Managing Pacemaker-Related Complications And Malfunctions In The Emergency Department

Abstract

The use of implanted pacemaker devices is increasing worldwide, owing to technological advances, new indications, and an aging population. Despite greater experience in implantation and improved device sophistication, patients continue to face complications associated with hardware implantation and device malfunction. This review summarizes current indications for permanent pacing, reviews epidemiologic data relevant to implant complications, and describes a clinical approach to the patient with potential pacing malfunction. The electrocardiographic diagnosis of hyperkalemia and acute myocardial infarction in paced rhythms is also discussed. Potential sources of electromagnetic interference and special considerations pertaining to the cardiac resuscitation of patients with implanted cardiac devices are reviewed. Finally, a basic approach to implanted cardioverter-defibrillator devices (which often accompany pacemaker devices) is presented.
Cardiac pacemakers have evolved from single-chamber devices that deliver a fixed pacing rate to multichamber systems with programmable features that preserve and more closely mimic normal cardiac electrophysiology. Preservation of atrioventricular synchrony, physiologic heart rate, and interventricular synchrony are significant improvements that have been made possible with dual-chamber, rate-adaptive, and biventricular pacing systems. For patients, improved quality of life, increased exercise tolerance, and decreased disease progression are among the clinical outcomes driving new indications for pacemaker implantation and cardiac resynchronization therapy (CRT). At the same time, mortality benefits associated with implanted cardioverter-defibrillator devices (ICDs) in patients with heart failure have translated into an increasing number of biventricular pacing systems with integrated ICD technology. While pacemakers have become more complex and sophisticated, patients continue to present to the emergency department (ED) with symptoms related to device malfunctions. In addition, special considerations with regard to implanted pacemakers are relevant to the diagnostic workup of comorbid illnesses.

Epidemiology

Implanted pacemakers are increasingly prevalent in the United States. Approximately 370,000 pacemakers are placed annually, the most common indication for which is sinus node disease. Regardless of the indication for pacing, dual-chamber pacemakers have been adopted as the technology of choice. However, since United States Food and Drug Administration (FDA) approval in 2001, CRT devices are being implanted more frequently. Most of these are combined with defibrillation technology, and CRT-defibrillator (CRT-D) devices have come to comprise approximately 40% of all pacemakers in the United States. Compared with the general population, patients receiving implanted devices are older (mean age of implantation of 75.6 years) and carry a higher burden of age-adjusted comorbid illness.

Critical Appraisal Of The Literature

PubMed and the Cochrane Database of Systematic Reviews were searched for English-language articles pertaining to the management of patients with implanted pacemakers and cardiac devices published between January 1, 1990 and February 21, 2014. Search terms included permanent pacemakers and the thematic topics of pacemaker complications, cardiac resynchronization therapy, pacemaker malfunction, and electromagnetic pacemaker interfer-

### Table 1. American College of Cardiology/American Heart Association Classification Of Recommendations And Level Of Evidence

<table>
<thead>
<tr>
<th>Certainty of Treatment Effect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Recommendations derived from multiple randomized clinical trials or meta-analyses</td>
</tr>
<tr>
<td>B</td>
<td>Data derived from a single randomized clinical trial or nonrandomized studies</td>
</tr>
<tr>
<td>C</td>
<td>Only consensus opinion of experts, cases studies, or standard of care</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Size of Treatment Effect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>Conditions for which there is evidence and/or general agreement that the test is useful and effective</td>
</tr>
<tr>
<td>Class II</td>
<td>Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness or efficacy of performing the test</td>
</tr>
<tr>
<td>Class IIa</td>
<td>Weight of evidence/opinion is in favor of usefulness or efficacy</td>
</tr>
<tr>
<td>Class IIb</td>
<td>Usefulness or efficacy is less well established by evidence/opinion</td>
</tr>
<tr>
<td>Class III</td>
<td>Conditions for which there is evidence and/or general agreement that the test is not useful or effective and in some cases may be harmful</td>
</tr>
</tbody>
</table>
**Review Of Normal Pacemaker Function**

**Components And Lead Placement**

The basic components of a pacemaker are the pulse generator and the lead. The pulse generator consists of hardware, programmable software, and a battery with a 5- to 10-year lifespan. The longevity of any battery depends on the proportion of paced and sensed events, stimulation impedance, and programmed output. Lead components include a conductor, electrode, fixation mechanism, terminal connector pin, and insulation. The connector pin connects the lead to the generator and must be well seated in the connector block of the generator for the device to function.

Atrial leads are most commonly positioned in the right atrial appendage. Selective site pacing in the right atrial septum may be chosen to suppress atrial fibrillation. Right ventricular leads are traditionally placed in the right ventricular apex; however, pacing from this site has been shown to result in ventricular dyssynchrony and subsequent impairment in left ventricular function.\(^{12-15}\) Right ventricular leads may be placed in the right ventricular outflow tract (RVOT) or interventricular septum to minimize these effects, but evidence to support improved outcomes from selective site pacing in the RVOT is contested. The left ventricular lead of a biventricular pacing system is placed through the coronary sinus into one of its tributaries, in an epicardial location along the posterior or lateral free wall of the left ventricle. The final position of the left ventricular lead depends on cardiac venous anatomy, avoidance of phrenic nerve stimulation, and location of myocardial scar.

**Pacemaker Modes**

A 5-letter code has been adopted to identify different types of pacemakers. (See Table 2.) The first letter corresponds to the lead location. The second letter corresponds to the chamber (or chambers) being sensed by the pacemaker. The third letter indicates the response of the pacemaker to a sensed impulse. The fourth letter indicates the pacemaker’s programmability and capacity for rate modulation. A pacemaker capable of rate modulation (R) is also capable of communicating with external telemetry (C) and multiprogrammability (M), in which 3 or more variables can be modified. Pacemakers capable of rate modulation mimic the rate response of a normal sinus mode when physiological changes are detected by sensors for minute ventilation, QT interval, or stroke volume. In 2002, the code was revised to include a fifth letter indicating whether multisite pacing is present in the atrium, the ventricle, or both.\(^{16}\) Emergency clinicians are most likely to encounter pacers that are AAIR, VVIR, DDD, and DDDR.\(^{17}\)

**Pacemaker Indications**

The indications for permanent pacing are continually amended and are categorized by type of underlying conduction disorder or disease process. The ACC/AHA/HRS guidelines for the use of cardiac pacemakers, ICDs, and CRT were most recently published in 2008,\(^{8}\) with a focused revision on the indications for CRT published in 2013.\(^{7}\) In general, class I recommendations apply in cases of symptomatic impaired conduction. With regard to asymptomatic conduction abnormalities, class I recommendations apply in disorders or diseases that are likely to progress. For more information about the classes and levels of evidence for the indications for permanent pacing, see Figure 1, page 4 for the URL and QR code to access the PDF of the full guidelines.

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**Table 2. The North American Society For Pacing And Electrophysiology/British Pacing And Electrophysiology Group Generic Pacemaker Code**

<table>
<thead>
<tr>
<th>Chamber(s) paced</th>
<th>Chamber(s) sensed</th>
<th>Response to sensing</th>
<th>Programmability, rate modulation</th>
<th>Multisite pacing</th>
</tr>
</thead>
<tbody>
<tr>
<td>A = Atrium</td>
<td>A = Atrium</td>
<td>T = Triggered</td>
<td>P = Simple programmable</td>
<td>A = Atrium</td>
</tr>
<tr>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
<td>I = Inhibited</td>
<td>M = Multiprogrammable</td>
<td>V = Ventricle</td>
</tr>
<tr>
<td>D = Dual (A + V)</td>
<td>D = Dual (A + V)</td>
<td>D = Dual (triggered + inhibited)</td>
<td>C = Communicating</td>
<td>D = Dual (A + V)</td>
</tr>
<tr>
<td>O = None</td>
<td>O = None</td>
<td>O = None</td>
<td>R = Rate modulation</td>
<td>O = None</td>
</tr>
</tbody>
</table>

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Sinus Node Dysfunction
Pacemakers are placed in patients with symptomatic sinus node dysfunction in order to reduce the frequency of syncopal episodes and the risk of stroke and atrial fibrillation. Sinus node dysfunction includes sinus bradycardia, sinus arrest, sinoatrial block, chronotropic incompetence, atrial tachycardia, and atrial fibrillation. The preferred pacemaker mode in patients with sinus node dysfunction is DDDR. Dual-chamber pacing in patients with sinus node dysfunction has been associated with reduced pacemaker syndrome symptoms, reduced atrial fibrillation, and improved exercise capacity compared with single-chamber pacing.\(^{18,19}\) Pacemaker syndrome manifests as intolerance to ventricular pacing in the absence of atrioventricular synchrony and will be discussed in more detail on page 9. In patients with preserved atrioventricular conduction, AAIR pacing may be a cost-effective choice to minimize unnecessary ventricular pacing. Subsequent atrioventricular block is a concern factored into the choice of device and develops at an annual rate of 1.4\% after atrial pacemaker implantation.\(^{20}\)

Acquired Atrioventricular Block
Pacing is recommended for patients with third-degree block and type II second-degree atrioventricular block to reduce syncope and improve survival. The indication for pacemaker placement in type I second-degree atrioventricular block is more controversial. Guidelines are based on whether the patient is symptomatic and on the exact location of the block as it relates to the likelihood that the disease will progress to complete heart block. (See Figure 1.) Dual-chamber pacing is recommended over ventricular pacing to reduce the incidence of pacemaker syndrome.

Chronic Bifascicular Or Trifascicular Block
Syncope is common in patients with bifascicular and trifascicular block, but the risk of progression to complete heart block and death varies based on the presence of atrioventricular block. In the absence of atrioventricular block, the indication for pacemaker placement depends on the presence of prolonged His-ventricular (H-V) intervals during electrophysiologic study.

After Acute Myocardial Infarction
New-onset atrioventricular block in the acute setting of ST-segment elevation myocardial infarction (STEMI) usually resolves spontaneously within days.\(^{21}\) Temporary pacing is recommended in the setting of acute STEMI for patients with symptomatic bradycardia that is unresponsive to medical treatment (class I, level of evidence [LOE] C).\(^{22}\) In the acute phase of inferior STEMI, atrioventricular block is more often mediated by increased vagal tone; in anterior STEMI, the development of atrioventricular or intraventricular block is related to the extent of the infarcted area. Patients with atrioventricular block and impaired intraventricular conduction after myocardial infarction (MI) have a high likelihood of mortality that most likely relates to extensive myocardial necrosis rather than impaired conduction. Patients with anterior infarction and impaired systolic dysfunction should be considered for CRT.

Cardiac Resynchronization Therapy
Systolic heart failure is accompanied by prolonged intraventricular conduction in approximately 30\% of patients.\(^{23}\) Delayed conduction results in further reduction of systolic function, ventricular remodeling, and functional mitral regurgitation. Ventricular dysynchrony, defined by a prolonged QRS duration, is associated with increased mortality in patients with heart failure.\(^{24}\) CRT improves ventricular dyssynchrony by pacing from leads in both right and left ventricles. In patients with heart failure, improved electromechanical synchrony has been shown to induce reverse ventricular remodeling, decrease mitral regurgitation, decrease neurohormonal activation, and improve peak oxygen consumption.

Results from several landmark studies have led to the established role of resynchronization therapy in treating heart failure. Initial randomized studies were designed to focus on functional outcomes of CRT. They demonstrated improved quality of life, New York Heart Association (NYHA) functional classifications for heart failure, exercise tolerance, and left ventricular ejection fraction.\(^{5,25,26}\) Later studies demonstrated improved morbidity (unplanned hospitalizations) and mortality outcomes in CRT patients with NYHA Class II, III, or IV heart failure. However, improved outcomes are not universal; approximately 10\% to 40\% of patients are nonresponders, depending on how response is defined functionally.\(^{32}\) Research aimed at defining the subgroups most likely to benefit from CRT has been incorporated into the most recent indications for CRT.\(^{7}\) (See Table 3, page 5.) According to a meta-analysis, the morbidity and mortality benefits of CRT are limited to patients with QRS duration \(\geq 150\) msec.\(^{33}\) Moreover, patients with left bundle branch block (LBBB) morphology have also been

Figure 1. Indications For Permanent Pacing
Scan the QR code above with your smartphone or tablet, or go to: https://my.americanheart.org/idc/groups/ahaecc-internal/@wcm/@sop/documents/downloadable/ucm_423809.pdf

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shown to have greater benefit than those with non-LBBB conduction abnormalities, including right bundle branch block (RBBB) and nonspecific intraventricular conduction delay.\(^{34}\)

**Neurocardiogenic Syncope And Carotid Sinus Syndrome**

The role of pacemaker therapy in neurocardiogenic syncope is controversial, as the evidence supporting its use is mixed. Open-label trials have demonstrated a decrease in syncopal events,\(^{35-37}\) while 2 double-blinded placebo-controlled trials (total of 129 patients) have failed to demonstrate any benefit.\(^{38,39}\)

The alleged benefit of pacemaker therapy in carotid sinus syncope is based solely upon observational studies\(^{40-42}\) and open-label randomized trials.\(^{43,44}\) In a double-blind, placebo-controlled crossover trial that included 34 patients, there was no statistically significant difference in unexplained falls between those with the pacemaker turned on and those with it turned off.\(^{45}\) However, this study was considered underpowered because of the study’s high attrition rate.

**Emergency Department Evaluation**

**History**

Even with the availability of advanced device-monitoring capabilities, obtaining the history from a patient with an implanted device remains important. This includes cardiac history as well as noncardiac medical history and, perhaps most importantly, the original indication for pacemaker placement. The type of pacemaker should be identified so that appropriate interrogation can be performed. Patients should be asked for dates of implantation and revision; if and when programming changes were made; and about a history of exposure to any electromagnetic interference, blunt trauma, or environmental extremes. A complete list of medications should be obtained, as some drugs can alter pacing thresholds.

Pacemaker-related symptoms should be reviewed with the patient. These include light-headedness, palpitations, syncope, sense of slowed or racing heart, and extracardiac muscle twitching.

**Physical Examination**

In addition to a standard thorough cardiac examination, the implantation site itself should be examined for erythema, bruising, swelling, and tenderness. Signs of swelling in the neck or ipsilateral arm should raise suspicion for venous thrombosis. Moving the patient’s ipsilateral arm or having the patient perform isometric exercise may help to identify intermittent dysfunction.

**Diagnostic Studies**

**Electrocardiogram**

The 12-lead ECG is important for determining whether depolarization is intrinsic or paced. Bipolar leads produce smaller pacing artifacts that may be imperceptible in some leads and easily recognized in others. The absence of a pacer artifact before P waves or QRS waves indicates that depolarization is intrinsic. Pacing artifacts preceding depolarizations indicate successful pacing and capture.

### Table 3. Indications For Cardiac Resynchronization Therapy\(^{7}\)

<table>
<thead>
<tr>
<th>Class</th>
<th>Indication</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>LVEF ≤ 35%; sinus rhythm; LBBB with a QRS duration ≥ 150 msec; and NYHA class II, III, or ambulatory class IV symptoms on goal-GDMT</td>
<td>B</td>
</tr>
<tr>
<td>IIa</td>
<td>LVEF ≤ 35%; sinus rhythm; LBBB with a QRS duration 120-149 msec; and NYHA class II, III, or ambulatory class IV symptoms on GDMT</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>LVEF ≤ 35%; sinus rhythm; a non-LBBB pattern with a QRS duration ≥ 150 msec; and NYHA class III/ambulatory class IV symptoms on GDMT</td>
<td>A</td>
</tr>
<tr>
<td></td>
<td>Atrial fibrillation and LVEF ≤ 35% on GDMT if: (a) patient requires ventricular pacing or otherwise meets CRT criteria, and (b) AV nodal ablation or pharmacologic rate control will allow near-100% ventricular pacing with CRT</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>LVEF ≤ 35%, undergoing new placement or replacement of a device with anticipated requirement for significant (&gt; 40%) ventricular pacing</td>
<td>C</td>
</tr>
<tr>
<td>IIb</td>
<td>LVEF ≤ 30%, ischemic etiology of heart failure, sinus rhythm, LBBB with QRS duration ≥ 150 msec, and NYHA class I symptoms on GDMT</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td>LVEF ≤ 35%, sinus rhythm, a non-LBBB pattern with QRS duration 120-149 msec, and NYHA class III/ambulatory class IV on GDMT</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>LVEF ≤ 35%, sinus rhythm, a non-LBBB pattern with a QRS duration ≥ 150 msec, and NYHA class II symptoms on GDMT</td>
<td>B</td>
</tr>
<tr>
<td>III</td>
<td>NYHA class I or II symptoms and non-LBBB pattern with QRS duration &lt; 150 msec</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>Patients whose comorbidities and/or frailty limit survival and good functional capacity to less than 1 year</td>
<td>C</td>
</tr>
</tbody>
</table>

Abbreviations: AV, atrioventricular; CRT, cardiac resynchronization therapy; GDMT, goal-directed medical therapy; LBBB, left bundle branch block; LOE, level of evidence; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association.
The 12-lead ECG is also essential for evaluating the morphology of paced QRS complexes. Leads positioned in the right ventricular apex produce a LBBB pattern with appropriate discordance in the ST segments and T waves. A change in this morphology may indicate lead dislocation. Specifically, the presence of a new RBBB pattern may indicate inadvertent placement of the right ventricular lead into the left ventricle or septal perforation. A RBBB pattern may also appear in a minority of correctly positioned right ventricle leads. Simultaneous depolarization of the left and right ventricles from a biventricular pacing system typically produces a dominant R wave in lead V₁.⁴⁶

**Hypermalemia**

Delayed intraventricular conduction causes paced QRS complexes in the hyperkalemic patient to widen. Severe hyperkalemia may result in a sine wave following each pacing artifact. (See Figure 2.)

Severe hyperkalemia may cause ECG changes to be mistaken for pacemaker malfunction. The pacing threshold is elevated in hyperkalemia, leading to increased latency, intermittent capture (including Wenckebach phenomenon), or continuous loss of capture,⁴⁸,⁴⁹ as well as loss of sensing.⁵⁰ Latency or failure to capture may be temporarily overcome by programming pacemaker outputs to maximum voltage, but definitive treatment for hyperkalemia should be instituted without delay.

**Acute Myocardial Infarction**

The pattern of depolarization and repolarization in ventricular-paced rhythms may obfuscate the electrocardiographic diagnosis of acute myocardial infarction (MI). Sgarbossa found in a retrospective review of 17 ventricular-paced patients selected from the GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) trial population, that concordant ST elevation, concordant ST depression in leads V₁ through V₃, and > 5 mm of discordance in leads with predominantly negative QRS complexes were insensitive but specific findings for acute MI.⁵¹ (See Figure 3.) In a retrospective review of 57 ECGs of ventricular-paced patients diagnosed with acute MI, discordant ST elevation > 5 mm was the most specific finding (99% specificity; 95% confidence interval [CI], 93%-99%).⁵² However, discordant ST elevation can be > 5 mm in ventricular-paced rhythms in the absence of MI when negative QRS amplitudes in leads V₁ through V₃ are large.⁵³ As with application of the Sgarbossa rule to LBBB, an elevated ratio of ST-segment elevation to S-wave depth may be more specific for MI than the absolute measurement of 5 mm.⁵⁴

**Radiology**

A basic knowledge of the normal and abnormal radiographic appearances of cardiac conduction devices and their components can be important in diagnosing complications associated with implantation and device integrity. (See Table 4.) Both posterior-anterior and lateral views should be obtained.  

**Figure 2. Electrocardiogram Tracing Of Paced Rhythms In The Setting Of Severe Hyperkalemia**

<table>
<thead>
<tr>
<th>A</th>
<th>K= 7.8 mEq/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>K= 8.7 mEq/L</td>
</tr>
</tbody>
</table>

Arrows point out pacing artifacts.  

**Table 4. Radiographic Assessment Of The Permanent Pacemaker/Implantable Cardioverter-Defibrillator**

- Obtain posterior-anterior and lateral views.
- Locate pulse generator and compare with previous chest x-ray.
- Identify manufacturer, if needed.
- Ensure connector pin is seated deep within header.
- Identify number of leads and lead location.
- Confirm integrity of each lead.
be obtained to confirm correct lead position, and radiographs should be compared with any prior studies. In addition, the manufacturer and model on the pulse generator can often be identified from the chest radiograph.

Lead location and integrity should be confirmed systematically. On a lateral view, the right atrial lead has a J-shaped appearance as it enters the right atrium and curves upward and anteriorly into the right atrial appendage. The J portion of the lead is medial on the posterior-anterior projection and anterior on the lateral projection. Atrial leads may also be placed in the interatrial septum. Right ventricular leads should point downward, with the tip between the left of the vertebral column and the cardiac apex on a posterior-anterior radiograph. The lateral radiograph is required to confirm the anterior and caudal course of a correctly positioned right ventricular lead. Leads placed in the left ventricle or coronary sinus should course posteriorly on a lateral radiograph.

Magnification may be required to appreciate small defects in leads. A common site of lead fracture is between the first rib and clavicle (subclavian crush syndrome). Extraneous wires present on chest radiographs may represent abandoned leads. Malfunctioning leads are disconnected from the generator, capped, and left in place to avoid the risks associated with their extraction.

The key to differentiating a permanent pacemaker from an ICD is the presence of shock coils (see Figure 4), which appear as thick bands on the leads. ICD leads may contain a single high-voltage coil in the right ventricle or may have an additional coil in the superior vena cava.

Complications of implantation that can be diagnosed on plain radiographs include pneumothorax, hemopneumothorax, myocardial perforation, and improper seating of a connector pin. Winding of a lead around the pulse generator may be indicative of twiddler syndrome. (See page 8.)

Device Interrogation

Noninvasive testing allows acquisition of measured data, interrogation of programmed parameters, and analysis of stored arrhythmias. Basic parameters include pacing mode, and lower and upper tracking rates can be shown during device interrogation. Measurements of the battery’s voltage can help to determine whether battery failure might be the cause of pacemaker malfunction. With some devices, the estimated remaining life of the battery can be determined. Measurements of lead status can help to identify lead failures (such as a break in insulation or conductor fracture). A sudden drop in impedance, for example, might indicate an insulation break.

The pacemaker is also capable of telemetry. The relative time spent between paced and native activity in each chamber is recorded. Heart rate measurements are stored and a graphical representation of time spent in different heart rate intervals can be displayed as a histogram. The date, time, and duration of arrhythmia episodes and their associated electrograms are also stored by implanted devices. This capability makes implanted pacemakers valuable diagnostic tools for ruling out tachyarrhythmia as the underlying cause for a patient with recent symptoms. (See Table 5.)

Table 5. Methods To Identify Device Manufacturer

<table>
<thead>
<tr>
<th>Method</th>
<th>Information Provided</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ask patient for pocket card</td>
<td>This will specify the type of pacemaker, type of leads, manufacturer, date of implantation, paced rate, as well as contact information for the patient’s primary physician.</td>
</tr>
<tr>
<td>Place magnet over pulse generator</td>
<td>This will identify magnet-induced pacing rate (specific to each model).</td>
</tr>
</tbody>
</table>
| Call manufacturer hotlines                  | Medtronic, Inc.: 1-800-328-2518  
The Boston Scientific Corporation: 1-800-CARDIAC  
St. Jude Medical, Inc.: 1-800-722-3774 |
| Obtain chest x-ray                          | Look for manufacturer code on pulse generator.                                       |

Figure 4. Radiograph Of Dual-Chamber, Biventricular Pacemaker/Implantable Cardioverter-Defibrillator System

Abbreviations: LV, left ventricle; RA, right atrium; RV, right ventricle.
Lead Dislodgement

A relatively more common lead-related issue is lead dislodgment. Dislodgement occurs in 2.4% of implantations, and may result in inadequate sensing or failure to capture.62 This issue usually occurs very early post procedure but may occur as late as 3 months afterward.57,63 Dislodgement is often suspected when undersensing or failure to capture is detected on follow-up telemetry. Subtle cases may be detectable only upon device interrogation demonstrating changes in pacing thresholds, while more pronounced cases can be visualized by chest radiography.63 Rarely, temporary external or transvenous pacing may be needed until repositioning or replacement of the leads can be performed. Lead dislodgement can sometimes result in lead migration or malposition. Migration of a lead through an occult septal defect can result in pacing of the left ventricle rather than the right. Malposition near the phrenic nerve may stimulate the nerve or the diaphragm directly and cause chronic singultus (hiccoughs).63,64 Lastly, lead dislodgement can cause myocardial perforation in 0.3% of cases in the days to weeks following implantation, resulting in pericardial effusion and, potentially, tamponade.62,64

Twiddler Syndrome

Lead dislodgement can also be caused by a patient’s own compulsive manipulation of the pacemaker generator, causing the leads to become retracted and coiled around the manipulated pulse generator. This is referred to as twiddler syndrome, and the diagnosis is made by the radiographic appearance of pacing leads coiled about the generator. Woven Dacron® pouches have been used to prevent migration and flipping of the pulse generator, but subpectoral implantation may be required to prevent recurrent device rotation.65,66

Table 6. Implant-Related Complications

<table>
<thead>
<tr>
<th>Complication</th>
<th>Diagnosis Method</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pocket hematoma</td>
<td>Examination</td>
<td>Conservative (rarely, surgical evacuation)</td>
</tr>
<tr>
<td>Pocket infection</td>
<td>Examination</td>
<td>Explantation, antibiotics</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>Echocardiography, blood cultures, TEE</td>
<td>Explantation, antibiotics</td>
</tr>
<tr>
<td>Lead dislodgement</td>
<td>Chest radiography, telemetry/ECG</td>
<td>Repositioning or replacement</td>
</tr>
<tr>
<td>Venous thrombosis</td>
<td>Sonography/venography</td>
<td>Anticoagulation, angioplasty, or surgery</td>
</tr>
<tr>
<td>Pneumothorax, hemothorax</td>
<td>Chest radiography</td>
<td>Conservative or aspiration/thoracostomy</td>
</tr>
<tr>
<td>Tricuspid regurgitation</td>
<td>Echocardiography</td>
<td>Lead extraction and/or annuloplasty/valve replacement</td>
</tr>
<tr>
<td>Cardiac perforation</td>
<td>Computed tomography, echocardiography</td>
<td>Pericardiocentesis, lead withdrawal and repo-sitioning</td>
</tr>
</tbody>
</table>

Abbreviations: ECG, electrocardiogram; TEE, transesophageal echocardiogram.
Venous Thrombosis
Subclavian or brachiocephalic venous thrombosis is a relatively more common occurrence after pacemaker implantation, with reported incidental rates of 2% to 22% from several days up to 9 years postoperatively. The range of reported rates (both in incidence and time) is a product of how the disease is typically discovered. Venous thrombosis is typically an incidental finding during a preoperative venogram in an asymptomatic patient undergoing lead revision. Thrombosis may be more common in patients who have systemic infection. Signs are often absent, but there may be typical manifestations of acute deep venous thrombosis and even superior vena cava syndrome, with the latter occurring in 0.2% of procedures. Ultrasonography may not be diagnostic, even in symptomatic cases, and a venogram will be required. If thrombosis is found, vascular surgery consultation should be sought. Although it is a relatively common finding, venous thromboses associated with cardiac devices are of questionable clinical importance, as pulmonary embolism is a rare complication in these patients. Treatments range from heparin/warfarin to percutaneous angioplasty or even open surgery.

Pneumothorax
As in other procedures that involve subclavian venous puncture, device implantation may result in pneumothorax from inadvertent pleural injury. In one study, pneumothorax was the second most common complication overall and occurred in 1.5% of patients. Hemothorax may occur due to unintentional arterial puncture or laceration, which can be minimized by proper use of fluoroscopic guidance. Postprocedural pneumothorax and hemothorax are often asymptomatic, but some cases may require needle aspiration or tube thoracostomy. Not surprisingly, these complications are less likely with axillary vein cannulation techniques.

Tricuspid Regurgitation
Tricuspid regurgitation may occur after pacemaker lead placement, although the mechanism of this occurrence is not fully understood. Commonly postulated mechanisms include direct damage to valve leaflets or impairment of valve closure due to scar formation or thrombus. Ventricular asynchrony is also a possible mechanism. Many patients are asymptomatic, but some may present with signs of right-sided congestive heart failure and a systolic murmur that increases with inspiration. Diagnosis is made by 3-dimensional transthoracic echocardiography. Leads that are adherent to the valve may require extraction. Unfortunately, the lead extraction procedure itself can lead to progression of tricuspid regurgitation.

Pacemaker Syndrome
Pacemaker syndrome is defined as intolerance to ventricular pacing in the absence of atrioventricular synchrony. The Mode Selection Trial (MOST) investigators suggest the following criteria: (1) new or worsened dyspnea, orthopnea, rales, elevated jugular venous pressure, and edema with ventriculoatrial conduction during ventricular pacing; or (2) dizziness, weakness, presyncope or syncope, and a > 20 mm Hg reduction of systolic blood pressure when the patient has VVIR pacing compared with atrial pacing or normal sinus rhythm. It is hypothesized that ventricular pacing leads to ventriculoatrial contraction with resultant increases in atrial pressures. Baroreceptor overload then alters vagal tone and there is resultant reduced cardiac output. This reduced output is further complicated by the loss of atrial contribution to preload. Pacemaker syndrome is usually treated by the restoration of some level of atrioventricular synchrony. This may involve replacing a single-chamber device with a dual-chamber device or reprogramming a dual-chamber device from VVI to DDD function.

Pacing System Malfunctions
Pacemakers and ICDs are complex medical devices and, while they are generally regarded as safe and reliable, they are subject to electrical and hardware malfunction. Generator malfunctions occur at a mean annual rate of 4.6 per 1000 implanted pacemakers and 20.7 per 1000 implanted ICDs. A meta-analysis of prospective device registries demonstrated that rates of generator failure have improved over the past 2 decades, and the most common cause of device failure was battery malfunction.

The majority of lead failures are caused by insulation defects. Typically, insulation breaks are a late complication, occurring at a median time of 7.5 years (+/- 2.5 y) after implantation. Lead problems can be detected by measuring lead impedance. Very high impedance results from an open circuit (conductor fracture), while low impedance implies an insulation defect.

Clinical symptoms of device malfunction include syncope, palpitations, dizziness, fatigue, and muscle twitching from extracardiac stimulation. Overall, however, pacemaker malfunction is a rare cause of syncope. In one retrospective study, device malfunction was found to be the cause of syncope in only 8 of 162 patients.

An assessment of pacing system malfunction should include evaluation of the pulse generator, the leads, programmed settings, and algorithms, as well as any patient status (eg, electrolyte disturbance) that may affect the electrode-myocardial interface. From an electrocardiographic standpoint, pacemaker...
Failure To Capture

Failure to capture is defined as the delivery of a pacing stimulus without subsequent depolarization. (See Figure 5, view A, page 11.) Failure of depolarization may be functional or pathologic. Functional failure to capture occurs if a pacing impulse is delivered when the myocardium is in a refractory state. Pathologic failure to capture may occur in the setting of myocardial disease, electrolyte disturbance, and antiarrhythmic drugs. The same conditions can also result in latency, which can sometimes be mistaken for failure to capture. Occasionally, isoelectric depolarization can give the appearance of failure to capture, in which case multichannel recording can confirm that depolarization is occurring.

Lead dislodgement, perforation, fracture, or an insulation defect can increase the pacing threshold and cause intermittent or persistent failure to capture. Increased pacing threshold can occur several weeks after pacemaker placement due to excessive fibrosis at the electrode-myocardial interface. Capture threshold can also be increased in the setting of hypothyroidism and infiltrative cardiomyopathies. Among the medications that are most likely to affect pacing thresholds are class IC antiarrhythmic drugs. Hyperkalemia is also an important cause of failure to capture.

Failure To Pace

Failure to deliver a stimulus to the heart is referred to as failure to pace. The clearest indication of this is absence of pacing artifact when the intrinsic rate is less than the set lower limit of the device. (See Figure 5, view B, page 11.) Oversensing is the most common cause of failure to pace. Oversensing can occur due to partial lead fracture or insulation defects. Crosstalk refers to a specific type of oversensing, in which the ventricular lead senses an atrial pacing stimulus, thereby inhibiting ventricular output. A programmed ventricular blanking period following atrial pacing helps to prevent crosstalk. Intrinsic signals that can be oversensed include retrograde P waves, T waves, ventricular afterdepolarizations, and skeletal muscle myopotentials. Myopotentials from the pectoralis, diaphragm, and rectus abdominus muscles can all cause oversensing. External electromagnetic interference can also result in inappropriate oversensing.

Blunt trauma to the pulse generator may cause failure to pace due to output failure. Lead fracture, dislodgement, or loose connections can also result in output failure and failure to pace.

Undersized pacing artifacts may not be visible on the ECG and can give the appearance of failure to pace. Intrinsic events (such as a premature atrial contractions or premature ventricular contractions) that may not be appreciated on a single telemetry lead can give the false impression of failure to pace when, in fact, there is appropriate inhibition of pacing.

Table 7. Causes Of Pacemaker Malfunction, By Category

<table>
<thead>
<tr>
<th>Source of Malfunction</th>
<th>Failure To Capture</th>
<th>Failure To Sense</th>
<th>Failure To Pace</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional failure</td>
<td>Pacing output delivered when myocardium functionally refractory</td>
<td>Intrinsic event delivered during programmed blanking period or refractory period</td>
<td>Appropriate inhibition</td>
</tr>
<tr>
<td>Pseudofailure</td>
<td>Isoelectric depolarization; recording artifacts</td>
<td>Not applicable</td>
<td>Small bipolar pacing artifacts; ventricular avoidance algorithms</td>
</tr>
<tr>
<td>Pulse generator</td>
<td>Circuit failure; battery depletion</td>
<td>Battery depletion</td>
<td>Blunt trauma; battery depletion</td>
</tr>
<tr>
<td>Lead</td>
<td>Lead fracture; lead insulation break; lead dislodgement</td>
<td>Lead fracture; lead insulation break; lead dislodgement</td>
<td>Lead fracture; lead insulation break</td>
</tr>
<tr>
<td>Electrode-myocardial interface</td>
<td>Electrolyte abnormality (hyperkalemia); drug effect (class IC antiarrhythmic drugs); myocardial infarction; defibrillation/cardioversion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intracardiac signals</td>
<td>Not applicable</td>
<td>Change in intrinsic complex (new bundle branch block)</td>
<td>Retrograde P waves; afterdepolarizations; T waves; crosstalk; far-field sensing</td>
</tr>
<tr>
<td>Extracardiac signals</td>
<td>Electromagnetic interference</td>
<td>Electromagnetic interference</td>
<td>Electromagnetic interference</td>
</tr>
</tbody>
</table>
change in the morphology or vector of the depolarization pattern. Premature ventricular contractions, bundle branch block, and certain forms of ventricular tachycardia may not exceed the sensing thresholds applied to the previous intrinsic depolarization pattern; however, undersensing is most commonly the result of a break in lead insulation. Undersensing that occurs soon after implantation can be caused by lead dislodgement or perforation. Class IC antiarrhythmic drugs and hyperkalemia may also alter the sensing threshold. Finally, undersensing can occur as the pacemaker battery becomes depleted.

**Electromagnetic Interference**

For simplification, recent reviews have separated electromagnetic interference sources into nonmedical sources and medical sources. For simplification, recent reviews have separated electromagnetic interference sources into nonmedical sources and medical sources.

**Figure 5. Electrocardiogram Appearance of Pacemaker Failure**

Electrocardiographic appearance of different types of pacemaker malfunction. (A) The appearance of appropriately timed pacing artifacts that are not immediately followed by expected P or QRS waves may indicate that a pacemaker’s failure to capture is the cause of a patient’s symptoms. In this case of atrioventricular pacing, atrial pacing artifacts are followed by inverted P waves, whereas ventricular pacing artifacts are not immediately followed by any QRS waves. The asterisk identifies where QRS waves would be expected to appear in the first 2 beats. The first beat in this strip is a native atrial impulse (appropriately sensed) followed by ventricular pacing artifact that fails to capture. The following beats are atrial-paced with capture and ventricle-paced with failure to capture. (B) Failure of pacing artifacts to appear when expected may indicate that a pacemaker is malfunctioning by failing to pace. This is a ventricular-paced rhythm. The absence of a fifth pacing artifact represents failure to pace, commonly caused by oversensing. (C) The appearance of a pacing artifact at an inappropriate location on the electrocardiogram may indicate that the pacemaker is failing to sense native conduction. The asterisk indicates a pacing artifact appearing too soon after a native QRS wave.


**Nonmedical Sources Of Interference**

Common household appliances (such as televisions or microwave ovens) do not interfere with pacemaker function. Potential nonmedical sources of electromagnetic interference include cell phones, security gates, and electronic article surveillance devices. Pacemaker device manufacturers have developed filters that account for common frequencies employed by cell phones. In a study of 679 patients, interaction between cell phones and pacemakers was found in only 0.3% of patients, and there were no clinical manifestations. The consequences of the interaction were mostly activation of asynchronous pacing and, rarely, oversensing or inappropriate tracking. Interactions occurred at higher rates with unipolar lead pacemakers and only during the ringing phase when the phone was positioned within 10 cm of the pacemaker. Cell phones have not been shown to interact with ICDs.

Despite the low incidence of interaction with cardiac devices, the common-sense recommendation for patients should be to position the phone at the ear contralateral to the device. Patients with cardiac devices may trigger alarms at airport security gates when they are screened, but no alteration of function should be expected. In a study of 388 patients screened with handheld metal detectors, there were no observed pacing or sensing abnormalities or reprogramming. Interactions with article surveillance devices in retail stores are rare but may occur during prolonged exposure, resulting mostly in asynchronous pacing. It should be recommended that patients avoid lingering near these security gates. Additionally, digital music players can cause some interference but do not directly affect the device. However, portable headphones often include a powerful magnetic substance that can produce electromagnetic interference and interactions when placed within 3 cm of the cardiac device.

**Medical Sources Of Interference**

Medical sources of electromagnetic interference include electrocautery (most common), magnetic resonance imaging (MRI), ventricular assist devices, extracorporeal shock wave lithotripsy, therapeutic ionizing radiation, and external cardioversion. Physical movement of the cardiac device has not been reported during MRI, but MRI may affect pacemaker function through 3 mechanisms: (1) the static magnetic field of the MRI will typically close the reed switch and lead to asynchronous pacing; (2) the gradient magnetic fields of the MRI may cause alteration of function or dysrhythmia through direct current effects on pacemaker output circuits; and (3) the radiofrequency energy of the MRI may be captured by pacemaker leads, which act as antennae to concentrate the energy to myocardium.

In one study, 555 MRI studies were performed
on 438 patients with cardiac devices. Only 3 devices went into a temporary back-up mode, and there were no significant changes in function, leading the authors to conclude that MRI can be done safely in monitored patients with selected devices. The AHA and the American College of Radiology (ACR) offer guidelines that state that the decision to perform an MRI on patients with cardiac devices should be patient-specific, with assessment of risks and benefits, and coordination by both cardiology and radiology services. Therapeutic radiation can produce electromagnetic interference that may directly damage the device circuitry. Manufacturers recommend against radiation therapy if the device is within the treatment field, and there are recommendations for maximum doses.

External direct cardioversion or defibrillation may also cause device malfunction. Pacing and sensing function may be affected as a result of associated increases in the myocardial stimulation thresholds, some of which may be permanent. Activation of the backup function/rate or mode switch may also occur. In patients with bipolar systems, anterio-posterior cardioversion with pads more than 8 cm away from the device will rarely cause adverse effects. Contemporary extracorporeal shock wave lithotripsy can be performed safely in patients with cardiac devices, as the rate of interaction is estimated to be less than 1%.

Management

Approach To The Pacemaker Patient With Tachycardia

Pacemaker-mediated tachycardia (also known as pacemaker re-entrant tachycardia or endless loop tachycardia) is observed exclusively in patients with dual-chamber devices. Pacemaker-mediated tachycardia is similar to other re-entrant dysrhythmias except that the pacemaker itself forms part of the re-entry circuit. An intrinsic premature complex is sensed by the atrial lead of the pacemaker, which responds by generating a ventricular impulse. This ventricular impulse is then conducted retrograde through the atioventricular node to the atria. The impulse, now in the atria, is sensed by the atrial lead to complete the loop and again trigger the pacemaker to generate a ventricular impulse. The ECG will show a regular wide-QRS tachycardia with a pacing artifact prior to each complex, and retrograde P waves may be seen. (See Figure 6, view A.) This re-entrant tachycardia will not exceed the pacemaker’s programmed upper rate limit; however, tachycardia may be significant enough to cause symptoms necessitating emergent treatment. A trial of adenosine may or may not be effective in treating this tachycardia. On the other hand, placing a magnet over the pacemaker will definitively terminate this re-entrant dysrhythmia. After a pacemaker-mediated tachycardia event is successfully treated, the device will need to undergo adjustments to its atrial sensing thresholds. Most modern devices include programming to prevent pacemaker-mediated tachycardia. With such programming, the device may disregard atrial stimuli when atrial rates exceed its own upper limit, or it can mode-switch to discontinue atrial tracking altogether. (See Figure 6, views B and C.)

In patients who have older devices with unipolar leads, abnormal, rapid ventricular pacing may occur through a different mechanism. Unipolar systems are more susceptible than bipolar devices to sensing of myopotentials from neighboring muscle activity, and these myopotentials can trigger a rapidly paced rhythm.

In patients who are ventricular paced, sinus tachycardia (whether physiologic or pathophysiologic) will result in rapid ventricular pacing up to the preset rate limit. Some pacemakers have rate-responsive sensors that can detect changes in physiological parameters (ie, minute ventilation, QT interval, and stroke volume) and permit higher heart rates when dictated by the environment. It must be noted that sensory-induced tachycardia will appear identical to pacemaker-mediated tachycardia on an ECG; differentiation between them may be possible only through device interrogation.

Figure 6. Pacemaker-Mediated Tachycardia

(A) First 3 asterisks indicate premature ventricular contractions with retrograde P that triggers onset of PMT. (B) Arrow shows lack of atrial capture; a failed attempt at termination by device. (C) Arrow indicates successful atrial capture and termination of PMT.

Abbreviation: PMT, pacemaker-mediated tachycardia.

Magnets turn off the antitachycardia functions of an ICD without affecting its ability to perform backup bradycardia pacing. As such, magnets can be used to terminate inappropriately delivered device shocks. Some ICDs are programmed to play alert tones to indicate high lead impedance, pacing impedance, battery depletion, and delivery of more than 3 shocks. These tones can be replayed to the clinician by applying a magnet over the ICD.106

Advanced Cardiovascular Life Support In Patients With Pacemakers Or Implantable Cardioverter-Defibrillators

With few exceptions, Advanced Cardiovascular Life Support (ACLS) procedures should be performed in patients with pacemakers or ICDs in the same manner as in those without the devices. Shocks delivered by an implanted device during a cardiac arrest do not pose a risk to ACLS providers or other equipment.107 External defibrillation is ordinarily not necessary in a patient with a device that has antitachycardia function; however, it should be noted that a dysrhythmia may persist after a normally functioning pacemaker or ICD has completed its programmed sequence of therapy. To conserve battery life, typical sequences are limited to 5 successive shocks. In some cases, the implanted device

The Magnet

Most pacemakers and ICDs have magnetic reed switches. In pacemakers, placement of a magnet turns off the sensing function, effectively making the pacemaker fire asynchronously at a specified rate. Removal of the magnet causes the pacemaker to switch back to its programmed mode.

The magnet response is specific to the manufacturer, model, and programmed response of the device. Each pacemaker has a fixed rate programmed to reflect different levels of battery status. For example, in a Medtronic pacemaker (Medtronic, Inc., Minneapolis, MN), a rate of 85 beats/min reflects beginning-of-life status and 65 beats/min indicates that elective replacement is indicated. At the battery’s end of life, the response of a pacemaker to the magnet is unpredictable.105 If there is no electrocardiographic response to a magnet, there are several possibilities to consider. (See Table 8.) The battery may be at the end of its life or completely depleted, the magnet may need to be repositioned, or the pacemaker may be distanced by adipose tissue. More than 1 magnet may be needed to activate the reed switch of a pulse generator placed in an obese patient. Rarely, a device may also be programmed to ignore magnet application (magnet “OFF” mode).

There are several clinical applications for placing a magnet on a pacemaker, one of which is to terminate pacemaker-mediated tachycardia, as the ventriculoatrial limb of the re-entrant circuit is broken when atrial sensing is disabled by a magnet. (See Table 9.) Inappropriate sensing (whether from electromagnetic interference or another cause) will be inhibited by placement of a magnet. Absent pacing artifacts on an ECG can distinguish output failure or lead disruption from oversensing.

Some devices are programmed to initiate ECG storage (electrogram mode) under the influence of a magnet. By applying a magnet, the patient can trigger the pacemaker to store ECG data during episodes of palpitations or light-headedness.

### Table 8. Expected Response To Magnet Placement Over Pacemaker/Implantable Cardioverter-Defibrillator And Clinical Implications Of Different Magnet Responses

<table>
<thead>
<tr>
<th>Tachyarrhythmia therapy</th>
<th>Implantable Cardioverter-Defibrillator</th>
<th>Permanent Pacemaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effect on pacing</td>
<td>Not applicable</td>
<td>Asynchronous pacing (DOO, AOO, VOO)</td>
</tr>
<tr>
<td></td>
<td><strong>•</strong> At 85 beats/min*: Functioning battery at beginning of life</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>•</strong> At 65 beats/min*: Elective replacement interval</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>•</strong> No pacing response: Depleted battery; battery at end of life; pacemaker is programmed to ignore magnet for safety (electrogram mode); magnetic field doesn’t reach device (deep placement or obese patient); poorly positioned magnet</td>
<td></td>
</tr>
</tbody>
</table>

| Upon magnet removal     | Tachyarrhythmia therapy restored     | Sensing function restored |

*Rates specific for Medtronic pacemaker (Medtronic, Inc., Minneapolis, MN, USA).
For an explanation of pacemaker codes, see Table 2, page 3.
**Clinical Pathway For Suspected Pacemaker Malfunction**

1. **Obtain device history**
2. **Obtain 12-lead ECG**
3. **Pacemaker malfunction apparent on ECG?**
   - **YES**
   - **Electrolyte abnormality?**
     - **YES**
       - Correct electrolyte abnormality (Class II)
     - **NO**
       - Change in ST segments or positive cardiac markers?**
         - **YES**
           - Antithrombotic therapy
           - Revascularization (Class II)
         - **NO**
   - **NO**
     - Patient asymptomatic during ECG but reports recent symptomatic episodes
     - Interrogate device
       - Replace battery, if necessary
       - If abnormal lead impedance, obtain chest x-ray (Class II)

**Learning Objectives**

1. To understand the symptoms and diagnosis of pacemaker malfunction.
2. To learn the appropriate actions to take when suspecting a pacemaker malfunction.

**Abbreviation:** ECG, electrocardiogram.

---

**Class Of Evidence Definitions**

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

**Class I**
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

*Level of Evidence:*
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

**Class II**
- Safe, acceptable
- Probably useful

*Level of Evidence:*
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

**Class III**
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

*Level of Evidence:*
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

**Indeterminate**
- Continuing area of research
- No recommendations until further research

*Level of Evidence:*
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

---

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient’s individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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1. “The ventricular-paced rhythm makes the ECG uninterpretable for diagnosing MI. I’ll call cardiology if the troponin comes back positive.” While concordant ST-segment changes and exaggerated discordance are insensitive findings for diagnosing acute MI in paced rhythms, they are diagnostically useful, when present.

2. “Pacing artifacts appear stranded among widened and regular QRS complexes at a ventricular rate of 42. There must be a problem with the pacemaker.”

Hyperkalemia can cause QRS widening, bradycardia, and failure to capture. Obtain a serum potassium level quickly and consider immediate administration of calcium. Always consider hyperkalemia before attributing failure to capture to a hardware problem or programming error.

3. “I thought electrical cardioversion was contraindicated in patients with permanent pacemakers.”

Electric direct-current cardioversion is safe as long as the electrodes are placed in anterior-posterior orientation at least 8 cm away from the pacemaker device. Device interrogation for proper functioning should be performed after cardioversion.

4. “Pacemaker-mediated tachycardia was on my differential, but I was afraid to place a magnet over the pacemaker.”

A pacemaker magnet only disables the sensing function, not the pacing function, and it can be effective in terminating pacemaker-mediated tachycardia as well as for differentiating among various types of potential pacemaker malfunction.

5. “I thought all newer pacemakers were MRI-compatible.”

While advances in pacemaker design have led to the development of pacing leads and pulse generators that are safe for the magnetic environment, safe MRI requires careful screening, established protocols, and physician supervision.

6. “I didn’t think to compare the chest x-ray with a previous one or consider lead dislodgement as a complication that could occur so far out from implantation.”

Lead migration more commonly occurs shortly after implantation, but it can be a late complication. Comparison with previous x-rays is helpful in detecting macro lead dislodgements.

7. “The 12-lead ECG appeared normal. I didn’t think the patient needed device interrogation.”

Pacemaker interrogation is critical in evaluating the possibility of pacemaker dysfunction that might not be apparent on a 12-lead ECG.

8. “The patient experienced syncope and has a pacemaker, so she is a high-risk cardiac patient and needs to be admitted to telemetry.”

Pacemaker interrogation can be performed in the ED to rule out dysrhythmia and pacing malfunction as underlying causes of palpitations, syncope, and light-headedness. Device interrogation may help to avoid unnecessary hospital admission in some of these patients.

9. “I didn’t think that hiccoughs could be related to the patient’s pacemaker.”

Direct stimulation of the diaphragm or the phrenic nerve occurs with lead dislodgement or with high output from a left ventricular lead placed in the coronary sinus.

10. “Ventricular tachycardia clearly wasn’t the cause of his symptoms. If he had ventricular tachycardia, his CRT-D device would have shocked him.”

Ventricular tachycardia may occur at rates below the programmed tachycardia detection rate (TDR). Above the TDR, ICD therapy is administered. A low TDR can be programmed, with the risk of administering therapy inappropriately to supraventricular tachyarrhythmias. Antitachycardia pacing and algorithms to improve the specificity of VT detection have reduced the incidence of inappropriate ICD therapy for supraventricular arrhythmias.
may fail to deliver appropriate therapy, as in situations where rhythm sensing or shock delivery is compromised. For example, an ipsilateral tension pneumothorax may raise the defibrillation threshold, leading to ineffective shocks. Antiarrhythmic agents that block fast inward sodium channels (ie, lidocaine) may also increase the threshold, but agents that prolong repolarization (ie, sotalol) may decrease the threshold. Keeping all of these in mind, if external electrical therapy is required, transcutaneous pads should be placed anterior-posteriorly if possible, and as far from the pacemaker as possible to avoid damage to the device. The rescuer may also consider magnet placement to avoid unintentional device-mediated therapies during resuscitation. After successful resuscitation, the device should be interrogated to ensure proper status of its parameters.

If central venous access is required, the femoral vein is the safest approach in a patient with a pacemaker. Pacemaker wires in the subclavian vein are often thrombosed, and may preclude normal ipsilateral subclavian vein cannulation. In addition, contact between a procedural guidewire and a lead can trigger an inappropriate response.

Approach To The Pacemaker/Implantable Cardioverter-Defibrillator Patient Who Receives A Shock

Many contemporary pacemakers include antitachycardia functions via combinations of overdrive pacing, cardioversion, and/or defibrillation programmed to occur at designated detection zones. Newer-generation devices employ algorithms to distinguish ventricular from supraventricular tachycardia in order to reduce the risk of inappropriate shocks. These devices use rhythm characteristics such as onset, rate stability, electrogram morphology, and atrioventricular dissociation to differentiate ventricular from supraventricular tachycardia. In one study, a dual-chamber algorithm reduced the rate of inappropriate detection by 50%. The occurrence of a single shock in a patient without preceding symptoms does not require immediate ED consultation with an electrophysiologist; referral within the week is generally appropriate. However, in the presence of symptoms such as dyspnea, chest pain, or weakness, underlying etiologies such as myocardial ischemia, decompensated heart failure, or electrolyte imbalance should be considered. The patient who receives multiple shocks should receive immediate attention and cardiac monitoring in the ED until the device can be interrogated. Multiple inappropriate shocks may occur in response to supraventricular tachycardia and may require reprogramming by an electrophysiologist or representative from the device manufacturer. If defibrillation therapy is found to be appropriate (delivery of 3 or more appropriate shocks within a 24-hour period is termed electrical storm), the patient should be evaluated for possible electrolyte abnormalities (potassium and magnesium), myocardial ischemia, proarrhythmic drug effect, or other causes of QT prolongation. In cases of electrical storm, amiodarone, intravenous beta-adrenergic receptor antagonists, and propofol are suggested drug therapies. Refractory cases of electrical storm may require general anesthesia, left ventricular assist devices, and emergent radiofrequency ablation. The diagnosis of MI may be difficult in these patients, as transient postshock ST-segment deviation is common and myocardial injury can be caused by the shocks themselves.

Disposition

The majority of patients presenting with implant-related complications or pacemaker malfunction will require consultation with a cardiologist and, potentially, hospital admission. Pacemaker interrogation can be performed in the ED to rule out arrhythmia and pacemaker malfunction, and to avoid unnecessary hospital admission in some of these patients.

Controversies And Cutting Edge

Remote Monitoring

Some devices allow for remote monitoring of data, including heart rate variability, tachyarrhythmia frequency and duration, intrathoracic impedance, and percentage of time that the rhythm is paced. Integration of these data points can be clinically useful in predicting heart failure hospitalizations. Future research is required to define how remote monitoring might lead to improved clinical outcomes.

Leadless Pacing

As described above, many pacemaker complications relate either to transvenous leads or the subcutaneous pulse generator. Leadless pacing systems are currently under development and early experience has demonstrated safety and efficacy in single-chamber ventricular pacing.

Device Deactivation

Patients with terminal illness may request device deactivation along with do not resuscitate (DNR) status. The HRS has published a consensus statement that declares deactivation of an implanted device as ethical and legally permissible. Emergency clinicians should engage patients in conversation about advance directives as they relate to their cardiac device.
Summary

Emergency clinicians must be familiar with the indications for pacemaker placement and aware of the complications associated with implantation. A systematic approach to evaluating the pacemaker is key to expeditious diagnosis of device malfunction. This includes careful history taking, a thorough physical examination of the patient, and accurate interpretation of the 12-lead ECG and chest radiographs. Device interrogation is a critical step in troubleshooting with regard to both pacemaker and antitachycardia functions.

Case Conclusions

Before reflexively calling the electrophysiologist to interrogate the pacemaker of the 78-year-old woman with widened QRS complexes and bradycardia, you paused to consider a life-threatening cause of both QRS widening and failure to capture. You sent for a potassium level on a blood gas sample and administered calcium gluconate before finding out the lab result. You watched the QRS complexes on the monitor narrow to their previous width as the heart rate increased. After another dose of calcium gluconate, you noticed that all pacing artifacts were captured. Lab results returned, showing a serum potassium level of 8.7 mEq/L, with prerenal azotemia. You consulted the nephrologist and admitted the patient to the ICU.

You obtained a chest x-ray on the 38-year-old woman with sarcoidosis and compared it to the x-ray obtained shortly after implantation of her pacemaker. You noticed that both atrial and ventricular pacing leads were retracted into the superior vena cava and wound around the vertical axis of the pulse generator. The patient denied the vertical axis of the pulse generator. You no

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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CME Questions

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1. Cardiac resynchronization therapy is indicated for patients with heart failure and:
   a. Recurrent ischemic events
   b. Ventricular dysrhythmias
   c. A left bundle branch block
   d. Atrioventricular nodal conduction delay

2. Electrocardiographic findings indicative of severe hyperkalemia in a patient with a paced rhythm include:
   a. QRS widening
   b. Failure to capture
   c. Sine wave QRS morphology
   d. All of the above

3. Acute myocardial infarction in a paced rhythm:
   a. Is always electrocardiographically silent
   b. Can be detected by elevated ST segments in a pattern of exaggerated discordance
   c. Is the most common cause of failure to sense
   d. Can be detected by applying a magnet

4. The manufacturer of a pacemaker can be identified by:
   a. The amplitude of pacing artifacts on the 12-lead ECG
   b. The size of the pulse generator
   c. Placing a magnet over the pulse generator
   d. None of the above

2879. (Randomized controlled trial; 400 patients)
120. Whellan DJ, Ousdigian KT, Al-Khatib SM, et al. Combined heart failure device diagnostics identify patients at higher risk of subsequent heart failure hospitalizations: results from PARTNERS HF (Program to Access and Review Trending Information and Evaluate Correlation to Symptoms in Patients With Heart Failure) study. J Am Coll Cardiol. 2010;55(17):1803-1810. (Retrospective observational trial; 694 patients)
5. Management of an infected device usually involves:
   a. Intravenous antibiotics and device removal
   b. Needle aspiration of the infected pocket and intravenous antibiotics
   c. A prolonged course of oral antibiotics
   d. Incision and drainage of the pocket followed by wound care

6. What is the most common cause of failure to pace?
   a. Lead fracture
   b. Oversensing
   c. Lead dislodgement
   d. Battery depletion

7. MRI in patients with pacemakers:
   a. Is uniformly contraindicated
   b. Can be performed without complications as long as the pacemaker was manufactured after 2002
   c. Can be performed, as long as a magnet is placed over the implanted device
   d. Can be performed in some cases, but only if specific guidelines are met

8. What is the mechanism of pacemaker-mediated tachycardia?
   a. A re-entrant circuit
   b. Increased automaticity at the site of the ventricular electrode tip
   c. Cardiac remodeling
   d. Delayed afterdepolarizations

9. Placing a magnet on a pacemaker effectively:
   a. Switches the pacemaker into an asynchronous mode
   b. Turns pacing off
   c. Turns sensing on
   d. Switches the pacemaker into a demand pacing mode

10. External defibrillation in a patient with an implanted cardiac device:
    a. Is contraindicated
    b. Is not necessary if the device has anti-tachycardia function (ICD)
    c. Can be performed if pads are placed a safe distance away from the device
    d. Can be performed, but only at 50 joules

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