PERSPECTIVE

Electrical cardiac pacing for the management of bradyarrhythmias was first described in 1952, and permanent transvenous pacing devices were introduced into clinical practice in the early 1960s. The first devices for endocardial defibrillation were implanted in surviving victims of sudden cardiac death in 1980. Implanted electrical devices for the management of cardiac dysrhythmias have changed rapidly over the years, with both increasing complexity and miniaturization. Survey data from 2002 indicated that approximately 612 new pacemakers per million population were implanted in the United States. Indications for the use of permanent pacemakers in the management of congenital and acquired heart disease include cardiac resynchronization therapy for heart failure.

A number of large clinical trials comparing implantable cardioverter-defibrillators (ICDs) with antiarrhythmic drugs for the prevention of sudden cardiac death resulting from ventricular dysrhythmias have indicated that ICDs significantly improve survival. Such studies have led to a dramatic increase in ICD implantations, and it is estimated that there are more than 125,000 new ICD implants annually in the United States. The widespread use of these devices ensures that the emergency physician frequently encounters such patients, often with symptoms that may be related to malfunction of the pacemaker or ICD.

INDICATIONS FOR PERMANENT PACEMAKERS AND ANTIARRHYTHMIA DEVICES

Guidelines for the implantation of these devices have been developed by a joint task force of the American Heart Association (AHA) and the American College of Cardiology (ACC) and are periodically updated. With use of an evidence-based approach, recommendations are categorized as class I, II, or III. Class I includes conditions for which there is general agreement that a device should be implanted. A class II recommendation includes conditions for which these devices are frequently used but for which there is disagreement about their need or benefit. Class III is reserved for conditions for which there is general agreement that a device is not needed.

In the case of pacemaker therapy, additional factors are considered when selecting the mode of pacing and include, but are not limited to, overall health, lifestyle, and occupation of the patient. Class I indications for a permanent pacemaker or ICD are listed in Boxes 80-1 and 80-2. In general, pacing is recommended for patients with symptomatic heart block, symptomatic sinus bradycardia, and atrial fibrillation with a symptomatic bradycardia (low ventricular response rate) in the absence of medications that affect atrioventricular (AV) conduction. Controversial indications include pacing in patients with syncope, heart block, or fatigue in the presence of some conduction disease or bradycardia. The likelihood of a patient’s improvement after pacing can be assumed only if the symptoms can be closely correlated with inadequate rate.

Pacemaker Terminology

A letter code, initially established in 1974 and revised as technology advances, standardizes nomenclature for pacemakers. Table 80-1 includes an explanation of the five-letter code scheme and the standard abbreviations in each category. The first three code letters are used most commonly. Using this table, one should be able to understand the features of any pacing mode. For example, a VDD pacemaker is capable of pacing only the ventricle, sensing both atrial and ventricular intrinsic depolarization, and responding by dual inhibition of both atrial and ventricular pacing if intrinsic ventricular depolarization occurs; a paced ventricular beat is triggered in response to a sensed intrinsic atrial depolarization. The codes of a permanent pacemaker that are used most frequently and the indications, advantages, and disadvantages of each are listed in Table 80-2. Detailed algorithms for matching a patient with a pacemaker exist. The majority of permanent pacemakers are dual chamber and often rate adaptive.

Pacemaker Components

All pacemaker systems have three basic components: the pulse generator, which houses the power source (battery); the electronic circuitry; and the lead system, which connects the pulse generator to the endocardium.

Nearly all implanted pacemakers are lithium powered. Lithium-powered pulse generators function normally for 4 to 10 or more years, depending on the pacemaker features, such as single versus dual chamber, pacing threshold, and rate adaptiveness. This long “battery life” and the fact that the output voltage of the lithium-iodine cell decreases gradually rather than abruptly, as occurred with the early mercury-zinc cell, make sudden pulse generator failure an unlikely cause of pacemaker malfunction.

Permanent pacemakers have endocardial leads that are positioned in contact with the endocardium of the right ventricle and, in the case of a dual-chamber device, the right atrium, with a subclavian or cephalic vein approach used for insertion. Occasionally, an epicardial lead may be implanted during open-heart surgery performed for another indication, such as prosthetic valve insertion or correction of a congenital cardiac defect. Pacemaker leads, like power sources, continue to undergo major technical advances.
improvements. Innovations include resilient plastic insulation surrounding the electrodes that reduces the chance of complete lead disruption or breakage (resulting in failure to pace or sense) and the chance of partial fracture (resulting in a “make or break” contact with intermittent failure to sense or pace). Despite these advances, problems with the electrical circuitry remain the most common cause of pacemaker malfunction. A lead capable of active fixation is more commonly used in patients with cardiomyopathies and right ventricular dilation complicated by tricuspid regurgitation.

Pacemaker leads may be either bipolar or unipolar in configuration. A bipolar endocardial lead has both the negative (distal) and the positive (proximal) electrodes, separated by approximately 1 cm, within the heart. A unipolar lead has the negative electrode in contact with the endocardial surface, and the positive pole is the metallic casing of the pulse generator. Each lead system has potential advantages and disadvantages. The unipolar configuration is not compatible with ICD systems and is prone to oversensing of myopotentials and electromagnetic interference but is of smaller diameter and less susceptible to fracture. The bipolar configuration is compatible with ICD systems but is larger and more prone to lead fractures. Oversensing, however, is rarely a problem. The selection of lead configuration usually depends on the experience and preference of the operator.

### Box 80-1 Class I Indications for Permanent Pacing in Adults

1. Third-degree and advanced second-degree AV block at any anatomic level associated with any of the following:
   - Symptomatic bradycardia (including heart failure) or ventricular dysrhythmia presumed to be a result of AV block
   - Symptomatic bradycardia secondary to drugs required for dysrhythmia management or other medical condition
   - Documented periods of asystole lasting more than 3 seconds or an escape rate of less than 40 beats/min or an escape rhythm originating below the AV node in an awake, asymptomatic patient in sinus rhythm
   - Awake, asymptomatic patients with atrial fibrillation and bradycardia a documented pause of 5 seconds or longer
   - After catheter ablation of the AV node
   - Postoperative AV block that is not expected to resolve
   - Neuromuscular disease with AV block (e.g., the muscular dystrophies)

2. Symptomatic bradycardia resulting from second-degree AV block regardless of type or site of block
3. Asymptomatic, persistent third-degree AV block with awake ventricular rate over 40 beats/min with cardiomegaly or left ventricular dysfunction or if block is below AV node
4. Chronic bifascicular or trifascicular block with intermittent third-degree AV block or type II second-degree AV block
5. Second or third-degree AV block with exercise in the absence of myocardial ischemia

AV, atrioventricular.

### Table 80-1 Five-Letter Pacemaker Code

<table>
<thead>
<tr>
<th>CHAMBER PACED</th>
<th>CHAMBER SENDED</th>
<th>SENSING RESPONSE</th>
<th>PROGRAMMABILITY</th>
<th>ANTITACHYCARDIA FUNCTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>A = atrium</td>
<td>A = atrium</td>
<td>T = triggered*</td>
<td>P = simple</td>
<td>P = pacing</td>
</tr>
<tr>
<td>V = ventricle</td>
<td>V = ventricle</td>
<td>I = inhibited</td>
<td>M = multiprogrammable</td>
<td>S = shock</td>
</tr>
<tr>
<td>D = dual</td>
<td>D = dual</td>
<td>D = dual (A and V inhibited)</td>
<td>R = rate adaptive</td>
<td>D = dual (shock pace)</td>
</tr>
<tr>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
<td>C = communicating</td>
<td></td>
</tr>
</tbody>
</table>

*In the triggered response mode, the pacemaker discharges or fires when it recognizes an intrinsic depolarization. As a result, pacemaker spikes occur during inscription of the QRS complex. Because this mode results in high energy consumption and a shortened battery life and because the sensing response can be misinterpreted as pacemaker malfunction, this sensing mode is not used with modern pacemakers.

### Table 80-2 Common Permanent Pacemakers

<table>
<thead>
<tr>
<th>CODE</th>
<th>INDICATION</th>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>VVI</td>
<td>Intermittent backup pacing; inactive patient</td>
<td>Simplicity; low cost</td>
<td>Fixed rate; risk of pacemaker syndrome</td>
</tr>
<tr>
<td>VVIR</td>
<td>Atrial fibrillation</td>
<td>Rate responsive</td>
<td>Requires advanced programming</td>
</tr>
<tr>
<td>DDD</td>
<td>Complete heart block</td>
<td>Atrial tracking restores normal physiology</td>
<td>No rate responsiveness; requires two leads and advanced programming</td>
</tr>
<tr>
<td>DDR</td>
<td>Sinus node dysfunction; for rate responsiveness atrioventricular block and need</td>
<td>Universal pacer; all options available by programming</td>
<td>Complexity, cost, programming, and follow-up evaluation</td>
</tr>
</tbody>
</table>

VF, ventricular fibrillation; VT, ventricular tachycardia.
The Standard Electrocardiogram during Normal Cardiac Pacing

The modern pacemaker has two basic functions: to stimulate the heart electrically and to sense intrinsic cardiac electrical activity. Additional functions are available and are noted in the pacemaker code system (see Table 80-1, letters 4 and 5). The pacemaker delivers an electrical stimulus to either the atrium or the ventricle if it does not recognize (sense) any intrinsic electrical activity from that chamber after a selected time interval. This interval is usually programmed at the time of implantation and can be changed noninvasively at a later time, if necessary, with use of a programming and “interrogating” device provided by the pacemaker manufacturer. If the pacemaker recognizes or senses an intrinsic atrial depolarization (P wave) or ventricular depolarization (QRS complex), it inhibits or resets its output to prevent competition with the underlying intrinsic rhythm. The stimulus intensity and sensing threshold (amplitude of electrical activity that is detected as being intrinsic) are typically set at the time of implantation but can also be reprogrammed later.

The two basic functions of a pacemaker can be easily recognized and confirmed on a standard 12-lead electrocardiogram (ECG) or rhythm strip. The normal function of a single-chamber VVI pacemaker is most easily recognized (Fig. 80-1). After a programmed interval is surpassed during which intrinsic ventricular activity does not occur, a pacer “spike” or stimulus artifact appears. The pacer spike is a narrow deflection that is usually less than 5 mm in amplitude with a bipolar lead configuration and usually 20 mm or more in amplitude with a unipolar lead. A wide QRS complex appears immediately after the stimulus artifact. Depolarization begins in the right ventricular apex, and the spread of excitation does not follow normal conduction pathways. Characteristically, a left bundle branch block conduction pattern is seen. A right bundle branch pattern is abnormal and suggests lead displacement. In VVI pacing the paced QRS complexes are independent of intrinsic atrial depolarization if present (AV dissociation).

The recognition of normal dual-chamber pacing is more complex owing to the interactive sensing and pacing of the right atrium and ventricle (Fig. 80-2).14 Pacing intervals are preprogrammed, may be changed noninvasively at a later time, and are generally specific to the patient’s needs. Pacing rates and delay intervals typically vary from patient to patient. Dual-chamber devices are typically used in patients with nonfibrillating atria.

Figure 80-1. Normal VVI pacemaker (rhythm strip). This rhythm strip was recorded in a patient with a VVI pacemaker implanted for the treatment of symptomatic complete heart block. The pacing rate is approximately 75 beats/min (determined by measuring the time between consecutive pacemaker spikes). Each pacemaker spike is followed by a paced QRS complex. The third QRS from the left has a slightly different morphology than the paced QRS complexes. It is an intrinsic QRS complex that is sensed by the pacemaker, and a paced beat does not occur again until the programmed rate of the pacemaker is exceeded. The time interval between the spontaneous QRS and the next paced beat is approximately the same as the interval between consecutive pacemaker spikes. This sequence is subsequently repeated twice on this strip.

Figure 80-2. Normal DDD pacemaker (12-lead electrocardiogram). Each QRS complex is preceded by two pacemaker spikes. The first spike results in atrial depolarization, and the second produces a wide QRS complex. The QRS complex is conducted with a left bundle branch morphology, which is expected with endocardial pacing at the right ventricular apex.
coupled with intact AV conduction. A normal-appearing QRS complex may follow an intrinsic “P” wave as a result of normal sinoatrial node discharge if the intrinsic atrial depolarization is conducted to the ventricles. The intrinsic p wave and QRS complex inhibit the atrial and ventricular circuitry. A normal QRS complex is followed by a paced p wave if the paced atrial beat is conducted through the AV node and the programmed AV delay period is not exceeded. If it is not conducted to the ventricles (AV delay period exceeded), the pacemaker stimulates the ventricle, resulting in a paced p wave and a wide, paced QRS complex with left bundle branch block configuration.

Recognition of the interactivity of the paced chambers is important. A paced p wave may be mistaken for failure to sense or pace, and malfunction may be diagnosed when it is not present (pseudomalfunction). In addition, if the programmed rate of the pacemaker approximates the patient’s intrinsic heart rate, fusion of paced and native beats may occur and represents another common type of pseudomalfunction (Fig. 80-3).

Complications of Implantation

Infection

Pacemaker implantation is a surgical procedure and, like all surgery, carries a risk of infection; the presence of a foreign body enhances this risk. The incidence of infection is small—approximately 2% for wound and subcutaneous pacemaker “pocket” infection and approximately 1% for bacteremia with sepsis. The presence of a foreign body complicates management, and few cases of bacteremia that develop after implantation can be managed with antibiotics alone. In most instances, reimplantation and replacement of the lead system are necessary.13

Pain and local inflammation at the site of the pacemaker are the first manifestations of a wound infection, cellulitis, or pocket infection. Approximately 20 to 25% of patients with a local infection have positive blood cultures. Bacteremia may occur in the absence of a focal infection and may arise with the typical manifestations of the systemic inflammatory response syndrome or sepsis. A hematoma of the pacemaker pocket may mimic a wound or pocket infection. Needle aspiration of the pocket should be done only under fluoroscopy because the needle may cut the insulation surrounding the pulse generator or the portion of the pacemaker lead that lies within the pacemaker pocket.

When a local infection or bacteremia is suspected, blood cultures should be obtained and intravenous antibiotic therapy initiated. Staphylococcus aureus and Staphylococcus epidermidis are isolated in approximately 60 to 70% of cases. Empirical antibiotic therapy should include vancomycin pending culture and sensitivity data. If blood cultures are positive, the pulse generator and pacemaker lead are usually removed, temporary transvenous pacing is performed, and intravenous antibiotic therapy is continued for 4 to 6 weeks. The permanent pacemaker and lead are subsequently reimplanted.15

Thrombophlebitis

The incidence of venous obstruction associated with permanent transvenous pacemakers ranges from 30 to 50%, with approximately one third of patients having complete venous occlusion.16 Thrombosis of varying degrees can involve the axillary, subclavian, and innominate veins or the superior vena cava (SVC). The site of insertion does not appear to affect the incidence of this complication. Chronic thrombosis of the veins of the upper arm is common and usually asymptomatic owing to extensive venous collateral circulation.

Because of extensive collateralization, only approximately 0.5 to 3.5% of patients develop symptoms usually indicative of acute thrombosis. These patients typically have edema, pain, and venous engorgement of the arm ipsilateral to the site of lead insertion. Although rare, SVC syndrome resulting from pacemaker lead-induced thrombosis occurs. The symptoms and signs of lead-induced SVC syndrome are identical to those described in patients with SVC syndrome and malignancy. Whether pulmonary embolism is associated with pacemaker therapy and thrombosis is controversial.

Although symptoms might suggest thrombosis, definitive diagnosis of acute thrombosis usually requires duplex sonography of the jugular venous system or contrast-enhanced computed tomography. The symptoms typically respond to intravenous heparin therapy followed by long-term warfarin administration. Directed thrombolytic therapy is most effective if used early in management (within 7-10 days).

The “Pacemaker Syndrome”

After pacemaker implantation, a patient may develop new complaints or report a worsening of the symptoms that prompted evaluation and eventual pacemaker therapy. Such complaints often include syncope or near-syncope, orthostatic dizziness, fatigue, exercise intolerance, weakness, lethargy, chest fullness or pain, cough, uncomfortable pulsations in the neck or abdomen, right upper quadrant pain, and other nonspecific symptoms.

These symptoms are termed the pacemaker syndrome.17 The cause of this syndrome is the loss of AV synchrony and the presence of ventriculoatrial conduction, and it is most commonly encountered in the setting of VVI pacing. It is also described with the DDI mode. With VVI pacing, the ventricle is electrically stimulated and depolarized, resulting in ventricular systole. If sinus
node function is intact, the atria can be depolarized by a sinus impulse and contract when the tricuspid and mitral valves are closed. This contractile asynchrony results in an increase in jugular and pulmonary venous pressures and may produce symptoms of congestive heart failure.

Atrial distention can result in reflex vasodepressor effects mediated by the central nervous system. Elevated levels of B-type natriuretic peptide and diuresis are considered markers for the syndrome in its more severe form. If the contribution of atrial contraction to late diastolic ventricular filling is important in maintaining an adequate cardiac output, basal and orthostatic hypotension may occur. DDI pacing in a patient with AV block may result in this syndrome if the sinus node discharge rate exceeds the programmed rate of the pacemaker.

Approximately 20% of patients report symptoms suggesting the pacemaker syndrome after pacemaker insertion. In most instances, symptoms are mild and patients adapt to them. In approximately one third of these patients, symptoms are severe. Treatment usually requires replacing a VVI pacemaker with a dual-chamber pacemaker or lowering the pacing rate of the VVI unit. If symptoms occur in a patient paced in the DDI mode, optimizing the timing of atrial and ventricular pacing is usually required. Patients appear to prefer dual-chamber pacing to the VVI modality. Persistence or worsening of symptoms of congestive heart failure may also be caused by the altered ventricular activation sequence inherent with stimulation of the right ventricle. Biventricular pacing may be required in selected patients.

Although the pacemaker syndrome may be suspected in the ED in the patient with suggestive symptoms soon after pacemaker implantation, consultation with a cardiologist for interrogation of the pacemaker is recommended. The same symptoms may be observed in patients with true pacemaker malfunction, which may necessitate pacemaker reprogramming or replacement of the pulse generator or pacemaker lead.

Pacemaker Malfunction

The term pacemaker malfunction refers specifically to problems with the circuitry or power source of the pulse generator, the pacemaker lead (most commonly displacement or fracture), or the interface between the pacing electrode and the myocardium (pacing or sensing threshold). In addition, environmental factors, such as extracardiac or extracorporeal electrical signals, may interfere with normal pacemaker function. With use of the standard ECG, pacemaker malfunction can be separated into three broad categories: (1) failure to capture (no pacemaker spikes or spikes not followed by an atrial or ventricular complex), (2) inappropriate sensing (oversensing or undersensing spikes occur prematurely or do not occur even though the programmed interval is exceeded), or (3) inappropriate pacemaker rate. Symptomatic pacemaker malfunction after implantation occurs in less than 5% of patients and is rarely immediately life-threatening. Malfunction is most commonly a result of inappropriate sensing, followed by failure to capture. Typical presentations and causes of pacemaker malfunction are listed in Box 80-3.

In the context of suspected pacemaker malfunction, knowledge of the pacing modalities (see Table 80-1) and what is normal for a given pacing modality are critical when the ECG is reviewed. Fortunately, patients are provided with important identifying information, usually in the form of a wallet card, after pacemaker implantation. The most important information is provided in the five-letter code. If this information is not available, a standard posteroanterior and a lateral chest radiograph can provide critical information. A single lead in the apex of the right ventricle indicates a VVI pacemaker. With VVI pacing, only one stimulus artifact or spike is seen with each stimulated ventricular depolarization (see Fig. 80-1). If sinus node activity is present, the paced QRS complex is dissociated from the intrinsic P waves. If separate leads are identified in the right atrium and right ventricle, the pacing modality is most often DDD or DVI, and paced p waves and QRS complexes (two spikes for each QRS complex) are seen (see Fig. 80-2). Although DDD and DVI units are capable of pacing both the right atrium and the right ventricle, only one spike may be seen (Fig. 80-4). Failure to identify two spikes with a DDD or DVI unit can represent normal pacemaker function.

A magnet placed externally over the pulse generator is frequently used in the assessment of pacemaker function. Magnet application causes closure of a reed switch within the pacemaker circuitry, converting the pacemaker to an asynchronous or fixed-rate pacing mode, and the pacemaker is no longer inhibited by the patient’s intrinsic electrical activity. The technique is most commonly used when the patient’s intrinsic heart rate exceeds the pacemaker’s set rate and pacemaker function is inhibited. Magnet application then allows pacing to occur, despite the patient’s native cardiac activity, and pacing rate and the presence of capture can be determined. Magnets are made by each manufacturer, but any cardiac pacemaker magnet will typically activate the reed switch within any of the devices.

Failure to Capture

Failure to capture may range from the complete absence of pacemaker spikes to spikes not followed by a stimulus-induced complex (Fig. 80-5). A complete absence of pacemaker spikes may result from battery depletion, fracture of the pacemaker lead, or disconnection of the lead from the pulse generator unit.

Current lithium-iodine batteries are not subject to sudden power failure, and they display typical end-of-life functional changes over a period of months to a year before complete depletion. Usually the first sign of voltage depletion is a decrease in the programmed pacing rate. This change is gradual and should be detected during the regular follow-up evaluations that pacemaker patients receive. When voltage output falls to a critical level, stimulus strength falls below the required threshold, and failure to capture or intermittent failure to capture may be observed late in battery life. As a result, urgent or emergent battery replacement is rare.

Failure to capture, which may be complete or intermittent, is most commonly a lead problem. Lead displacement is the most common cause and is most likely to occur within the first month of pacemaker insertion. The chest radiograph may demonstrate

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**Box 80-3 Causes of Pacemaker Malfunction**

**Failure to Capture**
- Lead disconnection, break, or displacement
- Exit block
- Battery depletion

**Undersensing**
- Lead displacement
- Inadequate endocardial lead contact
- Low-voltage intracardiac P waves and QRS complexes
- Lead fracture

**Oversensing**
- Sensing extracardiac signals: myopotentials
- T wave sensing

**Inappropriate Rate**
- Battery depletion
- Ventriculoatrial conduction with pacemaker-mediated tachycardia
- 1:1 response to atrial dysrhythmias
Figure 80-4. Normal DDD pacemaker (half-standard 12-lead electrocardiogram [ECG]). Three paced QRS complexes preceded by a stimulus artifact or spike are evident in leads I, II, and III. Paced QRS complexes occur after spontaneous or intrinsic P waves are sensed and atrioventricular (AV) conduction delay exceeds the pacemaker’s programmed AV interval. The first QRS complex in the augmented leads, best seen in lead aVF, demonstrates both a paced P wave and a paced QRS complex. Although the pacemaker is a dual-chamber device, two spikes may not always be seen preceding every QRS complex, and the presence of only one spike, or no spikes, should not be interpreted as evidence of pacemaker malfunction. Also evident on this ECG are the different amplitudes of the pacemaker spikes from lead to lead. When a single-lead rhythm strip is recorded, the selected lead should be the one in which the pacemaker spikes are most easily identified.

Figure 80-5. Intermittent failure to capture and slow pacing rate (lead I). This lead I rhythm strip demonstrates intermittent failure to capture of a VVI pacemaker. The first and second pacemaker spikes are followed by wide-paced QRS complexes; the third and fourth spikes are not. The pacemaker spikes occur at a rate of approximately 50/min. The device was programmed to pace at a rate of 75/min. This is a typical example of “end-of-life” pacing characteristics of a depleted battery.

Exit block (the failure of an adequate stimulus to depolarize the paced chamber) can also result in failure to pace. Exit block should be considered when the preprogrammed pacing stimulus output fails to result in capture in the presence of a normally functioning pulse generator and an intact lead system. This problem is most commonly a result of changes in the endocardium in contact with the pacing system. Causes include ischemia or infarction of the endocardium in contact with the electrodes; systemic hyperkalemia; and the use of class III antiarrhythmic drugs, such as amiodarone, which affect ventricular depolarization. Although other drugs alter pacemaker threshold, the effect is small and is rarely clinically important. Exit block is usually noted as a change in pacing threshold during pacemaker interrogation. Lead fractures occur at predictable locations, usually at the site of attachment to the pulse generator or at abrupt angulations that serve as stress points. Inadequate contact of the lead with the pulse generator can mimic a lead fracture. Occasionally, when a lead fracture is complete or nearly complete, a break in the catheter or its insulation can be detected on an overpenetrated posteroanterior chest radiograph. Loss of lead-pulse generator contact can be detected on the chest radiograph with close inspection of the pulse generator.

Inappropriate Sensing
For a pacemaker to function in a noncompetitive mode, it must be capable of sensing the intrinsic or “native” electrical activity of
the heart. The electrical activity that is sensed is determined by the pacing modality (see Table 80-1). Sensing parameters are determined at the time of pacemaker insertion on the basis of the signal size of the intracardiac ECG and can be changed or fine-tuned externally at a later time if needed.

### Undersensing

Failure to sense may be complete or intermittent. It may result from a change in the sensing parameters selected at the time of insertion. This is most commonly encountered after acute right ventricular infarction or during the progressive fibrosis that accompanies many cardiomyopathies, causing intracardiac signals to decrease in amplitude. Lead displacement, fracture, and poor contact with the endocardium may also cause undersensing.

Undersensing is typically recognized electrocardiographically as the appearance of pacemaker spikes occurring earlier than the programmed rate. The spike may or may not be followed by a paced complex, depending on when it occurs during the cardiac refractory period (Fig. 80-6). Failure of a stimulus spike to produce a complex when it occurs during the atrial or ventricular refractory period should not be interpreted as failure to pace.

### Oversensing

In rare instances the pacemaker may detect electrical activity that is not of cardiac origin. The result may be intermittent, irregular pacing or an apparent complete absence of pacemaker function. Myopotentials produced by the pectoralis muscle (Fig. 80-7) and extracorporeal electrical signals are frequently oversensed when a unipolar lead system is used. T waves that follow an intrinsic ventricular depolarization are the most common oversensed cardiac signals. Common medical sources of electrical interference include electrocautery, which can cause temporary pacemaker inhibition, and magnetic resonance imaging, which can alter pace-

### Inappropriate Pacemaker Rate

A pacing rate below the programmed rate is a typical finding in pulse generator depletion and does not occur abruptly with lithium-iodine batteries. An extreme increase in pacing rate, the so-called “runaway pacemaker,” is rarely, if ever, encountered with current pacemaker technology and circuitry in which upper rate limits are set (typically 140 beats/min). An “endless loop” tachyar-

### Management

#### History

The patient should be asked for the pacemaker identification card. The information on the card explains why a pacemaker was placed and the pacing modality used.

Most patients with pacemaker malfunction have symptoms reminiscent of those that prompted pacemaker therapy: syncope, near-syncope, orthostatic dizziness, lightheadedness, dyspnea, or palpitations.

The majority of pacemaker complications and most instances of pacemaker malfunction occur within the first few weeks or months of pacemaker implantation. After wound healing,
The pacemaker syndrome should be a diagnosis of exclusion. Syncope or near-syncope may also occur, but these complaints may be caused by fluttering or “pounding” sensation in the neck or abdomen. Fatigability, generalized weakness, dyspnea, or an uncomfortable sensation in the chest may occur. Acute thrombophlebitis, manifested by pain in the arm ipsilateral to the site of insertion, should suggest a diagnosis of exclusion.

Patients who develop the pacemaker syndrome secondary to the loss of AV synchrony may have nonspecific complaints of easy fatigability, generalized weakness, dyspnea, or an uncomfortable sensation in the chest. Syncope or near-syncope may also occur, but these complaints should prompt an evaluation for true pacemaker malfunction. The pacemaker syndrome should be a diagnosis of exclusion.

Physical Examination

A pacemaker infection should be suspected in the presence of fever, even if another potential source of infection can be identified. Extremely low (<60 beats/min) or high (>100 beats/min in the resting patient) pulse rates are suggestive of altered pacing parameters. Hypotension may be present in either instance. Cannon “A” waves on inspection of the jugular venous pulse wave indicate AV asynchrony. Auscultation of lungs may reveal bubbling rales if congestive heart failure is present.

During pacing, the first heart sound may vary in intensity as a result of AV dissociation (VVI mode), and the second heart sound may be paradoxically split when ventricular pacing occurs (the right ventricle is activated first). A pericardial friction rub may be audible if the tip of the pacing catheter has perforated the wall of the right ventricle. Perforation, however, usually occurs at the time of pacemaker implantation and is usually recognized at that time. Although the pacing catheter traverses the tricuspid valve, tricuspid regurgitation is rarely heard unless there is myocardial disease such as right ventricular dilation, which is common in the cardiomyopathies. Pedal edema may be present and is important if it is a new symptom or if chronic edema has recently worsened.

Chest Radiograph

A chest radiograph should be obtained to define pacing catheter tip position and to determine the number of pacing leads, unless this information is available from another source. A ventricular pacing catheter tip in the right ventricular outflow tract or an atrial catheter tip in the SVC or right ventricle is abnormal. The pulse generator site should also be examined on the radiograph.

12-Lead Electrocardiogram

A standard ECG and a long rhythm strip should be obtained in all patients. With bipolar pacing systems, the stimulus artifact may be extremely small and difficult to recognize in some leads (see Fig. 80-4). Inspection of the rhythm strip may reveal failure to sense or pace, a low pacing rate, or an abnormally rapid rhythm, suggesting a pacemaker-mediated tachycardia.

Disposition of the Emergency Department Patient with a Pacemaker

As a result of the current design of modern pacemakers and the frequent follow-up evaluation of patients with pacemakers, life-threatening emergencies resulting from pacemaker malfunction requiring immediate ED intervention are rare. Most instances of malfunction are subtle and difficult to recognize without interrogation of the pacemaker with manufacturer-specific devices by someone skilled in the technique. In all instances of suspected pacemaker malfunction, the patient’s cardiologist should be consulted.

Advanced Cardiac Life Support Interventions

Electrical defibrillation at recommended shock strengths (200, 300, and 360 J) can be safely performed in the patient with a pacemaker. If the sternal paddle is placed adjacent to the sternum, it is at a safe distance (>10 cm) from the pulse generator. Alternatively, defibrillation electrodes can be placed in an anteroposterior configuration. A cardiologist or technician should ensure that the pacing parameters of the unit are not altered if the resuscitation is successful. A chest radiograph should also be obtained after resuscitation to ensure that the pacing catheter was not displaced during chest compression, although this is an extremely uncommon occurrence.

Immediate return of pacing (capture) may not occur after defibrillation; this is commonly the result of global myocardial ischemia and increased pacing threshold and is not an indication of pacemaker malfunction. Temporary transcutaneous pacing may be needed if the pacemaker cannot be reprogrammed or normal pacing does not resume spontaneously. Transcutaneous pacing can also be safely used because the anterior and posterior pacing electrodes, if properly positioned, are distant from the pulse generator. Attempting temporary transvenous pacing is usually not necessary and is unlikely to be successful, especially if undertaken without fluoroscopic guidance. Chronic venous thrombosis, which is common and most often asymptomatic after pacemaker insertion, may preclude temporary catheter insertion through the neck veins. Insertion through the femoral vein is also difficult because the permanently implanted catheter may prevent entry into the right ventricle. Blind insertion may also dislodge the permanent catheter.

IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS

The ICD was first used clinically in 1980. Technical refinements in this modality for treating ventricular dysrhythmias have progressed even more rapidly than refinements in the less complex standard pacemaker. A surge in the use of ICDs reflects improved survival with ICDs versus antiarrhythmic therapy in patients at risk for sudden death resulting from ventricular dysrhythmias. Generally accepted indications for ICD implantation are noted in Box 80-2. Many patients still require drug therapy after ICD implantation to suppress ventricular dysrhythmias, minimize the frequency of ICD shocks, improve patients’ tolerance, and decrease energy use, which prolongs ICD life.

Terminology and Components

The majority of ICDs are now placed percutaneously in a manner similar to that of the standard pacemaker. A transvenous electrode system has largely replaced epicardial lead placement, which required thoracotomy. An epicardial defibrillation lead may occasionally be placed during coronary artery bypass surgery or in a few patients who cannot be defibrillated with use of existing transvenous electrode systems.

The typical modern ICD consists of components similar to those in the standard permanent pacemaker—namely, a power source, electronic circuitry, and lead system. In addition, the standard ICD has a high-voltage capacitor and complex microprocessor memory. The power source is lithium chemistry based with a battery life of 5 to 10 years. The longevity is largely determined by the daily energy use, which prolongs ICD life.
by the frequency of shocks. All ICDs are also ventricular pacemakers, providing pacing for bradycardias.

The right ventricular lead is used for sensing and pacing, and shocks are typically delivered between a coil in the right ventricular lead and the pulse generator. If dual-chamber pacing is required, a second lead is placed in contact with the endocardium of the right atrium. A biphasic waveform is currently the preferred waveform for internal defibrillation. The shape and characteristics of the shock waveform vary among manufacturers. The biphasic waveform is more effective at lower energies than earlier monophasic waveforms and allows a smaller capacitor to be used, thereby reducing the size and increasing the comfort of the ICD unit.

The diagnostic and treatment functions of the ICD are determined at the time of implantation. In most instances, the cardioversion and defibrillation thresholds are determined at the time of ICD insertion by inducing ventricular tachycardia (VT) and ventricular fibrillation (VF) and adjusting the shock strength at a level above the minimum required to terminate the induced rhythm. Optimally, the required shock strength for defibrillation is less than half the maximum output (approximately 30 J) of the device. VT is typically managed with use of either low-energy shocks or programmed pacing that interrupts the VT reentrant circuit. Programmed pacing is less likely to have proarrhythmic effects and requires less energy, thereby extending battery life. In the setting of VF, ICDs are capable of delivering up to five additional discharges if the first shock fails.

The patient with an ICD should have close follow-up monitoring by a cardiologist familiar with ICD programming. This allows the cardiologist to determine the frequency of ICD activation (programmed antitachycardia pacing or shocks) and to confirm the programmed functions of the device. The majority of patients with ICDs have underlying heart disease, most commonly extensive atherosclerotic coronary artery disease, which is complicated by a low ejection fraction and congestive heart failure. The patient’s medications and metabolic status, such as electrolyte disorders that accompany diuretic usage, may also affect ICD function.

Complications of Implantation

Complications of ICD implantation are nearly identical in type and frequency to those of permanent pacemaker implantation. They include infection of the wound, the subcutaneous pouch fashioned for the device, and the lead system as well as acute thrombophlebitis and chronic thrombosis of the veins traversed for lead insertion. Management of these complications is similar to that for patients with permanent pacemakers.

Malfunction

Patients with ICD malfunction usually come to the ED with a limited number of specific symptoms (Box 80-4).

In contrast to patients with a permanent pacemaker, ICD patients are aware of when the ICD discharges to terminate VT or VF. The most common complaint of ICD patients is the occurrence of frequent shocks (i.e., occurring at a rate greater than they are accustomed to). An increasing shock rate may be appropriate and not indicative of ICD malfunction if the patient is experiencing an increase in the frequency of VT or VF episodes. An increase in the frequency of episodes may occur in the setting of hypokalemia, hypomagnesemia, ischemia (with or without infarction) related to underlying coronary artery disease, or the proarrhythmic effect of drugs administered to decrease the frequency of ventricular tachyarrhythmias. Many ICD patients, particularly those to whom the technology is new, report that their device discharged, but subsequent device interrogation reveals that no discharge occurred.

**Box 80-4 Causes of Implantable Cardioverter-Defibrillator Malfunction**

- Increased frequency of VF or VT (consider ischemia, electrolyte disorder, or drug effect)
- Displacement or break in ventricular lead
- Recurrent nonsustained VT
- Sensing and shock of supraventricular tachyarrhythmias
- Oversensing of T waves
- Sensing noncardiac signals

**Syncope, near-syncpe, dizziness**
- Recurrent VT with low shock strength (lead problem, change in defibrillation threshold)
- Hemodynamically significant supraventricular tachyarrhythmias
- Inadequate backup pacing for bradyarrhythmias (spontaneous or drug induced)

**Cardiac arrest**
- Assume malfunction, but probably caused by VF that failed to respond to programmed shock parameters

An increase in the shock frequency is a manifestation of ICD sensing malfunction if (1) a supraventricular tachyarrhythmia is inappropriately sensed as VT, (2) shocks are delivered for nonsustained VT, or (3) intracardiac T waves detected by the ICD system are sensed as QRS complexes and the ICD interprets this as an increased heart rate. Temporary ICD deactivation with magnet application may be necessary if oversensing is the problem. Syncope, near-syncpe, dizziness, or lightheadedness in the patient with an ICD may indicate undersensing of sustained VT or inappropriately low shock strength to terminate the rhythm. An approach to the evaluation of ICD malfunction is shown in Figure 80-8.

**Advanced Cardiac Life Support Interventions**

An ICD does not prevent sudden death in all patients at risk, and a patient with an ICD may arrive in cardiac arrest (2% annual incidence in patients with implanted devices). Cardiac arrest is not necessarily an indication of ICD malfunction. Appropriate repeated shocks may have been delivered but were ineffective. Alternatively, the ICD may not have sensed VF or the ventricular ectopic activity that typically precedes VF. Resuscitation efforts in the patient with an ICD should be undertaken in accordance with current recommendations. Transthoracic defibrillation can be performed in the standard manner with a monophasic or biphasic defibrillator if VF is the arrest rhythm. The sternal electrode or paddle should be placed in a parasternal location approximately 10 cm from the ICD subcutaneous pouch if the device has been implanted in the right deltopectoral area. If it has been implanted in the left deltopectoral region, this recommended safety distance is usually exceeded.

ICD discharge during manual chest compressions poses no risk to providers, although the rescuer may feel a weak shock. Although this generally is not indicated, the device can be deactivated with magnet application during resuscitation efforts. Deactivation is probably more important in the immediate postresuscitation period because recurrent ventricular dysrhythmias are common at this time after prolonged global myocardial ischemia during the arrest period, reperfusion, and the hyperadrenergic state, which is worsened by the use of intravenous epinephrine during resuscitation. ICD malfunction should be assumed, and these postresuscitation rhythms treated with standard pharmacologic agents (lidocaine and amiodarone). Although class I
Figure 80-8. Approach to the patient with shocks. Top, Flow diagram for one or infrequent shocks. Bottom, Diagram for multiple or repetitive shocks. ICD, implantable cardioverter-defibrillator; SVT, supraventricular tachycardia; VT/VF, ventricular tachycardia/ventricular fibrillation. (Redrawn from Swerdlow CD, Zhang J: Implantable cardioverter defibrillator shocks: A troubleshooting guide. Rev Cardiovasc Med 2:61, 2001.)

Antidysrhythmic agents may raise the defibrillation threshold of the ICD, their impact on the defibrillation threshold during trans-thoracic countershock is clinically inconsequential owing to the high shock strengths that are used.

**Disposition of the Emergency Department Patient with an Implantable Cardioverter-Defibrillator**

As a result of the difficulty in documenting or excluding ICD function or malfunction in the patient with transient symptoms, the device should be interrogated to guide further evaluation and therapy. In cases in which the patient reports a single ICD shock, an assessment for acute cardiac ischemia, worsening of chronic congestive heart failure, symptoms of new-onset heart failure, and electrolyte abnormalities should be performed. In the absence of a change in clinical status, such patients can be discharged in consultation with the managing or consulting cardiologist after timely follow-up is ensured. For patients reporting multiple shocks, interrogation is essential, because in many of these cases the defibrillator has not discharged and the patient is experiencing hiccoughs, diaphragmatic twitching, or other nonelectrical phenomena. In such cases, discharge home is the rule. When multiple defibrillator discharges are confirmed by interrogation, immediate consultation is required along with admission to a monitored setting for extended telemetric observation. If frequent ventricular ectopy is noted, intravenous amiodarone should be given. If a lead problem is detected, reimplantation is required. A magnet can be placed over the ICD to inactivate the defibrillator. This should be done only if the emergency physician is confident that the ICD is delivering inappropriate shocks, such as a supraventricular tachycardia.

**Biventricular Pacing**

Biventricular pacing, also known as cardiac resynchronization therapy, is a therapy for patients with left-sided heart failure and ventricular dyssynchrony. It is beneficial for patients with New York Heart Association (NYHA) class III or IV heart failure despite optimal medical therapy, a left ventricular ejection fraction of 35% or less, and sinus rhythm with QRS duration of 120 msec or greater. Left bundle branch block causes an altered sequence of depolarization of the left ventricle such that the interventricular septum contracts before the left ventricular free wall, leading to inefficient mechanical pumping. Biventricular pacing “resynchronizes” the ventricles by simultaneously pacing the left and right ventricles, eliminating the delay in left ventricular free wall contraction and improving systolic function. Right atrial and right ventricular leads are positioned as for conventional atrial and univentricular pacing. The left ventricular lead is positioned in a left ventricular epicardial location via the coronary sinus and veins, preferably in a posterolateral or lateral location. The QRS duration of paced ventricular complexes is often but not always less than the QRS duration measured before resynchronization therapy.

Biventricular pacing can usually be recognized on the standard ECG (Fig. 80-9). Two stimulus artifacts or “spikes” may be seen preceding a paced QRS complex. With biventricular pacing, a predominantly negative QRS complex is seen in lead I, in contrast to the typical upright complex seen with right ventricular pacing (see Fig. 80-2). A predominantly positive QRS complex is seen in lead V1 with biventricular pacing.

The complications and malfunctions inherent with conventional cardiac pacing are also observed with biventricular pacing. In addition, biventricular pacing has unique complications related to placement of the left ventricular pacing lead through the coronary sinus. In large clinical trials, coronary sinus dissection occurred in 0.3 to 4.0% of patients and coronary vein or coronary sinus perforation in 0.8 to 2.0% of patients. Cardiac tamponade caused by perforation of the coronary venous system is seen in less than 1% of patients. Dislodgement of the left ventricular electrode with resultant loss of pacing occurs as an early complication in approximately 10% of patients. Patients with malfunction of a biventricular pacing system frequently report palpitations or acute decompensation of chronic heart failure.

**Left Ventricular Assist Devices**

Mechanical ventricular assistance devices have been used as a “bridge” to transplantation since the 1960s. These implanted devices replace or support the pump function of the left ventricle and were originally bulky and mechanically complex owing to the pulsatile nature of the pump mechanisms. This usually resulted in extended in-hospital care while the patient awaited cardiac transplantation. Newer devices, such as the Jarvik 2000 and HeartMate II, are continuous flow pumps that are portable and powered with a comparatively smaller battery pack. Implantation is associated with fewer infectious complications and lower immediate postoperative mortality, and survival for more than 1 year is increasingly common. The greatest mortality is noted within the first 30 days after implantation and during hospitalization. Left ventricular assist devices (LVADs) are commonly used as “destination” therapy in patients who do not qualify for cardiac transplantation.
Biventricular

Up to 3

blood pressure.

(PVC).

synchronization

Figure 80-9. Biventricular pacemaker. This 12-lead ECG demonstrates an atrial sensed, biventricular pacemaker in a patient with cardiac synchronization therapy (CRT) and implantable cardioverter-defibrillator (ICD) system (CRT-D). The PR interval is 350 msec and represents the programmed AV delay for this patient. The paced QRS complexes have an S wave in lead I and an R wave in lead V1, that are distinctly different from the morphology and axes seen with right ventricular apical pacing. The second beat from the left is a premature ventricular contraction (PVC). There is a pacemaker “spike” superimposed on this complex, likely representing safety pacing in CRT-D.

Table 80-3

Comparison of Common Ventricular Assist Devices

<table>
<thead>
<tr>
<th></th>
<th>THORATEC VAD</th>
<th>HEARTMATE II</th>
<th>HEARTMATE I OR XVE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Flow type</strong></td>
<td>Pulsatile—patient will have a pulse and BP.</td>
<td>Axial—patient will not have a pulse or BP.</td>
<td>Pulsatile—patient will have a pulse and BP.</td>
</tr>
<tr>
<td><strong>Backup method</strong></td>
<td>Hand pump</td>
<td>No external method</td>
<td>Hand pump</td>
</tr>
<tr>
<td><strong>Battery life</strong></td>
<td>Up to 3 hr</td>
<td>Up to 10 hr</td>
<td>Up to 10 hr</td>
</tr>
<tr>
<td><strong>Defibrillation or cardioversion</strong></td>
<td>No precautions</td>
<td>No precautions</td>
<td>Use hand pump during procedure</td>
</tr>
<tr>
<td><strong>Cardiac arrest</strong></td>
<td>Use hand pump</td>
<td>No external method</td>
<td>Use hand pump</td>
</tr>
</tbody>
</table>

BP, blood pressure.

Patients with an LVAD typically require care at cardiac transplant centers. As technology progresses, emergency physicians not based at a transplant center may encounter a patient with an LVAD. In such cases, telephone consultation with an expert at a transplant center may help avoid missteps. The more common complications with this device include infection and embolic stroke. Device failure usually results in severe heart failure. If necessary, dopamine, dobutamine, or a combination of these drugs may be given to treat congestive heart failure symptoms while the patient awaits transfer. If cardiac arrest occurs, chest compressions may dislodge the device. The hand pump should be used to provide circulation if this backup method is available and early transition to cardiopulmonary bypass should be considered (Table 80-3).

**KEY CONCEPTS**

- Pacemaker malfunction soon after implantation (within 6-8 weeks) is usually a result of a lead problem, such as a lead displacement, or a pacemaker programming failure, such as a pacing rate too slow for the patient’s needs.
- Pacemaker malfunction arises in a limited number of ways: failure to pace, oversensing, undersensing, and pacing at an inappropriate rate (too fast or too slow).
- With lithium-iodine batteries, abrupt failure is an unlikely cause of pacemaker malfunction.
- If a patient with a pacemaker has a fever of unclear cause, pacemaker lead infection and endocarditis should be considered.
- Because paced ventricular complexes are conducted with a left bundle branch block pattern, a paced rhythm obscures the electrocardiographic diagnosis of acute myocardial infarction. A right bundle branch pattern is abnormal and suggests lead displacement.
- Magnet application does not turn off a pacemaker, it turns off the sensing or inhibition function. Fixed-rate pacing that is independent of or in competition with the underlying native rhythm will ensue. Removal of the magnet restores the inhibitory activity of the pacemaker and returns it to demand pacing mode.
- Defibrillation is safe in patients with a pacemaker or ICD. Paddles should be placed at least 10 cm from the subcutaneous implant site of the device. Alternatively, anteroposterior defibrillation with adhesive defibrillation electrodes can be performed. There are no reports of injury to rescuers from ICD discharges during manual chest compressions.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.
References


