a short period (usually less than 100 ms) starting at the delivery of a pacing stimulus in the opposite channel. That is, when an atrial stimulus occurs, the ventricular channel is “blanked” for several milliseconds, so that the leading edge of the pacing stimulus itself is not sensed inappropriately. Though the leading edge of the atrial pacing stimulus is not sensed due to the blanking period, the remainder of the pacing spike may be sensed. If this is the case, ventricular pacing is inhibited and inappropriate inhibition in this setting can be very dangerous in the pacemaker-dependent patient. This problem prompts the addition of another timer, the \textit{cross-talk detection} window. The cross-talk detection window timer begins at the end of the blanking period and, like the blanking period, is a part of the AVI (see later).

Another important timer is the \textit{postventricular atrial refractory} period, or PVARP. The PVARP begins when a ventricular event is sensed or a ventricular pacing stimulus is delivered. The atrial channel is refractory to sensed events during the PVARP. Its primary purpose is to prevent sensing of retrograde P waves and subsequent triggered pacing of the ventricle, which may initiate pacemaker-mediated tachycardia (see later discussion).

The \textit{atrial escape interval} (AEI) is the time between a sensed or paced ventricular event and time at which the next atrial stimulus is delivered (if no atrial event is sensed). The AEI corresponds to the VA interval on a sinus rhythm ECG.

The \textit{upper rate limit} (URL) or \textit{maximum tracking rate} (MTR) is the maximum rate at which ventricular pacing will occur in response to sensed atrial events. The URL is programmable and may be set at a high rate in the vigorous patient who requires a relatively high heart rate during activity. The rate may be set lower in other patients who may not tolerate high rates, particularly if they are prone to atrial arrhythmias.

\textbf{Atrial Versus Ventricular-Based Timing}

The timing of a dual-chamber pacemaker must be based on either the ventricular timing or atrial timing.

In ventricular-based timing, the VV interval is considered paramount and is not allowed to drop below the LRL interval. The VV interval is composed of AEI + AVI. This AEI is kept constant and equals the LRL - AVI. The consequence of this is that if the patient's AV node function is intact and the PR interval is less than the programmed AVI, the actual ventricular rate will be slightly faster than the programmed rate. That is: \(\text{AEI} + \text{PR} < \text{AEI} + \text{AVI}\). Since the ventricular rate is most important, a sensed R wave always resets the entire basic cycle and therefore resets the AEI timer.

If the timing is atrial based, the AA interval is considered paramount and is held constant. In this case, rapid AV conduction does not change the AA interval. However, if the AV conduction is not rapid in the next cycle, the ventricular cycle length (which equals the VA interval ‘AA minus the previous PR’ plus the AVI) may slightly exceed the lower rate limit cycle length. Since the AA interval remains fixed, the following VV cannot be longer than the basic cycle length. So though a single VV cycle can exceed the lower rate limit cycle length, the overall rate cannot drop significantly below the lower rate limit.

In most atrial-based timing systems, a ventricular premature complex resets the AA interval. The AA interval and the following AVI pass before the next ventricular pacing stimulus occurs, another situation in which the VV cycle length exceeds the lower rate limit cycle length. Some manufacturers have added further complexity by modifying these basic atrial-based and ventricular-based systems.

\textbf{Complex Dual-Chamber Pacing Modes}

Having discussed some of the details of timing cycles of dual-chamber pacing, we can proceed to discuss how these concepts are applied in several of the more commonly used dual-chamber modes.

In the \textit{DDD mode} (fully physiologic dual-chamber pacing), the pacemaker senses both chambers and is inhibited by sensed events. If atrial activity faster than the programmed lower rate limit is sensed, the atrial stimulus is inhibited; if the atrial activity is not sensed within time period allowed by the lower rate limit, an atrial stimulus is produced. A ventricular stimulus is triggered (after an appropriate programmed AV delay) by sensed or paced atrial activity, but inhibited if the intrinsic ventricular activity is sensed before the end of the AV delay. In this sense, the DDD mode is a triggered mode as well and an inhibited mode, as the D (for \textit{dual}) in the third position of the NASPE/BPEG code suggests.

DDD (with or without rate responsiveness) is the most commonly used mode in current dual-chamber pacemakers. It allows for physiologic AV synchrony whether the atrium is sensed or paced, and it allows for normal ventricular activation through the intrinsic conduction system if AV nodal conduction occurs. Ventricular pacing will “track” the sensed atrial rate (up to the upper rate limit), allowing the patient's intrinsic atrial rate to dictate the ventricular rate. It should be noted that in patients with AV node dysfunction but with normal sinus node function and chronotropic competence, DDD mode is more physiologic than DDDR, since the ventricular pacing rate is determined by the patient's intrinsic sinus rate alone. DDD pacing is shown schematically in Fig. 22.3.
Other sensor strategies are used less commonly and are still in development. These include central venous temperature, changes in rate and depth of respiration. These results can be used to estimate minute ventilation and thereby modulate the pacing rate.

Between the tip of the lead and the pulse generator, the impedance increases as the chest expands, so it is dependent on the respiratory impedance across the thoracic cavity. The impedance is measured frequently by delivering a low-amplitude pulse. Another strategy to estimate metabolic demand is to sense minute ventilation. Minute ventilation can be estimated by measuring to detect patient motion in a single axis (e.g., in the anteroposterior direction).

The activity sensor is the accelerometer. The accelerometer’s piezoelectric crystal is isolated from the case of the pulse generator and is intended to detect patient motion in a single axis (e.g., in the anteroposterior direction). A similar activity sensor is the vibration of the crystal, which results in an electrical signal that can be used to estimate the intensity of patient activity. A similar sensor strategy is the motion sensor, which consists of a piezoelectric crystal mounted on the case of the pulse generator.

The most commonly used sensor is the motion sensor. This consists of a piezoelectric crystal mounted on the case of the pulse generator. Vibration of the crystal results in an electrical signal that can be used to estimate the intensity of patient activity. A similar activity sensor is the accelerometer. The accelerometer’s piezoelectric crystal is isolated from the case of the pulse generator and is intended to detect patient motion in a single axis (e.g., in the anteroposterior direction).

**Rate Responsiveness**

The most commonly used parameter in the fourth and fifth positions of the NASPE/BPEG code is an “R” in the fourth position to indicate rate responsiveness. Many advanced pacemakers have this feature, in which the lower-paced rate increases in response to the patient’s activity. Other parameters, such as the AVI, may also be adjusted to sensed activity.

Rate-responsive pacemakers must have a sensor of patient activity. Ideally, such a sensor would match the lower rate limit to the metabolic demands associated with increased patient activity, in the same way the normal sinus node does.

The most commonly used sensor is the motion sensor. This consists of a piezoelectric crystal mounted on the case of the pulse generator. Vibration of the crystal results in an electrical signal that can be used to estimate the intensity of the patient’s activity. A similar activity sensor is the accelerometer. The accelerometer’s piezoelectric crystal is isolated from the case of the pulse generator and is intended to detect patient motion in a single axis (e.g., in the anteroposterior direction).

Another strategy to estimate metabolic demand is to sense minute ventilation. Minute ventilation can be estimated by measuring impedance across the thoracic cavity. The impedance is measured frequently by delivering a low-amplitude pulse between the tip of the lead and the pulse generator. Since the impedance increases as the chest expands, it is dependent on the respiratory rate and depth of respiration. These results can be used to estimate minute ventilation and thereby modulate the pacing rate.

Other sensor strategies are used less commonly and are still in development. These include central venous temperature, changes in the QT interval with autonomic activity, and mixed venous oxygen saturation.

It should be obvious that all these sensors are vulnerable to various artifacts and inaccuracies. Some newer pacemakers utilize a combination of sensor strategies (e.g., motion and minute ventilation) in an attempt to mimic physiologic rate response more accurately. However, rate responsiveness still cannot replace intact sinus node function with intact response to the autonomic nervous system.

**Hysteresis**
Some syncope syndromes, such as vasovagal syncope and carotid sinus hypersensitivity, are often characterized by precipitous decreases in heart rate. The patient may need rapid pacing at the times of the would-be syncopal episodes but may not need pacing at all at any other time. The hysteresis function was designed to address this problem. In typical programming, the pacemaker will pace when the intrinsic heart rate falls below the lower rate limit, at a rate equal to the lower rate limit. When hysteresis is programmed, pacing is initiated whenever the intrinsic heart rate falls below a programmed lower rate limit, but the pacing rate is faster than the lower rate limit. If the intrinsic rate has recovered (to a programmed rate), pacing ceases until the patient's rate again declines below the programmed minimum.

**Some Common Problems and Solutions**

Interactions between the patient's intrinsic activity and pacemaker output may make for results that are surprising to the uninitiated. Safety features have been built in to prevent many of these problems.

**Pacemaker Syndrome**

Most of the problems in this section are related to the complicated relationships between sensed and paced events. Pacemaker syndrome, in contrast, is most often a problem of single chamber ventricular pacemakers. The symptoms vary from patient to patient but may include fatigue, malaise, headache, a sensation of throbbing in the neck, and lightheadedness or syncope. The symptoms are thought to result from lack of physiologic AV synchrony. P waves occurring during ventricular systole result in atrial contraction against closed mitral and tricuspid valves and retrograde transmission of the pressure of atrial contraction (a cannon A wave). The symptoms are attributed to depressed cardiac output due to loss of "atrial kick" or to the elevated venous pressure associated with cannon A waves. In patients with intact retrograde AV nodal conduction, there may be a retrograde P wave and a cannon A wave with every paced beat, and the symptoms can be debilitating.

The only solutions for pacemaker syndrome are reduction of the ventricular pacing lower rate limit so that pacing rarely occurs (if feasible), or upgrade to a dual-chamber pacemaker.

**2:1 AV Block**

When a dual-chamber pacemaker senses atrial activity and paces the ventricle (e.g., in the case of complete heart block with normal sinus node function), 2:1 AV block occurs at a predictable rate for a given set of programmed parameters. The 2:1 cycle length is equal to the total atrial refractory period (TARP); the TARP is equal to the AV interval plus the postventricular atrial refractory period (PVARP); that is,

\[ 2:1 \text{ block cycle length} = \text{TARP} = \text{AVI} + \text{PVARP} \]

The implication of this formula is that an atrial event that is sensed before the expiration of total atrial refractory period will not be followed by a ventricular stimulus from the pacemaker. Therefore sinus tachycardia or an atrial tachycardia that occurs faster than this cycle length will result in lack of ventricular pacing after every second atrial event (Fig. 22.4). This 2:1 block may occur precipitously at the time that atrial rate exceeds the 2:1 block rate and is often quite symptomatic. For example, 2:1 block may occur suddenly with sinus tachycardia during exercise, causing the patient to experience a sudden halving of the ventricular rate. Many modern pacemakers have rate-smoothing algorithms that anticipate 2:1 block and change the pacing rate gradually to prevent sudden ventricular rate decreases.

**FIG.22.4** Schematic representation of pacemaker 2:1 AV block in a patient with complete heart block. The pacemaker is in DDD mode. The atrial rate exceeds the maximum tracking rate (MTR). The MTR interval is equal to the total atrial refractory period (TARP), which in turn is equal to the sum of the AV interval (AVI) and the PVARP. When an intrinsic atrial event falls within the TARP, it does not trigger a ventricular pacing stimulus. The next P wave does trigger a ventricular stimulus and the cycle continues as long as the atrial rate exceeds the MTR.

**Pacemaker Wenckebach**

The 2:1 AV block rate is defined by the TARP. When the upper rate limit of the pacemaker is set below the 2:1 AV block rate, pacemaker Wenckebach may occur. When the patient's sinus rate exceeds the programmed upper rate limit, the pacemaker will pace the ventricle only as fast as the upper rate limit. The result is progressive prolongation of the AV delay until one of the sensed P waves occurs within the refractory period after the paced ventricular event. There is no ventricular pacing after this P wave, and the cycle repeats itself with the next sensed P wave. On the surface ECG, this strongly resembles typical AV Wenckebach block, except the QRS complexes are paced. The remedy for pacemaker Wenckebach, which may be symptomatic in the active patient, is an increase in the upper rate limit of ventricular pacing. If the 2:1 AV block rate is slower than the programmed upper rate limit, it serves as the effective upper rate limit because 2:1 AV block will occur before the programmed upper rate limit is reached.

**Pacemaker-Mediated Tachycardia**
Any tachycardia in which the pacemaker participates falls into the category of pacemaker-mediated tachycardia. The term usually refers to an endless-loop tachycardia in which the pacemaker AV sequential pacing serves as the antegrade limb of a reentrant tachycardia (51),(52). The endless-loop tachycardia occurs when retrograde AV nodal conduction results in atrial activity that is sensed by the pacemaker and triggers ventricular output. If the patient’s intrinsic retrograde conduction is consistent, this cycle will occur repeatedly until an intervention is performed (Fig. 22.5).

**FIG.22.5**

Pacemaker-mediated tachycardia (PMT) or endless-loop tachycardia. During atrial threshold testing in DDD mode, atrial capture is lost when the atrial output is decreased (heavy arrows). The occurrence of a retrograde P wave (light arrow) triggers ventricular pacing. PMT, repeated retrograde P waves with ventricular tracking, was thus initiated. Telemetry from the pacemaker confirmed that P waves were being sensed though they are obscured by the T wave in the ECG tracing. The PMT was terminated by temporarily extending the postventricular atrial refractory period (PVARP). Ventricular pacing is then not triggered by the P wave falling within the PVARP, and the cycle is broken. (Courtesy M. Daoust, RN, BIDMC Pacemaker Clinic.)

Treatment for endless-loop tachycardia is aimed at creating a block in one of the limbs of the reentrant circuit. The simplest approach is to place a magnet over the pulse generator. Magnet application trips a magnetic reed switch in the generator that changes the pacing mode to asynchronous (DOO) pacing. When the pacer stops sensing and tracking retrograde P waves, there is a block in the antegrade limb of the tachycardia circuit.

Prevention of endless-loop tachycardia requires reprogramming. Changing modes permanently prevents tracking of retrograde P waves, but sacrifices the advantages of DDD mode. Extension of the Post-Ventricular Atrial Refractory Period (PVARP) is usually effective, and DDD mode can still be used. However, the PVARP cannot be extended too far. As discussed earlier, increase of the PVARP reduces the rate at which 2:1 block occurs, which may be especially troublesome in patients with prolonged VA conduction. Most new pacemakers have “PMT intervention” designed to interrupt an endless-loop tachycardia. For example, a temporary prolongation of the PVARP may be programmed to occur after pacing at the upper rate limit occurs for a given period of time. The retrograde atrial activation that occurs within the prolonged PVARP is not sensed, and the tachycardia is terminated. Endless-loop tachycardia can be prevented by a temporary prolongation of the PVARP after sensation of intrinsic ventricular activity without preceding atrial activity (most likely a ventricular premature complex).

**Cross-talk**

Cross-talk refers to inappropriate sensing of the stimulus of one lead by the other lead. For example, in DDD mode, cross-talk can result in inhibition of ventricular pacing due to inappropriate sensing of the atrial stimulus artifact by the ventricular channel. In the patient dependent on ventricular pacing, cross-talk can be catastrophic. This may be a matter of incorrect sensitivity programming. However, another cause may be dislodgment of the atrial lead into the ventricle. Safety pacing is designed to prevent inappropriate inhibition of ventricular pacing by the sensed atrial stimulus artifact. If an electrogram is sensed by the ventricular lead within the cross-talk detection window, a ventricular stimulus is delivered. Safety pacing prevents inappropriate inhibition, but the source of inappropriate sensing must also be sought. Examination of a chest x-ray is important to ensure good lead positioning. If a lead has been dislodged, revision is necessary. If the leads are in good position, reprogramming of the ventricular sensitivity is required. Examples of safety pacing are shown schematically in Fig. 22.6.

**FIG.22.6**

Schematic representation of safety pacing. In each case the ventricular channel senses an event during the cross-talk detection interval (CDI), prompting delivery of a ventricular pacing stimulus at the end of the CDI. A: The decay of the atrial pacing stimulus is sensed by the ventricular channel after the expiration of the ventricular blanking period. Note the size of the atrial stimulus artifact. Safety pacing occurs and the resulting AV delay is shorter than the programmed AV delay. B: A ventricular premature complex occurs during the CDI, prompting safety pacing. C: The atrial lead has dislodged into the ventricle. The atrial pacing stimulus captures the ventricle. Ventricular activation is detected in the ventricular channel during the CDI, prompting safety pacing.

**Tracking Atrial Tachyarrhythmias**

In DDD mode, ventricular pacing is triggered by sensed atrial activity, and the ventricular rate “tracks” the atrial rate. If the atrial rhythm is abnormal, tracking the atrial rate may be inappropriate. The most prominent examples are atrial tachycardia and atrial fibrillation. If these were tracked without limit by ventricular pacing, ventricular rates could easily achieve dangerously high levels, particularly in atrial fibrillation. This eventuality is prevented by the existence of the maximum tracking rate. However, atrial tachycardias in which the atrial rate exceeds the maximum tracking rate will cause ventricular pacing at the maximum tracking rate.
The possibility of inappropriate tracking must be considered in patients prone to develop atrial fibrillation and atrial tachycardia. One option is not to use DDD mode. DDI allows dual-chamber pacing and prevents bradycardia but sacrifices the advantages of atrial tracking when the patient is in sinus rhythm. 

Mode switching was developed to solve this problem. When a pacemaker with mode-switching programmed on senses an atrial rate exceeding a programmed mode-switch rate, the mode changes to a nontracking one, such as DDI or VVI. The pacer's mode-switching circuit continues to monitor the atrial rate. When the atrial arrhythmia is terminated, the mode is switched back to DDD.

Pacemaker Leads

Pacing, sensing, and timing are functions of the pacemaker pulse generator. Pacemaker leads provide the direct electrical connection between the pulse generator and the heart. Each lead consists of one or two exposed metal electrodes, which are placed in contact with the heart, connectors specifically designed to fit into the head of the pulse generator, and insulated wires connecting the connectors to the electrodes.

Pacemaker leads are either unipolar (meaning that a single electrode is incorporated into the lead ‘typically the cathode’ and the other electrode ‘typically the anode’ is incorporated into the casing of the pacemaker generator) or bipolar (meaning that the anode and cathode are both incorporated into the distal end of the lead). Uni- or bipolarity applies both to pacing and sensing. The potential difference created or sensed using bipolar lead occurs over a few millimeters, while the difference using a unipolar lead is over the chest, from the tip of the lead to the generator. For this reason, unipolar pacing spikes are much more prominent on surface ECG; the pacing artifact of bipolar leads is sometimes difficult to discern, occasionally providing a challenge for even the experienced ECG reader.

Some operators still prefer unipolar leads, which have a long track record and are usually of smaller caliber than bipolar leads. However, bipolar leads have now proven quite successful and have several advantages over unipolar leads:

- No sensing of skeletal muscle myopotentials, which may result in inappropriate inhibition of pacing.
- Less chance of “cross-talk” between the two leads of dual-chamber pacemakers.
- Less chance of interference with an ICD. ICDs should not be used in conjunction with unipolar pacing.
- Less chance of stimulating skeletal muscle.
- If necessary (e.g., in the event of lead damage), unipolar pacing is still possible if the pulse generator has unipolar pacing capability.

For these reasons we advocate the use of bipolar leads.

Leads are currently insulated with either silicone or polyurethane. Silicone has been used for many years with good results. The original polyurethane formulations (e.g., P80A) were subject to unacceptable levels of deterioration and fracture (53), (54). Later formulations have resulted in improvements, making polyurethane a useful material. The P55D formulation currently in use has been quite successful over more than a decade of use. Choice of insulation is a matter of personal preference. Silicone leads tend to be more flexible, and many find the firmer polyurethane leads more manipulable. Silicone leads slide poorly against each other, though lubricant coatings have been applied to minimize this problem.

Placement of leads initiates an inflammatory reaction at the site of electrode contact with the myocardium. The inflammation in turn results in insulation of the myocardium from the electrode and an increase in pacing threshold (55–57). The threshold usually peaks 3 to 6 weeks after implantation. Over time, the inflammatory reaction diminishes and the capture threshold gradually decreases, reaching a stable “chronic” level after about 3 months (Fig. 22.7). Anticipation of the acute threshold rise should prompt programming high output at the time of implant to maintain a good safety margin as the threshold rises. In rare instances, the acute threshold rise can exceed the output of the pacemaker, a potentially dangerous situation in the pacemaker-dependent patient, possibly requiring placement of a temporary pacemaker. Systemic steroid therapy had been used to reduce the inflammatory response (58). Currently, the problem of acute threshold rise has largely been circumvented by the development of steroid eluting leads. The leads have a reservoir of steroid near the electrode that flows slowly through the porous electrode into the myocardium. Steroid-eluting leads reduce the magnitude of the inflammatory response and the acute rise in capture threshold (Fig. 22.7) (59–61).

FIG.22.7

Typical time course of capture threshold changes for two hypothetical ventricular leads. The threshold amplitude at a fixed pulse width of 0.5 ms rises sharply in the first few weeks after implantation, then declines to a level that is greater than the threshold at original implantation. The effect is markedly attenuated in steroid-eluting leads.

All currently used leads have some form of fixation mechanism, passive or active. Passive fixation mechanisms include tines, talons, and fins, designed to become entrapped in trebeculations in the heart to provide stability of positioning. Active fixation devices are designed to
penetrate the myocardium and remain embedded in it. Most of these are helical screws, which are twisted into the myocardium. Passive fixation leads work well in the right ventricular apex and right atrial appendage, which are trabeculated. Active fixation leads allow greater versatility in choice of implantation sites and reduce the risk of dislodgment of the atrial lead. Active fixation leads generate a more exuberant inflammatory response; both acute and chronic pacing thresholds are higher than those of passive fixation electrodes.

DEVICES FOR THE TREATMENT OF TACHYCARDIAS: IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS (ICDS)

Sudden cardiac death (SCD) affects approximately 450,000 people per year in the United States, and at least 1 million people per year develop conditions that place them at risk for SCD in the future. Survivors of one SCD episode have a recurrence rate of approximately 30% to 50% within 2 years, with malignant ventricular arrhythmias most often responsible. Options for treatment of these arrhythmias now include antiarrhythmic agents, catheter-based or surgical ablation, and implantation of implantable cardioverter defibrillators (ICD). This section of the chapter will focus on the indications for ICDs, description of the defibrillator system and device function, and follow-up of patients with ICDs.

The development of the ICD is primarily the result of pioneering work by Michel Mirowski. The first human implant was performed on February 4, 1980, and the first device was approved by the Food and Drug Administration (FDA) in 1985. Early, retrospective studies had established that ICDs can effectively detect and terminate ventricular arrhythmias and suggested that they improved survival (62–64). Subsequently, several randomized trials have attempted to define which patients benefit from ICD therapy.

Indications for ICD Therapy

Secondary Prevention

The most common indication for implantation of ICDs remains the secondary prevention of sudden cardiac death. There are currently three prospective, randomized trials comparing ICD with the best medical regimen as first-line therapy for patients who have survived one SCD episode, using total mortality as primary endpoint (65–67). These studies are reviewed in Table 22.13. Preliminary metaanalysis data from the Antiarrhythmics Versus Implantable Defibrillators (AVID), Canadian Implantable Defibrillator Study (CIDS), and Cardiac Arrest Study Hamburg (CASH) trials were presented at the 1999 North American Society of Pacing and Electrophysiology (NASPE) Scientific Sessions. In patients with cardiac arrest or hemodynamically unstable ventricular tachycardia, subgroup analysis based on ejection fractions showed a clear benefit of ICD implantation over amiodarone therapy in patients with ejection fractions (EF) of less than 35% but no benefit in those with EF of 35% or more (unpublished data).

Primary Prevention

Recently, one prospective study has attempted to establish the role of ICD in primary prevention of SCD. The Multicenter Automatic Defibrillator Implantation Trial (MADIT) randomized 196 patients with a history of MI, EF of 35% or less, asymptomatic nonsustained ventricular tachycardia (NSVT), and inducible ventricular arrhythmias during electrophysiology study (EPS) that were not suppressible with IV procainamide, to either ICD implantation or conventional medical therapy (68). The trial was terminated prematurely due to the significant improvement in survival in the ICD group by almost 50% compared with the control group. Another study evaluated the effect on survival of prophylactically implanted ICDs in patients with LV dysfunction and abnormal signal-averaged ECGs at the time of their coronary artery bypass surgery (69). Although this study found no evidence of improved survival among patients with prophylactic ICDs, it did confirm the importance of adequate revascularization in patients with CAD who are at high risk for ventricular arrhythmias. A follow-up to MADIT, the MADIT II, is currently in progress (70). The only prerequisites for enrollment in MADIT II are a history of MI and EF of 30% or less. Another primary prevention study currently in progress is the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) (71). This trial tests the hypothesis that amiodarone or ICD, or a combination of both, can improve overall survival when compared with placebo in patients with clinical heart failure (New York Heart Association 'NYHA' II or III) and low ejection fraction (< 36%). A summary of the available data for some of the secondary and primary prevention ICD trials is shown in Tables 22.13 and 22.14.

Specific Cardiac Diseases

Recent information is now available, suggesting that certain underlying cardiac conditions, such as idiopathic dilated cardiomyopathy, hypertrophic obstructive cardiomyopathy, or congenital long QT syndrome, may predispose patients to malignant ventricular arrhythmias. Although no definitive data are available regarding the efficacy of ICDs in these patients, the severity of the disease states may influence the decision and the timing of ICD implantation.
Dilated Cardiomyopathy

In patients with idiopathic dilated cardiomyopathy, the combination of poor LV ejection fraction and nonsustained ventricular tachycardia (VT) is associated with increased risk of sudden death. In addition, electrophysiology studies in these patients is of limited value due to the low rate of inducibility. Results are available from two trials comparing amiodarone with standard therapy for primary prevention of sudden death in patients with heart failure and left ventricular dysfunction. In the GESICA trial, there was a 27% reduction in sudden death and a 28% reduction in total mortality in the amiodarone group (72). It is important to recognize that this study included approximately 20% of patients in each arm with idiopathic dilated cardiomyopathy. A second study, the Survival Trial of Antiarrhythmic Therapy in Congestive Heart Failure (STAT-CHF), failed to show any significant difference in overall mortality or sudden death rate between the amiodarone and placebo groups (73). However, the study did find a trend toward a reduction in overall mortality among the patients with nonischemic cardiomyopathy who received amiodarone. Although ICD may be the preferred treatment for patients with symptomatic VT or ventricular fibrillation (VF), and in patients with nonischemic cardiomyopathy and asymptomatic non-sustained VT or who are not optimal candidates for ICD implantation-the results of GESICA and STAT-CHF trials suggest that amiodarone may be beneficial.

Long QT Syndrome

The congenital long QT syndrome has variable clinical manifestations, from asymptomatic to recurrent syncope to sudden death due to torsades de pointes. Because it is primarily an electrical disorder and not associated with structural heart disease, long-term prognosis is excellent if arrhythmias can be controlled or prevented. Conventionally, beta blockers are used in patients with syncope or in asymptomatic patients with a strong family history of sudden death. Sympathectomies, as well as permanent pacing to avoid bradycardia-dependent arrhythmias, are also effective in selected patients. ICD implantation is recommended in these patients with recurrent syncope and sustained ventricular arrhythmias, sudden death despite drug therapy, and sudden death as initial presentation. ICD as primary therapy may be considered in patients with a strong family history of sudden death, or in whom drug compliance may be an issue (74).

Idiopathic Ventricular Fibrillation/Tachycardia

Approximately 10% of young patients resuscitated from cardiac arrest have no clear etiology of VF despite extensive evaluation. EP studies can usually induce polymorphic VT or VF, which is often suppressible with class IA agents, but the long-term efficacy of antiarrhythmic agents in these patients is unknown. Despite limited data, it is generally accepted practice to implant ICDs in patients with idiopathic VF, given the guarded prognosis and often the young age of the affected patients.

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy can be a cause of ventricular arrhythmias and should be suspected as a cause of sudden death in young athletes. Sudden death may be the first manifestation of the disease in previously asymptomatic people. Because patients with hypertrophic cardiomyopathy may have ventricular arrhythmias, it has been suggested that they should be routinely screened by Holter monitoring for the presence of nonsustained VT, which may in turn be a marker for sustained VT. No definitive data are available to support this routine screening, and risk stratification in this population remains ill defined.

The degree of outflow obstruction does not correlate with increased risk of sudden death, but inducibility of sustained ventricular arrhythmias on EP testing may be of prognostic value and appears to be associated with increased risk of cardiac arrest and syncope in some patients. Beta blockers and calcium channel blockers are often used for their negative inotropic properties to decrease outflow obstruction. Their efficacy in preventing sudden death, however, is not established. Empiric use of amiodarone has been used with some efficacy, but this remains controversial (75). Sudden death survivors should be considered for ICD therapy (76). The role of ICD in patients with hypertrophic cardiomyopathy with nonsustained VT and presyncope or syncope is not yet proven and remains controversial.

Right Ventricular Dysplasia

An important cause of congestive heart failure and ventricular arrhythmias is right ventricular dysplasia. Patients with RV dysplasia are considered to be at particularly high risk for sudden death if they have a strong family history of sudden death or if they have a history of syncope. Drug therapy is often ineffective. Ablations are sometimes useful and should be attempted first if VT is tolerated. In patients with cardiac arrest, recurrent arrhythmias, or syncope despite drugs or ablations, ICD implantation should be considered to prevent hemodynamically unstable VT or sudden death (77).

Syncope

Syncope is a common presenting symptom for which, in most cases, there is often no known etiology despite extensive evaluation.
Although syncope of unknown etiology usually has a benign prognosis, syncope attributable to cardiovascular causes is associated with increased sudden death and mortality. Therefore high-risk patients with a history of infarctions and low LVEF who have syncope without apparent etiologies and in whom clinically relevant VT/VF is induced at EP study, may be considered candidates for ICD therapy.

Heart Failure

ICD therapy has also recently been considered for patients with heart failure and in those awaiting cardiac transplantation. The stages of heart failure (NYHA classes I to III) appear to correlate with overall mortality and occurrence of ventricular arrhythmias. Preliminary data suggest that ICDs may prolong life in patients with NYHA functional classes I to III, with the initial benefit greatest in patients with classes II and III (78). In patients with end-stage heart failure awaiting heart transplantation, about 20% die suddenly while waiting for the donor organ. ICD therapy in this group of patients appears to decrease the incidence of sudden death. However, the rate of nonsudden deaths, mostly from pump failure, are slightly increased in this group. This raises the question as to whether ICDs can truly prolong survival in patients with end-stage heart failure or are merely changing the mode of their death (79).

Contraindications to ICD Therapy

ICD therapy or implantation is not recommended in patients in whom VT/VF is due to a reversible cause, such as acute myocardial infarction or severe electrolyte abnormalities. Patients with LV dysfunction undergoing routine bypass graft surgery without inducible sustained VT should also not have ICDs implanted (69). Patients with Wolff-Parkinson-White syndrome with VF due to atrial fibrillation should not receive ICD therapy but should instead undergo catheter ablation of their accessory bypass tracts. Patients with terminal illnesses and life expectancy of less than 6 months are unlikely to benefit from ICD therapy. Patients with ongoing infections should not have ICD implanted until the infection has been clearly resolved. Severe psychiatric disorders that may be worsened with ICDs or may preclude follow-up are relative contraindications for ICD implants. Patients with incessant VTs that are refractory to drugs may not benefit from ICDs because the arrhythmias would constantly trigger shocks. Rather, surgical or catheter ablation should be attempted before ICD implantation.

Guidelines

The American College of Cardiology and the American Heart Association have published guidelines for ICD implantation in the same document in which pacemaker guidelines are discussed (7). Table 22.15 is a summary of those guidelines.

Cardioverter-Defibrillator Systems

First-generation ICDs consisted of a large generator placed in an abdominal pocket, capable only of high-energy shocks. In the nearly 20 years since the first implantation in humans, advances in technology have resulted in significantly smaller devices, with sophisticated detection algorithms and tiered therapies. Ongoing developments in defibrillation systems are to continue to decrease the size of generators without sacrificing maximal available energy, to improve sensing and detection of arrhythmias, and to find ways to deliver the lowest energy possible that can defibrillate successfully—the defibrillation threshold (DFT). Despite these advances, the primary goal of the ICD remains to be the rapid and effective treatment of ventricular arrhythmias. The defibrillation system consists of the pulse generator and the leads. The generator supplies low-energy current to power the basic functions of the device as well as high current density for depolarizing the myocardium. The leads set up optimally uniform current density for defibrillation and may provide sensing and backup pacing.

Pulse Generators

The first-generation devices were large (180 cm³ device volume) and were implanted in the abdomen. Evolving ICD technologies have focused on decreasing the size of the generators, which would allow pectoral implantation, decrease local pocket complications, and improve patient comfort. The bulk of the generator consists of the lithium silver vanadium oxide battery and the capacitor. ICD generators must meet the following requirements: They must monitor electrical status through sense amplifiers, analyze waveforms for abnormal arrhythmias, deliver appropriate therapy, be reliable, and have significant lifetime before battery depletion.

The power for the ICD system is supplied by the battery, which serves as the energy storage reservoir. Capacitors store the energy drawn from the battery, because the battery itself cannot deliver a current fast enough for defibrillation, and it cannot deliver a voltage high enough for defibrillation.

Before an arrhythmia can be treated, it must be sensed by the electrodes. Local bipolar electrograms and amplifying systems are used to permit accurate sensing of small electrograms such as seen in VF (80).
ICDs, like pacemakers, can be subject to oversensing or undersensing. Oversensing occurs when the device detects an event that is not due to ventricular depolarization and may result in inappropriate shocks. Examples of oversensing are T-wave sensing, cross-talk (sensing electrical signals from another chamber, e.g., atrium), myopotential or diaphragmatic sensing, or lead fracture leading to electrical noise (Fig. 22.8). Undersensing occurs when the device does not register an event. This occurs most often when the electrogram is smaller than the sensitivity setting of the device. Changes in electrograms can occur with lead dislodgment, infarction at the site of the lead, inflammation or fibrosis at the electrode site, new bundle branch block, and lead fracture. Undersensing is particularly a concern with ICDs, which must deal with having to sense not only normal R waves, which may be up to 20 mV in amplitude, but also VF with R waves that can be less than 1 mV. Because of the variations in the amplitudes of the electrograms, fixed gain and sensitivity settings, such as those used in pacemakers, may result in undersensing of VF. Some devices attempt to enhance VF sensing with auto-amplifiers. The two most common types of amplifiers currently used in ICDs are the automatic gain control and autoadjustable threshold (Fig. 22.9).

FIG.22.8.

Examples of ICD oversensing. A: T-wave oversensing. During sinus rhythm, double counting of R and T waves led to VT detection (TD), triggering therapy with antitachycardiac pacing (ATP). ATP resulted in true VF, which was detected and treated with a single shock. (Courtesy of N. Hallette, RN, BIDMC Device Clinic.) B: Cross-talk. In this example, the ventricular lead is sensing not only R waves from the ventricle, but also activity in the right atrium. The patient is in atrial fibrillation, which is sensed by the device as VF. The patient subsequently received a shock. (Courtesy of N. Hallette, RN, BIDMC Device Clinic.) C: Oversensing of diaphragmatic myopotentials. The patient in this example received multiple shocks while having a bowel movement. Interrogation showed sensing of diaphragmatic myopotentials with valsala maneuvers, sensed by the ICD as VF. (Courtesy of Donald Love, M.D.) D: Lead fracture. Electrical noise (large, sharp spikes) was detected as VF. In this particular case, the shock was aborted when decreased noise at a later strip allowed for detection of sinus rhythm. (Courtesy of N. Hallette, RN, BIDMC Device Clinic.)

FIG.22.9


Leads

Although the pulse generator provides the power for defibrillation and contains circuitry for sensing and detection, the leads set up the current flow for defibrillation and provide the actual sensing of local electrograms. Initial electrodes used for defibrillation were patches which were sewn onto the epicardium or pericardium. With the advent of transvenous systems epicardial patches are now rarely used. Initially, sensing was achieved from the high-voltage patch electrodes. Oversensing, however, prompted the use of a separate sensing lead, either epicardial or endocardial (81).

Endocardial leads are made of high-voltage conductors. At least one conductor is used for the defibrillation coil, which is usually located near the tip of the lead and meant to be placed along the posterior wall of the right ventricle. Defibrillation leads can have either a single coil (one conductor) or two shocking coils (two conductors). The second coil is located more proximally to the distal coil. When a dual coil lead is implanted, the lead would be positioned with the distal coil in the right ventricle (RV) and the proximal coil anywhere from the subclavian vein (SVC) to the right atrium, depending on the anatomy of the patient. Alternatively, separate leads with a single shocking coil each may be implanted in the RV, SVC, coronary sinus (CS), or a combination thereof. Leads placed in the RV must also have sensing and pacing capabilities, whereas leads in the SVC or CS would not be required to have these features.

With the implant of devices in the pectoral region the housing of the pulse generator itself can serve as a second electrode. Because the pulse generator has a large surface area and can provide more even current distribution, defibrillation using the active pulse generator as one of the electrodes can be achieved with lower energies than defibrillation with a combination of leads. The defibrillation threshold (DFT) is the lowest clinically obtained energy that can achieve defibrillation. DFT achieved with a single RV coil and an active pulse generator are comparable to that of epicardial lead systems (82).

Three electrodes may also be used to attempt to lower DFT. The most common electrodes used in practice are the RV, the SVC, and the active pulse generator. Two electrodes may be connected together, with the current flowing between the two joined electrodes (anodes) and a third common electrode (cathode) (Fig. 22.10).

FIG.22.10.
Possible configurations with three electrodes. A: Pulse generator as cathode: area of low current density is across the RV. B: SVC coil as cathode: area of low current density is across the LV apex and LV free wall. C: Optimal configuration, with RV coil as cathode: area of low current density is extracardiac.

Sensing in an endocardial system is achieved through a distal electrode at the tip of the lead. The same conductor can be used for backup pacing. Unipolar leads have only one high-voltage conductor, for defibrillation, and thus do not have pace/sense capabilities. These leads are usually placed in the SVC or CS positions. Bipolar leads consist of two conductors, one for shocking and the other for sensing. Sensing, in this case, occurs between the tip of the lead and anywhere along the length of the shocking coil, and is termed integrated bipolar sensing. Because of the distance between the tip and the coil and the size of the coil, sensing in these leads is more susceptible to noise, farfield artifacts, and postshock undersensing than true bipolar sensing (83). True bipolar sensing occurs between the distal tip of the lead and a ring located approximately 1 cm proximally from the tip. Sensing is thus local and much more reliable. New lead technologies are testing quadrapolar leads, consisting of two shocking coils, a distal tip, and a proximal ring, which would allow for true bipolar sensing at the same time, giving the option for using dual defibrillation coils (Fig. 22.11).

**Tachyarrhythmia Detection**

As described earlier, an event is sensed when the detected R wave is above a set threshold. The time interval between two sensed events is the cycle length (CL). Detection is the process of analyzing recent cycle lengths and R-wave morphologies to classify rhythms and determine appropriate programmed therapy. It should be a rapid process so that therapy can be delivered before a patient develops symptoms or before an electrogram deteriorates, but not too rapid because some arrhythmias are nonsustained. Because ventricular arrhythmias can be sustained or self-limiting, hemodynamically stable or unstable, ICDs should be able to respond to each episode. The rate cutoff is defined as the heart rate above which the device will be triggered to deliver therapy. The original devices were not programmable, and the cutoff rate and sensitivity level were preset at the factory. Devices today have not only multiple zones of detection, but also specific therapies that can be individually programmed into each zone, termed tiered therapy. Detection zones are ranges of CLs that are programmable. An average of the most recent cycle lengths of the sensed events is compared against various detection zones, and the CL is counted if it falls within a specific zone. For example, if the VF zone is programmed to 320 ms (188 bpm), and the VT zone is programmed at 400 ms (150 bpm), a detected CL will be classified into the VF zone if it is less than 320 ms and will be counted in the VT zone if it is between 300 and 400 ms. If the CL is greater than 400 ms, it would fall outside tachyarrhythmia detection zones and would therefore not be classified (Fig. 22.12).

**FIG.22.11.**


**FIG.22.12.**

Examples of the multiple detection zones available with current devices. A: Ventricular tachycardia (VT) will be detected between 440 and 320 ms. Above 320 ms, ventricular fibrillation (VF) will be detected. B: Addition of a “fast” VT zone (FVT) as part of the VF detection window. C: Addition of a “fast” VT zone (FVT) as part of the VT detection window. Different therapies can be programmed for each zone.

Although detection based on cycle lengths of sensed ventricular electrograms is highly reliable, this method can sometimes lead to inappropriate shocks. This most commonly occurs with atrial fibrillation (83) and sinus tachycardia, when the rates of these and other nonventricular arrhythmias fall into the detection zones. To decrease the risk of inappropriate shocks, newer devices offer additional detection parameters to increase the specificity of VT detection. These include sudden onset criterion, rate stability criterion, and criterion based on electrogram morphology, and are only available, for safety reasons, in the lowest VT rate cutoff zones.

Sudden onset is intended to distinguish sinus tachycardia with a gradual increase in rate from VT with a sudden onset. Rate stability criterion allows the ICD to withhold VT detection for rapid, supraventricular rhythms with irregular intervals, and can be used to differentiate VT with minor rate variability from atrial fibrillation with large variations in cycle lengths. The electrogram width and/or morphology criterion is based on the premise that rhythms of ventricular origin generally have different intracardiac electromorphs than those of supraventricular origin. Although the use of these detection enhancement parameters may increase specificity of VT detection, the concern exists that these parameters may mistakenly inhibit therapy for true VT or delay time to detection.

The first ICDs were “committed” devices in that once detection occurred and therapy was initiated, it could not be aborted. Current devices can confirm a rhythm before discharge of energy, and therapy can be aborted if the tachycardia is nonsustained, thereby minimizing
unnecessary and painful shocks. Confirmation does not occur before delivery of antitachycardia pacing (ATP), however, because ATP is meant to be delivered rapidly and painlessly. Examples of an older, committed device and a newer device capable of aborting therapy are shown in Fig. 22.13.

**FIG. 22.13.**

Confirmation. **A (top strip):** Older device with committed therapy only. VF is sensed (FS) and detected (FD). During charging (no EGMs available on this device during charging), VF spontaneously terminated as shown by a sensed ventricular beat (VS) outside the VF detection zone. Because therapy is committed, charge is delivered (CD) despite termination of VF. **B (bottom strip):** Newer, noncommitted device. VF is detected at the left of the strip (FD) and charging begins, followed by spontaneous termination. At the end of the charging period (CE), sinus rhythm was detected during confirmation, and no shock was delivered.

If therapy either is diverted or is unsuccessful in restoring sinus rhythm, redetection of tachyarrhythmia will begin. Most algorithms use a smaller number of intervals for meeting redetection criteria, and most devices will not allow for confirmation in the redetection period after a diverted or unsuccessful therapy. In essence, therapy after redetection of VT/VF following a diverted or unsuccessful shock is committed, so that the overall duration of an episode is kept to a minimum. It is important to recognize that an episode does not terminate just because a therapy has been aborted. Termination of an episode requires that a specific number of CLs fall outside the detection zones and results in the resetting of all detection algorithms and therapies to zero.

**Dual-Chamber ICDs**

The newest generation of ICDs is made up of the dual-chamber pacemaker-defibrillators. The major advantages of a dual-chamber ICD/pacemaker are improvement in the detection and identification of arrhythmias to prevent inappropriate therapy, and availability of dual-chamber pacing capabilities. About 10% to 25% of patients receiving ICDs may require dual-chamber pacing at some point. Although older ICDs can provide backup bradycardia pacing, they are not meant to be used as permanent pacemakers, since the battery drain would be too great with the current systems and they are only capable of fixed-rate pacing. Dual-chamber ICDs add an atrial lead to improve detection specificity and to provide DDD pacing. Implantation of a dual-chamber system has the additional benefit of eliminating pacemaker/ICD interactions in patients who require both. The new dual-chamber ICDs must ensure that pacing function is not affected by shocks that may transiently lower sensing capabilities or increase pacing thresholds; that the defibrillation function is able to operate in all pacing modes; and that the mode-switch function of the pacemaker does not interfere with tachyarrhythmia detection.

**Therapy**

Early devices were limited to a single form of therapy with a single high-energy shock. Today's devices offer a range of therapies, from programmable high-energy defibrillation shocks to low-energy synchronized cardioversion to antitachycardia pacing.

**High-Energy Defibrillation**

The process of defibrillation involves halting ventricular fibrillation wave fronts within a critical mass of myocardium and is a statistical process. Changes in the autonomic or metabolic state of a person can mean that a shock strength is able to defibrillate at a given time but not at others. In addition, each episode of fibrillation has different activation wavefronts and may require different shock strengths. There appears to be a range of energy over which defibrillation can occur, with the probability of successful defibrillation increasing with increasing energy. Clinically, the lowest energy that successfully converts VF to sinus rhythm during ICD testing is taken as the DFT. This value is usually lower than the minimum energy required for consistently successful defibrillation, which is not an obtainable value clinically. A safety margin of usually at least 10 joules (J) must be demonstrated between the maximum output of a device and the DFT.

Many patients undergoing ICD implantation are also on antiarrhythmic drug (AAD) therapy, and the effects of AADs on the DFT should be taken into consideration. Such class I C and IB agents as encainide, flecainide, lidocaine, and mexiletine cause a reversible, dose-dependent increase in energy requirements for successful defibrillation (84–87). Amiodarone appears to have a bimodal effect on DFT. Acute administration may lower DFT in the animal model, but chronic use may elevate DFT (88). Type IA drugs, such as procainamide and quinidine, do not appear to affect DFT significantly (89), whereas sotalol and N-acetyl-procainamide have been shown to lower DFT (90).

One method of lowering DFT is by improving defibrillation waveforms, which describe the manner with which the energy is delivered across the myocardium. Defibrillation waveforms can be delivered as a single monophasic pulse, sequential or simultaneous monophasic pulses, biphasic pulses with the two phases in opposite polarity, or triphasic pulses with the first and third pulses in the same polarity. Current data show that biphasic waveforms achieve the lowest DFT and are commonly used in devices today.

**Low-Energy Synchronized Cardioversions**

[Additional content remains similar to the provided excerpt, discussing the importance of defibrillator waveforms and therapies, including the use of DFT in clinical practice and the impact of various pharmacological agents on defibrillation efficacy.]

[Note: Further content on defibrillator waveforms, patient-specific considerations, and the integration of modern ICD technology.]
Although VF often requires a relatively higher energy for termination, some ventricular tachycardias can be terminated with very low energies. Low-energy synchronized cardioversions have the advantage of faster delivery of therapy, may cause less discomfort, and can conserve battery when compared with high-energy shocks. Although each patient's pain threshold may vary, most tolerate a shock of 1 J or less (91). Although the advantages of being able to terminate ventricular tachycardias rapidly and with little discomfort are evident, the risks of utilizing this therapy are acceleration of stable VT to VF and delay of time to successful treatment if initial cardioversion was unsuccessful.

**Antitachycardia Pacing**

The ability to terminate tachycardias with pacing had been available before the advent of the ICD. However, due to the potential risk of acceleration of VT to VF, the use of ATP devices was limited to the treatment of SVTs until the ability to defibrillate the heart was also available. Similar to low-energy cardioversion, the advantages of ATP include rapid delivery of therapy, less discomfort to the patient, and conservation of battery life. The concept of ATP in termination of VT is based on the observation that ventricular arrhythmias, such as those in patients with prior coronary artery disease, are due to reentrant circuits involving the border zones of prior infarctions. In a reentrant circuit, the leading wavefront of activation must encounter excitable tissue for continuing propagation. Progressively more premature stimuli encroach on the refractory tail end of the wavefront. Termination of VT occurs when a stimulus interacts with the circuit both orthodromically (forward) and antidromically (backward), causing bidirectional block (92). Multiple extrastimuli increase the probability of a stimulus to interact with the tachycardia circuit, at the slightly increased risk of accelerating VT. An example of VT accelerating to VF after ATP, and successful treatment of VF with a synchronized shock, is shown in Fig. 22.14.

**FIG.22.14.**

Acceleration of VT to VF by ATP. A continuous strip is shown. VT is detected in the first part of the rhythm strip (TF), followed by a 12-beat burst of ATP (TP), which accelerated the tachycardia to VF (FS). VF is detected (FD) and charging begins. A synchronized shock is then delivered (CD), resulting in sinus rhythm. (Courtesy of N. Hallette, RN, BIDMC Device Clinic.)

The two most commonly used methods of ATP are rate-adaptive burst pacing and autodecremental or ramp pacing. With rate-adaptive burst pacing (Fig. 22.15A), the device is programmed to deliver a set number of pulses at a constant coupling interval based on a percentage of the VT CL. The sequence may be repeated in successive trains if VT is redetected. Each sequence is titrated by decrementing the coupling interval between pulses by a set amount per sequence, usually 10 ms. In the autodecremental or ramp pacing mode (Fig. 22.15B), the initial coupling interval within a sequence is also based on a percentage of the tachycardia cycle length. Within a sequence, each coupling interval is decremented by a set amount, usually 10 ms. The sequence is titrated by the addition of an extrastimulus at the end of each sequence until the coupling interval reaches the programmed minimum value. Comparisons of the two methods with regard to success of VT termination and acceleration of VT to VF have shown no significant differences between the two methods when 10-ms decrements are used (Table 22.16) (111–113).

**FIG.22.15.**

A: Rate-adaptive burst. The ICD is programmed to deliver on detection of VT an adaptive burst at 91% of the tachycardia cycle length. The number of pulses within the burst is programmed to four and the sequence will repeat itself on redetection of the tachycardia four times. The programmed decrement per sequence is 10 ms but not to shorten to less than a programmed minimum interval of 270 ms. In the example, a tachycardia of 350 ms is detected. The first burst sequence (A) should be 320 ms (350 ms × 91% = 320 ms). The first pulse is delivered accordingly at 320 ms from the R wave that fulfilled the programmed detection criteria. All subsequent pulses of this sequence are separated by 320 ms. Assuming that the VT is redetected and the RR interval remains at 350 ms, a second burst sequence (B) is decremented by 10 ms (320 ms - 10 ms = 310 ms). In the example, sequence (B) results in the acceleration of the tachycardia to 320 ms, which is again redetected. The calculated pulse interval (C) is now 270 ms (320 ms × 91% = 290 ms - 20 ms decrement = 270 ms). Assuming that the tachycardia is unaffected and redetected at 320 ms, the fourth and final burst sequence (D) will be 270 ms (the programmed minimum interval) despite the fact that the calculated pulse interval would have been 260 ms (320 ms × 91% = 290 ms - 30 ms decrement = 260 ms). B: Autodecremental ramp. The ICD is programmed in this case to deliver an autodecremental ramp of four pulses, starting at 91% of the average sensed RR, continuing on redetection for four sequences with a decrement per pulse of 10 ms, not to exceed a minimum interval of 270 ms. The first ramp sequence (A) should start at 320 ms (350 ms × 91% = 320 ms) with each interval thereafter shortened by 10 ms so that the fourth interval equals 290 ms. Assuming that the tachycardia is redetected (B), the initial ramp pulse will be 320 ms, with decrements of 10 ms with the ramp as above but with the addition of a fifth beat at 280 ms. Before the third ramp sequence (C), the average RR shortens to 320 ms. Accordingly, the initial pulse is 290 ms (320 ms × 91% = 290 ms). After a decrement of 10 ms for interval 2; intervals 3, 4, and 5; and the additional sixth are all delivered at the programmed minimum of 270 ms. Acceleration of the tachycardia to 310 ms determines that the first pulse will be delivered at 280 ms, with intervals 2 to 6 and the additional seventh at the minimum programmed value of 270 ms.
Programming of ATP can be performed using a number of methods. If an EP study is performed before ICD implant and ATP was able to terminate the induced VT, ATP can be programmed accordingly. Alternatively, noninvasive programmed stimulation (NIPS) can be performed after implantation through the device, and ATP can then be tailored to determine the setting that would best terminate VT without accelerating it to VF. ATP can also be programmed empirically, which is a reasonable approach since the success of termination may be different between induced and spontaneous VT.

The Technique of Pacemaker and ICD Implantation

Permanent Pacemaker Implantation

Currently, pacemaker and ICD leads are usually implanted transvenously and the pulse generator is implanted in a subcutaneous or submuscular pocket in the pectoral region. Venous access can be afforded by the subclavian, cephalic, or axillary vein. The technique of dual-chamber device implantation is described. Single-chamber device implantation follows the same technique without the placement of the second lead. Transvenous, pectoral implantation of ICDs utilizes the same techniques as pacemaker implantation, with the addition of defibrillation testing.

Preparation

We prefer the left pectoral location for ease of lead introduction and positioning. For right-handed patients the left side is also preferable. The right side can also be used if the left is inaccessible or if preferable (examples include very active left handed golfers or tennis players). The left side is preferred for ICD implantation in which one shocking electrode is housed in the pulse generator (even in the left-handed patient), because the field orientation for defibrillation (can to electrode) is superior to that of the right-sided device position.

Informed consent should be obtained from the patient (or a designated guardian if the patient cannot understand or give consent) before the procedure. The procedure and its risks (see Complications section later) should be explained, and the patient's questions should be answered.

Before the procedure, the history and physical and laboratory examinations should be reviewed with an emphasis on issues important to pacemaker implantation.

The history and physical examination should consider the possibility of injury or pathology in the potential region of device implantation. Injury or previous surgery suggests that abnormal venous anatomy may be present, making venous access more difficult. Allergies or intolerances to antibiotics, radiographic contrast agents, local anesthesia, and sedatives should be noted. Ongoing infection or signs or symptoms of infection (e.g., fever, leukocytosis, chills, productive cough, dysuria) should prompt investigation and resolution before implantation. Congestive heart failure therapy should be optimized to allow the patient to lie flat throughout the procedure. Similarly, the respiratory status should be evaluated and optimized before the procedure.

Some basic laboratory data should be scrutinized before the procedure. The chest x-ray and ECG are part of the original evaluation in most patients. The hematocrit and coagulation parameters should be known to avoid bleeding complications. Coumadin should be discontinued at least 4 days before implantation, and the INR should be 1.5 or less, though a recent study suggests the feasibility of pacemaker implantation in patients receiving oral warfarin (93). Aspirin should be discontinued 1 week before the procedure if possible.

The patient should be in the fasting, postabsorptive state. Meticulous sterile technique should be exercised throughout, with thorough scrubbing and draping of the region to be incised. General anesthesia is not necessary, but local anesthesia should be used liberally. Judicious use of an intravenous benzodiazepine (e.g., midazolam) and a narcotic (e.g., fentanyl) reduces both anxiety and discomfort. The drugs should be short-acting to minimize the risk of severe respiratory depression. A peripheral intravenous line in the arm ipsilateral to the implant is useful, should the need for an injection of intravenous radiographic contrast arise.

Propylactic antibiotic therapy is still somewhat controversial, but we favor its use. A recent metaanalysis of seven randomized trials of antibiotic use at the time of pacemaker implant suggested that antibiotics do reduce the risk of wound infection, pacemaker erosion, and septicemia (94). At our institution, patients receive a dose of intravenous cefazolin or vancomycin during the procedure followed by 48 hours of therapy.

Venous Access

A variety of techniques are available for gaining venous access. The cephalic, axillary, and subclavian veins can all be utilized. We prefer the cephalic vein cutdown approach when possible. Using this approach, venous access is achieved under direct visualization and there is no risk of pneumothorax. In addition, some believe that there is a lower incidence of lead fracture due to crushing at the junction between the first rib and the clavicle.
After the preparation and draping, the deltopectoral groove should be identified by palpation. An incision can be made after infiltration of local anesthesia. We use a mixture of lidocaine and bupivacaine (which has a longer duration of action and helps prevent postoperative discomfort). The incision may be oblique, overlying the deltopectoral groove, or it may be transverse, about 2 cm inferior to the clavicle with its lateral margin crossing the deltopectoral groove (Fig. 22.16). Some prefer the oblique incision because it allows greater exposure of the cephalic vein. Others prefer the transverse incision since it increases the chance of finding the deltopectoral groove. The incision is made with a no. 10 blade, perpendicular to the skin. Enough pressure should be applied to cut through the epidermis and dermis with a single incision. The incision is extended down to the prepectoral fascia. Blunt dissection should be used primarily; sharp dissection and electrocautery should be minimized to prevent inadvertent division of unrecognized structures (especially arteries and arterioles). Moreover, large areas of charred tissue from electrocautery heal poorly and increase the risk of infection or wound dehiscence.

**FIG.22.16.**

Cephalic vein cutdown. A: Surface anatomy of the deltopectoral region. The incision can be made in the groove or across the groove as noted by the marks. B: A fat streak is often seen in the groove between the pectoral and deltoid muscles. The cephalic vein lies just under this streak. C: The vein isolated with distal and proximal ties in place. D: After incising the vein a 5F sheath is introduced followed by a guidewire.

The deltopectoral groove can be identified by the fatty streak between the deltoid and pectoralis muscles. The orientation of the fibers of the two muscles is also different, which can help identify the deltopectoral groove in the very lean patient. The cephalic vein runs in the deltopectoral groove. Once it is identified, it is dissected free of fat and connective tissue and isolated. We typically ligate the vein distally to minimize bleeding. A suture is also passed under the vessel proximally and not tied; traction on this suture is used to control bleeding after the vessel is incised. The vein may be small in caliber and may collapse further with ligation but is usually sufficiently distensible for insertion of introducer sheaths and leads. The vein is incised with iris scissors. Although the lead can be inserted directly into the vein, we prefer to cannulate with a 5F introducer. Through the introducer, a guidewire is advanced (under fluoroscopic guidance) to the superior vena cava through the right atrium and into the inferior vena cava. If the guidewire is not advanced easily, radiographic contrast can be injected either through introducer or through the peripheral intravenous line, to confirm that the introducer is intraluminal and to help guide advancement of the wire. Venography will also reveal venous occlusion if present.

If the cephalic vein cannot be used, we then consider use of the axillary vein (95). The vein can be visualized by injection of radiographic contrast into a peripheral arm vein. Although contrast remains in the vein, it is cannulated under fluoroscopic guidance with an 18-gauge needle (Fig. 22.17) and a guidewire is then advanced to the inferior vena cava as described earlier.

Alternatively, the subclavian vein can be used. From within the incision, the subclavian vein is cannulated with an 18-gauge needle and a guidewire is advanced into the vein. Since the subclavian puncture is performed without direct visualization of the vein, there is a risk of pneumothorax and subclavian artery puncture. Before placing the sheath, the position of the wire within the vein should be confirmed with fluoroscopy. A chest x-ray to exclude pneumothorax is therefore mandatory after completion of the procedure.

**FIG.22.17.**

Fluoroscopic image of venographically guided axillary vein cannulation. Contrast was injected through a left arm peripheral intravenous line. A: The 18-gauge needle is entering the contrast-filled vein. B: A guidewire is passed through the needle and into the subclavian vein. C: The sheath and dilator are advanced over the wire. D: The lead is advanced through the sheath.

The subclavian approach is less desirable because of the risk of pneumothorax and the potential for crush injury to the lead(s) that may occur as they pass between the clavicle and first rib. Subclavian puncture often penetrates the ligaments connecting the first rib and the clavicle, making the lead at risk for being entrapped there and increasing the incidence of crush. Cannulation of the subclavian vein as lateral as possible helps minimize the risk of crush.

**Lead Placement**

Once venous access has been achieved and a guidewire is in place, the wire is used to guide the insertion of venous sheaths to introduce the leads into the vein. The sheath is of the “peel-away” type, to allow its removal after the lead is introduced. The sheath should be large enough to contain the guidewire with the ventricular lead. When the sheath’s dilator is removed, the sheath itself should be pinched to prevent both excessive bleeding and air embolism. The lead should be inserted immediately to minimize the time that the sheath is open to air, and the patient may be instructed to stop breathing during this process (without taking a deep breath first).

The ventricular lead is first advanced to the right atrium. Pacemaker leads are intentionally designed to be flexible; this lack of axial stiffness helps prevent cardiac perforation by the leads. However, such flexible leads are not easily manipulated, and the use of a stiffening wire...
An anterior oblique imaging can assist in ventricular lead positioning, since the length of the right ventricle can be viewed without the foreshortening seen in the anteroposterior view. In any case, the lead should be viewed in the right anterior oblique and left anterior oblique projections to ensure location of the tip of the lead along the floor of the RV septum and in the apex. Sensing of the R waves and pacing threshold are then checked for acceptable function. An ECG should be examined (especially lead V1) during pacing to ensure the presence of a left bundle branch block pattern. Right bundle branch block suggests pacing from the left ventricle, which may occur if the lead crossed the atrial or ventricular septum, if the lead is inadvertently placed in one of the cardiac veins via the coronary sinus, or if the lead has perforated the heart and pacing is occurring from the left ventricular epicardium. Inadequate sensing or pacing despite a good anatomic site suggests an injured area of myocardium, though the integrity of the lead, the testing equipment, and all connections must be confirmed before repositioning. After good ventricular lead placement is achieved, it must be maintained during placement of a second lead (if applicable). Some operators place an anchoring suture at this stage. However, if the lead is nonetheless dislodged, removal of this anchoring suture is usually required for repositioning. Lead stability is improved by retaining the stylet in place while inserting the second lead; withdrawing it approximately halfway helps maintain proximal stability while reducing axial stiffness at the tip and thereby reducing the risk of perforation.

Good ventricular lead positioning is shown in the anteroposterior and lateral orientations in the chest x-rays in Fig. 22.18 A and B. Right anterior oblique imaging can assist in ventricular lead positioning, since the length of the right ventricle can be viewed en face, without the foreshortening seen in the anteroposterior view. In any case, the lead should be viewed in the right anterior oblique and left anterior oblique projections to ensure location of the tip of the lead along the floor of the RV septum and in the apex. Sensing of the R waves and pacing threshold are then checked for acceptable function. An ECG should be examined (especially lead V1) during pacing to ensure the presence of a left bundle branch block pattern. Right bundle branch block suggests pacing from the left ventricle, which may occur if the lead crossed the atrial or ventricular septum, if the lead is inadvertently placed in one of the cardiac veins via the coronary sinus, or if the lead has perforated the heart and pacing is occurring from the left ventricular epicardium. Inadequate sensing or pacing despite a good anatomic site suggests an injured area of myocardium, though the integrity of the lead, the testing equipment, and all connections must be confirmed before repositioning. After good ventricular lead placement is achieved, it must be maintained during placement of a second lead (if applicable). Some operators place an anchoring suture at this stage. However, if the lead is nonetheless dislodged, removal of this anchoring suture is usually required for repositioning. Lead stability is improved by retaining the stylet in place while inserting the second lead; withdrawing it approximately halfway helps maintain proximal stability while reducing axial stiffness at the tip and thereby reducing the risk of perforation.

FIG.22.18.

Posteranterior (A) and lateral (B) chest x-rays showing typical dual-chamber pacemaker placement. This patient has biventricular enlargement. The ventricular lead is in the right ventricular apex, and the atrial lead is in the right atrial appendage.

Using the retained wire as a guide, a second peel-away sheath is advanced into the vein. The atrial lead is then advanced and placed in the atrium. In the patient who has not had previous cardiopulmonary bypass (and therefore whose right atrial appendage is intact), the right atrial appendage is the ideal site for lead placement. If the lead is of the preformed “J” shape, the straight stylet may be withdrawn partially after the lead has been advanced to the right atrium. Partial withdrawal causes the lead to resume a 90° angle. Torque applied to the stylet rotates the tip of the lead to the desired anteromedial position of the right atrial appendage. Further manipulation may be necessary to ensure good contact with the endocardium. If a straight atrial lead is used, a “J”-shaped stylet is used for lead placement. The stylet is placed to produce a 90° angle in the lead, and torque applied to the stylet is used to position the lead in the desired location. The straight atrial lead is easier to manipulate using the curved stylet, making it more desirable when sites other than the right atrial appendage are likely to be explored.

A lead in the right atrial appendage has a characteristic movement with each cardiac cycle, as shown in Fig. 22.19. During atrial systole, the tip of the lead moves laterally while the loop moves medially. In the patient with no right atrial appendage, a stable site in the anterolateral right atrium is sought. In either case, we prefer active fixation for the atrial lead to minimize the risk of lead dislodgment. With stylet in place to provide some pressure against the endocardium, the helical screw electrode is rotated to engage the myocardium. Some leads have a retracted screw, which is extruded and retracted by a rotating mechanism at the proximal end of the lead. Others have a fixed helical screw, requiring rotation of the entire lead. After the electrode is screwed into the myocardium, careful withdrawal of the stylet usually confirms fixation of the electrode. In addition, when using active fixation leads we examine the electrogram from the lead to ensure atrial sensing and the presence of current indicating that the electrode has indeed penetrated the myocardium (Fig. 22.20). Sensing and pacing parameters are then checked for the ventricular lead. Care must be taken to avoid displacement of the ventricular lead during atrial lead manipulation. Acceptable implant valves are shown in Table 22.17.

FIG.22.19.

Motion of pacing lead in the right atrial appendage. A: During atrial systole, the tip appears to move in a lateral direction, and the loop moves in a medial direction. B: During atrial diastole, the tip moves medially, and the loop moves laterally.

FIG.22.20.
Unfiltered bipolar electrograms obtained from leads during implantation of a dual-chamber pacemaker. Both leads were of the retractable screw type of active fixation. Lead II of the surface ECG is also shown. The tracings are simultaneous. A current of injury can be seen in both electrograms (arrows).

**Pulse Generator Implantation and Pocket Closure**

After further local anesthesia, a subcutaneous or submuscular pocket is made with blunt and sharp dissection, then irrigated liberally with antibiotic solution. We use a liter of saline with 1 g of vancomycin (or 1 g of cefazolin) and 80 mg of gentamicin added. The pacemaker pulse generator is connected to the lead(s), ensuring that the correct lead is placed in the correct position in the header of the generator and that the leads are secured in place. The system is then implanted into the pocket with the lead(s) coiled behind the generator to minimize the risk of damage to the leads in the event of reincision. The ECG monitor is then examined to ensure appropriate pacing and sensing. If the patient is in sinus rhythm at a rate exceeding the lower rate of the pacemaker, we place a sterile magnet over the generator to ensure that it will pace in asynchronous mode. The pocket is then closed with two layers of an absorbable suture. We typically use a continuous subcuticular stitch of absorbable suture to close the skin. We place adhesive tapes over the incision and then a gauze dressing over the area.

After satisfactory positions for both leads are found, the leads should be examined under fluoroscopy while the patient inspires deeply and while he/she coughs vigorously to ensure that the lead tips are not dislodged with these actions. The leads should be examined in the left and right anterior oblique orientations to confirm the positions of the lead tips. Pacing at high output should be performed to ensure that there is no pacing of the diaphragm, particularly by the atrial lead, since the right phrenic nerve courses along the lateral right atrium. Once good positioning of both leads has been confirmed they are anchored to the deltoid and pectoralis fascia using a strong nonabsorbable suture, such as no. 0 silk. Three sutures for each lead are recommended. The sutures are tied around an anchoring sleeve that should be advanced over the lead to a position in the deltopectoral groove. Lead parameters are again checked to ensure that no detrimental change has occurred while the anchoring sutures are placed. Some improvement in capture threshold is often seen after the lead has been in place for several minutes, particularly for active fixation leads.

**Pulse Generator Change**

Pulse generator changes are necessary due to the finite lifetime of the pacemaker battery. The procedure is simpler than the original implantation because the leads need not be placed or moved (though great care must be taken not to damage the leads while dissecting the pacemaker generator free). A temporary transvenous pacemaker is indicated for pacemaker-dependent patients and for those at risk for profound bradycardia. In this setting, femoral vein access is the recommended route, since fluoroscopy is available, the need for pulse generator free). A temporary transvenous pacemaker is indicated for pacemaker-dependent patients and for those at risk for profound bradycardia. In this setting, femoral vein access is the recommended route, since fluoroscopy is available, the need for

The pacemaker lead(s) and pulse generator should be examined fluoroscopically. After administration of local anesthesia, an incision should be made that will provide access to the pacemaker pocket and that will minimize the risk of damage to the leads. This will often be the site of the previous incision. In the patient with significant amounts of scarring, the scar from the old incision can be excised with an elliptic incision.

Blunt dissection to the pacemaker pocket and careful incision of its fibrous capsule allow delivery of the generator from the pocket. After disconnection of the generator from the lead(s) using the appropriate tool, the lead parameters should without fail be checked to ensure lead integrity. Before implantation of the new generator, the capsule should be disrupted to prevent creation of an isolated pocket that is more likely to harbor infection. The pocket should then be irrigated with antibiotic solution. The new pulse generator is connected to the lead(s) and implanted into the pocket, which is then closed as described earlier. Before the procedure, the type of existing lead(s) and its connector type should be determined. Every effort should be made to use a new generator compatible with the lead connector type, since the use of adapters increases the risk of malfunction. The new generator must be compatible with the uni- or bipolar nature of the existing lead(s).

Postprocedure wound care, including prophylactic antibiotic therapy, is similar to postprocedure care for a patient undergoing an original implant. An overnight hospital stay is unnecessary in the uncomplicated case, since the leads are chronic and the risk for dislodgment is low.

Before any procedure in which the pacemaker generator is manipulated, the pacemaker should be interrogated. Rate responsiveness should be programmed off to prevent an inappropriate increase in pacing rate with manipulation of the generator. Electrocautery should be minimized to prevent damage to leads and to prevent inappropriate inhibition of pacing, which may be dangerous in the pacemaker-dependent patient. Vigilance is required, and it is often necessary to program the temporary pacemaker to pace asynchronously during use of cautery.

In the case of procedures involving previously implanted ICDs, electrocautery or manipulation of the generator may be sensed by the ICD as tachycardia or fibrillation, prompting inappropriate therapy. Therefore the ICD detection and/or therapy should be programmed off to avoid inappropriate shocks by the ICD.

**Techniques Specific to ICD Implantation**


Surgical approaches for implantation of epicardial patches include the anterolateral thoracotomy, subcostal thoracotomy, subxiphoid, and median sternotomy (96). As stated earlier, this approach has been essentially abandoned since the development of the transvenous system.

The first transvenous systems required abdominal implant due to the size of the pulse generator. Transvenous leads were tunneled subcutaneously to the abdomen. Additionally, since early devices used monophasic waveforms for defibrillation, subcutaneous patches or arrays were often required to achieve adequate DFT. Abdominal implants are now placed rarely, and usually only in patients in whom a pectoral implant would carry significant risks of pocket erosion, in patients with previous multiple pectoral pocket infections, or in patients whose anatomy precludes pectoral implants. By the early 1990s, the size of the pulse generator had decreased sufficiently to allow for pectoral implantation. With the device in this location, the large surface area of the pulse generator could be used as an active electrode, which resulted in further lowering of DFT to the range seen with epicardial implantation (97–99). Currently, left pectoral implants using techniques similar to those used with pacemaker implants are the first choice for most institution. Right pectoral locations can be used but are often associated with slightly higher DFT, due to the greater distance from the heart. Occasionally, submuscular implants are desired by some patients for cosmetic purposes. However, this location may be associated with increased complications of bleeding, damage of the thoracoacromial nerves, and pectoralis major atrophy (100), as well as discomfort due to muscle sliding over the pulse generator.

Initially, ICDs were implanted by cardiothoracic surgeons in the operating room, and this practice continues in some hospitals. However, in most institutions today with an active electrophysiology program, implantation of ICDs has become the responsibility of electrophysiologists. Implantation under local anesthesia combined with intravenous sedation, performed by electrophysiologists in a laboratory with air-filtering facilities similar to those used in the operating room, has been shown to have high success and low complication rates, short implantation and fluoroscopy time, and is associated with earlier discharge from the hospital (101), (102).

Connecting ICD leads to the device is slightly different from connecting pacemaker leads to pacemaker generators. All ICD pulse generators have at least three ports. One is for the sense/pace conductor, and one for the defibrillation coil. The third port can be connected to a second defibrillation lead, such as SVC coil or subcutaneous patch, or may be capped if only one defibrillation coil is used. The newest-generation dual-chamber ICDs have a fourth port, for the atrial sense/pace lead.

Defibrillation Testing at the Time of ICD Implantation

Fundamental to successful ICD implantation is the ability to reliably sense and reproducibly defibrillate VF. Therefore meticulous testing of leads and device function must be carried out at the time of implant. The lead must be tested for adequate sensing in sinus rhythm, as assessed by R-wave amplitudes, which should be no less than 5 to 6 mV and preferably greater than 10 mV. Care must be taken to obtain a stable lead position and to rule out oversensing of myopotentials and diaphragmatic pacing. Pacing threshold must also be acceptably low to ensure reliable backup pacing after defibrillation. Repositioning of the lead must be carried out if any of the parameters are unsatisfactory.

An important part of ICD implantation procedure that is not part of pacemaker implantation is the testing of the device to determine DFT. This is generally performed before closing the wound, after satisfactory lead positions and parameters are obtained, the leads are connected to the ICD, and the system is implanted into the subcutaneous pocket. In our laboratory, anesthesia is often administered by the Anesthesiology Service, usually using short-acting agents such as Propofol. However, conscious sedation with a combination of narcotics and benzodiazepines can be used effectively. The testing is usually performed through a programmer, with a sterile programming wand placed over the implanted pulse generator. Occasionally, testing may be performed through an emulator, which substitutes for the actual pulse generator and connects the defibrillation lead to the programmer. This allows for the decision regarding the ICD model used to be made after the DFT is determined. An initial low-energy (1 to 2 J) synchronized shock may be utilized to ensure that all connections are intact and to determine the high-voltage lead impedance before VF induction. VF is commonly induced by critically timed T-wave shocks or high-frequency pacing. Alternatively, rapid-burst pacing at 50 Hz can be delivered, with the length of time of delivery at the discretion of the operator.

The DFT may be determined in several ways. The DFT testing protocol uses a series of inductions to determine the lowest energy that produces successful defibrillation. For example, with a device capable of a maximum energy output of 34 J, one may begin the test with a shock at 24 J. If successful, then a shock at 12 J can be tested, decreasing to 6 J or even 3 J. The lowest energy that successfully defibrillates VF is the DFT. This method more accurately determines DFT, but multiple VF inductions are needed. Alternatively, the margin-verification protocol requires testing of one selected energy that would allow for an adequate safety margin without having to determine the lowest energy that would successfully defibrillate. Fewer numbers of VF inductions are needed with the latter method and may be used in patients who may not tolerate repeated episodes of VF. In most patients, however, we favor the “step-down” approach because having at least one shock that fails to defibrillate allows for redetection also to be tested. Redetection problems are encountered more frequently with integrated bipolar sensing leads than with true bipolar sensing leads. During testing, we choose to set the second shock at device maximum output. If the first shock fails, the device is allowed to redetect VF and deliver a second shock, and if this fails, we deliver a 360-J shock externally. Usually, 5-minute intervals are allowed between each VF induction.

In addition to sensing, pacing, and determination of DFT, other parameters, such as the impedance and charge time, must also be assessed.
during intraoperative testing to ensure that the device is performing adequately. If an unacceptable value is repeatedly obtained during testing, replacement of the lead, or the device, may be necessary. Charge time is the time needed to charge the capacitor for energy delivery and may vary with generators, but should be short (less than 10 seconds) for new and well-functioning devices.

When adequate DFT is not achievable with a single-coil endocardial lead, reversing shocking polarity by designating the RV lead as the anode and the active can as the cathode may occasionally lower DFT. Also, repositioning the lead to be as close to the RV apex as possible may help to lower shocking energy. Alternatively, using another electrode in the SVC by changing to a dual-coil lead or by inserting a separate SVC or CS lead, or adding a subcutaneous patch or array, may be helpful. With today's devices, a subcutaneous patch or array is rarely required. The entire system should be implanted and the wound closed only after satisfactory sensing in both sinus rhythm and VF is verified, pacing thresholds are low, and lead impedances are acceptable. Because of the possibility of increases in the required defibrillation energy over time due to fibrosis around the tip of the lead, migration of the lead, changes in underlying cardiac status, or addition of antiarrhythmic drugs, a safety margin must be added to the DFT to compensate for future changes. This can usually be accomplished by adding 7 to 10 J to the DFT when programming therapies (103).

An important note should be mentioned regarding patients who have separate permanent pacemakers and ICDs. If pacing spikes are large, ICDs may sense pacer stimulus artifacts and count them as R waves, which would lead to “double-counting” and may trigger inappropriate shocks. A more significant concern is that electrograms from VF are often small and may not be sensed by the pacemaker, thus triggering pacing. The pacing spikes may reset the amplifier (decrease the gain) of the ICD, which may result in failure to sense VF. Unipolar leads are particularly problematic, since they produce large pacing spikes. For this reason, implantation of a unipolar pacing lead is absolutely contraindicated in patients with preexisting ICD. During intraoperative testing of a new ICD system in the setting of an existing pacemaker, the pacemaker should be programmed to full output (maximum amplitude and pulse width), pacing at either DOO or VOO mode to maximize pacer stimulus artifact size. If the pacemaker lead is not a committed bipolar lead, then it should be reprogrammed to unipolar during testing. The ICD is programmed at the least sensitive setting, to set up a worst-case scenario. Testing of the ICD must then include determination that these spikes do not interfere with VF sensing. Alternatively, the preexisting pacemaker may be extracted and a dual-chamber ICD implanted to avoid pacemaker/ICD interactions.

Postprocedure Care

After successful pacemaker or ICD implantation, the primary risk in the early postprocedure period is lead dislodgment. Therefore we place the arm ipsilateral to the implant in a sling and keep the patient at bedrest overnight. After 24 hours the patient is encouraged to move the arm but is admonished not to lift any object weighing more than 10 pounds and not to raise the arm above shoulder level for 6 weeks. Anticoagulation is withheld for several hours to minimize the risk of bleeding and hematoma in the pocket.

As noted earlier, we prescribe prophylactic antibiotic therapy for 48 hours after implantation. Intravenous antibiotics may be switched to an oral equivalent if the patient is to be discharged before the end of 48 hours.

Before discharge, the pacemaker (or ICD) is interrogated to ensure that no marked changes in lead impedance, pacing threshold, or sensing have occurred. Such changes raise the possibility of lead dislodgment. A posteroanterior and lateral chest x-ray is also examined for stability of lead placement. As noted earlier, a portable chest x-ray is taken immediately after the procedure in the case of subclavian vein cannulation to exclude pneumothorax as well as to verify lead position. An immediate postprocedure chest x-ray does not change the need for the posteroanterior and lateral chest x-ray the following day. The patient is then asked to follow up in the Pacemaker Clinic 7 to 10 days after implant to evaluate wound healing and to interrogate the device.

There is controversy about whether routine noninvasive programmed stimulation (NIPS) is necessary before discharge of the ICD patient, to test for acute changes in DFT or lead problems. One study noted that in 97 patients undergoing routine predischarge testing, three had ineffective shocks at maximum device energy, despite an adequate safety margin during implant. No change in lead positions was detected on chest x-rays or under fluoroscopy in those patients (104). However, the devices implanted in that study were abdominal units, and these problems may be less likely with implantations of active can generators. At our institution, we do not routinely perform NIPS before discharge if lead positions are verified by chest x-ray and interrogation of the device is satisfactory.

Complications

As stated earlier, the risk associated with transvenous implantation of a permanent pacemaker or ICD is low (101,105,106). Nonetheless, complications do occur, and the patient should be apprised of the risks in the informed consent process before the procedure.

The patient should be told of the risk of bleeding and vascular injury inherent in any vascular procedure. Placement of leads is often accompanied by ectopy. Sustained tachycardia requiring therapy is rare, and it is uncommon that urgent cardioversion or defibrillation is necessary. However, personnel should be prepared to treat atrial and ventricular arrhythmias induced by lead manipulation.

There is a small risk of perforation of the thin-walled right ventricle or atrium with the leads. Vigilance is necessary and the index of
suspicion for pericardial tamponade should be high during and after the procedure. Tamponade may present as an apparent “vagal”
episode, though the heart rate will be supported by the pacemaker. Since bradycardia and chronotropic incompetence are common
indications for pacemaker implantation, the tachycardia that typically accompanies pericardial tamponade may be absent in the pacemaker
patient.

As noted earlier, there is a risk of pneumothorax associated with subclavian vein puncture, and a portable chest x-ray is recommended after
implantation using the subclavian vein for access.

Lead dislodgment is most likely to occur early after implantation (within a day). Therefore pacemaker interrogation and chest x-ray are
recommended the day following the procedure. In the event of lead dislodgment, lead revision should be carried out as soon as feasible to
minimize the scarring and fibrosis around the lead. When the inflammatory response has progressed far, lead revision and/or extraction
becomes more difficult and risky.

The most feared complication of pacemaker implantation is infection. If there is evidence of systemic infection (fever, positive blood
cultures), removal of the entire system (pacemaker and lead[s]) is indicated to allow antibiotic therapy to clear the infection completely.
This situation is even more unfortunate in the pacemaker-dependent patient, in whom a temporary pacemaker is often required between
the time of removal of the infected system and implantation of a new one. The gravity of the risk of infection should serve to emphasize
the need for attention to sterile technique, both at primary implantation and during generator change. Some procedure-related complications
are listed in Table 22.18.

Limitations of the Pacemaker or ICD Patient

Pacemaker implantation is intended to free the patient from health-related limitations, not impose additional ones. Patients, however, are
frequently anxious that their “condition” of having an implanted device will result in more illness, not less. The patient should be reassured
that after recovery from the implantation procedure, with the limitations discussed earlier (see “Postprocedure Care”), he or she should be
able to proceed with the normal activities of life.

The single most important limitation is that magnetic resonance imaging is contraindicated, since exposure to the strong magnetic field may
affect device function unpredictably. Similarly, exposure to other high electromagnetic fields, such as those produced by arc welding, is
contraindicated. Activities that cause direct trauma to the pulse generator are contraindicated.

Recent policies on driving advise patients with ICDs to avoid operating a vehicle for a minimum of 3 months, preferably 6 months, after
the last symptomatic arrhythmic event, or until a stable pattern of VT/VF can be established (107).

High-dose radiation therapy directly to the pulse generator can damage the device. In the case of the patient whose neoplasm is located
such that the pacemaker cannot be kept out of the field, it may be necessary to relocate the pulse generator. An entirely new device can be
implanted from the opposite side, or the pulse generator may be placed in the abdomen and the lead(s) tunneled to the new site.

Medical equipment such as x-ray equipment and computed tomography scanning equipment do not interfere with ICDs or pacemakers.
Similarly, common household appliances should have no effect on the devices. Many patients are concerned about exposure to microwave
ovens. Modern microwave ovens are well shielded, however, and pose no threat to the pacemaker or ICD patient. There has been recent
concern about airport metal detectors and antishoplifting devices. One publication reported multiple shocks delivered to a patient with an
ICD who had inadvertently stood within an antitheft device for a prolonged period of time (108). Usually, however, antitheft devices or
airport security systems do not cause interference problems if the patient does not linger within the device itself. Similarly, it is possible
that such a device might inhibit a pacemaker, though only temporarily. Like ICD patients, pacemaker patients should avoid long periods in
close proximity to such devices.

Recent concern has been raised about the effect of cellular phones on pacemakers and ICDs. A study to address this problem concluded
that ordinary use of a cellular phones poses very low risk for malfunction (109). Holding a cellular phone directly over the device is not
recommended. All hand-held electronic devices should be kept more than 6 inches away from the device.

Transthoracic defibrillation may damage the circuits of an implanted device if very high output is used or if the electrodes are in close
proximity to the pulse generator. When transthoracic shocks take place, the electrodes should be placed at least a few inches away from the
pulse generator, and it is recommended that the device be interrogated after transthoracic defibrillation has taken place.

Care and Follow-up of the Patient with a Permanent Pacemaker or ICD

Routine Pacemaker Follow-up
After the first follow-up visit to ensure adequate wound healing and consistency of sensing and thresholds, further follow-up should occur periodically. Interrogation of the pacemaker is discussed later. The Pacemaker Clinic visits following the first visit for a wound check can be timed to follow the postimplantation threshold rise. For example, a 1-month visit would be near the peak of the threshold rise. Another visit at 3 months would coincide with the threshold reaching the chronic state. The output of the pacing stimulus can then be reduced (maintaining an adequate safety margin) to maximize battery life.

Routine follow-up should always include a history of any new symptoms as well as an examination of the pocket site for erythema, edema, tenderness, or threatened erosion.

Transtelephonic follow-up can be used to follow battery status. A single-lead rhythm strip can be transmitted over the phone with and without a magnet applied to the pulse generator. The Pacemaker Clinic personnel can monitor for malfunction detectable by ECG. The ECG recorded while the magnet is applied gives an indication of the battery status for most pacemakers (see later discussion).

As the battery life declines, the elective replacement indicator (ERI) is signaled. Plans should be made at this time to undergo pulse generator change. At end of life (EOL), the mode of most pacemakers changes to signify need for generator change, which should proceed as soon as possible.

Routine ICD Follow-up

After discharge, all patients with ICDs must have regular, meticulous follow-up to ensure proper functioning of the system. Generally, the first follow-up visit is scheduled within 1 or 2 weeks after time of implant, to check that the wound is healing properly and that leads have not dislodged. Follow-up visits afterward should take place approximately every 3 to 6 months. During these visits, the patients are interviewed as to whether the device has delivered any shocks and whether the patients had any symptoms before the shocks. A review of all current medications is necessary because of the potential effects of all antiarrhythmic agents on the defibrillation threshold. Interrogation of the device should include checking the battery status, assessing sensing and pacing parameters, evaluating event logs to record the number of sustained and nonsustained ventricular arrhythmias, as well as the number of delivered and aborted shocks. Current devices are capable of automatically reforming capacitors. In older devices, however, manual reformation of capacitors is performed during interrogations.

Noninvasive testing during follow-up is performed in patients who have begun a new antiarrhythmic agent, to ensure adequate safety margin of DFT on the medication. Data on routine noninvasive testing in patients who have never received any shocks from the device are controversial, but many support annual testing to assess the integrity of the system (110).

Magnet Application

Pacemakers

Placement of a magnet over a pacemaker pulse generator can be a therapeutic, diagnostic, or prophylactic maneuver. Therapeutic use, as in pacemaker-mediated tachycardia, has already been discussed. The diagnostic and prophylactic uses of magnet application lie in its inhibition of all sensing.

Application of a magnet over the pacemaker pulse generator temporarily changes its mode to asynchronous (DOO, VOO, or AOO). The rate will vary from model to model and will change as the battery status changes. These changes are specific to each manufacturer and in many cases are related to the programmed rate. Armed with appropriate information from the manufacturer, the magnet rate can therefore be used as a simple method to evaluate the battery status, and this method is commonly used for transtelephonic pacemaker follow-up.

Magnet application is prophylactic whenever there is a risk of inhibition due to inappropriate sensing. A common example is the use of electrocautery for surgical procedures. The pacemaker in the inhibited mode may sense the electrocautery, and pacing will be inhibited. The likelihood of inappropriate sensing is reduced by the use of bipolar leads. In magnet mode, the pacemaker will not sense any activity and will pace asynchronously. Extracorporeal shock-wave lithotripsy (ESWL), electroconvulsive therapy (ECT), and electrical cardioversion are other instances in which electric fields are brought into close contact with the patient. As with electrocautery, the risk of serious interaction, such as reprogramming or permanent damage, with the pacemaker is small, particularly if the stimulus is far from the pulse generator and electrodes. Inhibition of pacing due to inappropriate sensing of the stimulus remains a risk. Short durations of stimulus can limit the inhibition to a few cardiac cycles at a time. As with cautery, however, magnet application is a simple way to eliminate inappropriate inhibition. Noise reversion circuits provide a further safety feature. If electrograms are sensed at a rate faster than the noise reversion rate, the pacing mode is changed to an asynchronous one. The noise reversion mode will persist until the pacemaker is reprogrammed. Interrogation of the pacemaker to ensure appropriate programming and function after any procedure using electrical equipment is reasonable. In the absence of any sign of malfunction, interrogation can be done on an elective basis.

Complications of asynchronous pacing are rare, though some may worry about the risk of induction of ventricular arrhythmia. It is true
that ventricular fibrillation is reliably induced by a shock timed to fall on the T wave for ICD testing. The amplitude of such a shock is usually 1 J or more orders of magnitude greater than that of a pacemaker stimulus.

**ICDs**

External stimuli can often result in oversensing and can trigger delivery of inappropriate shocks to the patient. For this reason when patients with ICDs undergo any surgery in which electrocautery will be used, they should have their device inactivated or have the detection suspended. The safest method is to turn off detection via a programmer. When the programmer is not available, a magnet may be used to suspend detection and therapy temporarily. When a magnet is applied over an ICD, detection and therapy will be suspended without affecting antibradycardia pacing. For most of the commonly used ICDs, such as Medtronic, Intermedics, Ventritex, and Telelectronics models, all detection and therapy are resumed when the magnet is removed. The new CPI devices, such as the Ventak Mini and AV series, behave slightly differently in response to magnet application. As with ICDs from other manufacturers, a magnet applied over CPI devices will temporarily inhibit detection and therapy. However, CPI devices have an optional “change tachy mode with magnet” feature that can be programmed on or off. When this feature is off, the ICD behaves similar to models from other manufacturers and will resume detection and therapy once the magnet is removed. If the “change tachy mode with magnet” feature is on, a magnet placed over the ICD for more than 30 seconds will permanently inactivate the device. A magnet placed over these devices will also produce an audible tone, which is synchronized to R waves when the device is active and continuous if the device is inactive. Listening to the tone is one method of determining whether the device has been permanently inactivated by the magnet. Interrogation of the device after removal of a magnet to ensure that there has been no change in the programming is always prudent.

**Interrogation of the Pacemaker or ICD**

The cardiology consultant is often asked to “interrogate” a pacemaker or ICD to rule out any malfunction because of symptoms in a patient who has an implanted device. In the case of a pacemaker, the consulting physician may be searching for a cause of symptoms apparently related to bradycardia, such as syncope, lightheadedness, and dyspnea. If there is no evidence of malfunction on ECG or telemetry, the search for malfunction can only be general. If there is electrocardiographic evidence of a specific failure (e.g., failure to capture or failure to sense), the search for the cause and solution may be more focused. In the case of an ICD, interrogation may be prompted by inappropriate antitachycardia therapy or by concern for malfunction in antibradycardia pacing.

Sometimes there is a desire to make a specific change in the programming of the device, rather than seek a malfunction. Examples include changing the mode or the rate. The detection criteria for tachyarrhythmia or the nature of antitachycardia therapy can be changed in an ICD. In pacemakers with rate responsiveness, its “aggressiveness” can be adjusted. That is, the rate and degree of acceleration and deceleration of the lower pacing rate can be adjusted. Exercise testing can be of assistance in optimizing these settings. In the patient who has experienced endless-loop tachycardia, the PVARP should be adjusted or the PMT intervention should be turned on. In the absence of any suspected failure, the desired changes can be made without seeking malfunction.

The technical details of interrogation vary with the programmer, and some experience is necessary (preferably with a knowledgeable tutor) to gain facility with each one. However, the principles of interrogation are the same regardless of manufacturer. If the goal is simply to screen for malfunction, the basic functions and parameters must be checked. Unfortunately, a detailed discussion of this procedure is beyond the scope of this chapter.

**Troubleshooting**

A complete list of possible problems is beyond the scope of this chapter. Some common ones are listed here. Some are remediable by programming; others need more extensive intervention.

**Undersensing**

Undersensing is usually manifested by the appearance of stimulus artifacts despite the presence of ECG signs of intrinsic activity in the chamber in question (i.e. P waves or QRS complexes). The amplitude of the sensed electrograms can be determined by interrogation (see earlier discussion). Notice that reducing the value (in millivolts) of the sensitivity setting increases sensitivity (and vice versa).

Some possible causes of undersensing are the following:

- Lead dislodgment with loss of contact between the electrode and the endocardium, usually accompanied by loss of capture or increase in capture threshold.
- Lead insulation deterioration. If the body of the lead is exposed, the exposed portion acts an electrode, and the “electrogram” (or lack thereof) attenuates the electrogram signal from the true electrode.
- Inflammatory reaction. The inflammation initiated by lead implantation can affect sensing as well as pacing by insulating the electrode from the myocardium.
Decrease in amplitude of the electrogram. This may be reversible, as in the case of added medication (especially antiarrhythmic agents) or metabolic abnormalities, such as hyperkalemia or acidosis. Alternatively, the cause may be irreversible, as in the case of progressive cardiomyopathy or new myocardial infarction.

Programming error. This error can be avoided by carefully reviewing the programmed parameters when finishing a programming session.

In some cases, these problems may only be remedied by invasive correction, such as revising or replacing the lead. In other cases, reprogramming the sensitivity can correct the problem. Nominal sensitivity of the atrial channel is usually 0.5 to 1.0 mV; for the ventricular channel, 1.5 to 2.5 mV is typical. For most pacemakers the sensitivity can be increased if undersensing occurs.

**Oversensing**

Oversensing is manifest on the surface ECG by the absence of an expected pacing stimulus artifact. A pacing spike is expected when a pause exceeds the length allowed by the pacemaker's lower rate limit. It should be kept in mind that bipolar pacing spikes may be very low amplitude and may not be visible in all leads. Both physiologic phenomena and extrinsic signals may be inappropriately sensed as electrograms.

- Myopotential sensing. Skeletal muscle myopotentials are rarely sensed in bipolar lead systems, but this problem may occur in unipolar systems with high sensitivity settings.
- T-wave sensing. Usually, the T-wave amplitude is low relative to the R wave, and adjustment of the sensitivity is effective.
- Lead fracture. Intermittent contact of the lead fragments can create noise; the amplitude may be high enough to be sensed as electrograms.
- Inner insulation failure. This may allow contact between the conductor of the two electrodes in a bipolar lead, and the resulting noise may be inappropriately sensed.
- External electromagnetic interference. External signals may be inappropriately sensed as electrograms. These include surgical electrocautery, magnetic resonance imaging, extracorporeal shock wave lithotripsy, transcutaneous nerve stimulation, radiofrequency ablation, and arc welding. These sources of inappropriately sensed impulses are rarely difficult to diagnose. As discussed earlier, temporary programming to asynchronous pacing (as with magnet application) may be appropriate in some cases (e.g., magnetic resonance imaging).
- Inappropriate programming.

Oversensing can be disastrous in the case of a pacemaker-dependent patient. If oversensing results in complete inhibition of pacing, magnet application is the appropriate immediate action. Pacing will be asynchronous, and the oversensed signals will be irrelevant. The sensitivity should be checked with the programmer and reprogrammed if indicated. Further evaluation, including interrogation of lead impedance and examination of the chest x-ray for fracture, is required.

**Abnormal Lead Impedance**

Abnormally high lead impedance suggests a discontinuity in the circuit of the lead in question. The most likely cause is fracture of the lead. The two parts of the divided lead may be in contact intermittently, which may make the abnormal lead impedance (and failure) intermittent. If the lead fragments are separated by air, the impedance will approach infinity and will not function.

Abnormally low lead impedance suggests a short circuit in the system. This may be due to loss of integrity of the lead insulation, lead dislodgment, or lead perforation.

Lead failure can rarely be solved merely by programming. Dislodged leads can be revised, but fractured leads must be replaced. Insulation failure is often in the part of the lead in the pacemaker pocket and results from friction against the pulse generator. These failures can be sought and often repaired. If the damage cannot be found or malfunction persists after repair, the lead must be replaced to regain full function. If a bipolar lead has damaged outer insulation, the distal electrode (and inner conductor) can be used as a unipolar lead (if the pulse generator is capable of unipolar pacing).

**High Capture Threshold**

The pacing stimulus output should exceed the capture threshold with a comfortable safety margin. For a chronic lead (older than 8 weeks) the safety margin should be twice the amplitude threshold or three times the pulse width threshold. An acute change in the threshold of a chronic lead raises the possibility of disruption of the system. Lead impedance should be checked and a chest x-ray should be obtained to exclude lead dislodgment.

The possibility of perforation of the myocardium by the lead should be borne in mind. Good sensing with poor pacing may be a sign of perforation, since the ring electrode of a bipolar lead is likely to remain in contact with the myocardium when the tip of the lead has
advanced through to the ventricular wall. Pacing of the diaphragm at relatively low outputs is also a strong indicator that the lead has perforated.

As noted earlier, an acute (4 to 6 weeks) threshold rise is common after lead implantation. Therefore higher output should be programmed at the time of implantation. Some medications and electrolyte imbalances may change pacing thresholds, making a greater margin of safety desirable in patients likely to undergo changes in these factors.

**Battery Depletion**

Pulse generator change should occur soon after the ERI (elective replacement indicator) appears, since battery depletion to this extent does not change pacemaker function. In some pacemakers, when the battery reaches EOL (end of life), there is a change in the rate and/or mode, which may be symptomatic. The modes and rates are specific to individual pacemakers. These need not be memorized, since an unexpected change of mode should prompt interrogation. Battery depletion should not come as a surprise under normal circumstances with good follow-up. However, if some change occurs that dramatically increases current used to pace (such as a large decrease in lead impedance), the rate of battery depletion may increase rapidly. Only rarely will battery depletion cause frank failure. In this case, the programmer may no longer be able to communicate with the pacemaker. Such a failure can be catastrophic in the pacemaker-dependent patient; therefore the follow-up should be particularly frequent as the life of the battery nears its end.