Preoperatively

- Identify the generator manufacturer and model (pacemaker, transvenous defibrillator, subcutaneous defibrillator) of the cardiovascular implantable electronic device (CIED).
- Establish preoperative contact with the patient’s CIED physician or clinic to obtain appropriate records and a perioperative prescription (Heart Rhythm Society [HRS]). Have the CIED interrogated (American Society of Anesthesiologists [ASA]) by a competent authority shortly before the anesthetic regimen.
- Obtain a copy of this interrogation. Obtain a perioperative prescription from the CIED physician (HRS). Ensure that implantable cardioverter-defibrillator (ICD) treatment settings are appropriate and that the CIED will pace the heart.
- Consider replacing any CIED near its elective replacement period in a patient scheduled to undergo either a major surgical procedure or a surgical procedure where monopolar electrosurgery will be used within 25 cm of the generator.
- Determine the patient’s underlying rate and rhythm to determine the need for backup (external) pacing support.
- Ensure that all magnet behavior (pacing, suspension of shock therapy) is appropriate if magnet use is planned.
- Program minute ventilation rate responsiveness “off,” if present.
- Consider disabling all rate enhancements to prevent misinterpretation of cardiac rhythm.
- Consider increasing the lower rate limit to provide optimal oxygen delivery for major procedures.
- If electromagnetic interference is likely, (1) disable antitachycardia therapy if a defibrillator is present and (2) consider asynchronous pacing for some pacing-dependent patients. Magnet application may be acceptable for some ICDs (disable antitachycardia therapy) or pacemakers (provide asynchronous pacing). Asynchronous pacing from an ICD always requires reprogramming.

Intraoperatively

- Monitor cardiac rhythm with the pulse oximeter (plethysmography) or arterial waveform analysis.
- Consider disabling the “artifact filter” on the electrocardiogram monitor. If a minute ventilation sensor is present and active, ensure that respiratory rate monitoring is disabled.
- Ask the surgeon to operate without the monopolar electrosurgical unit (ESU).
- Use a bipolar ESU if possible; if not possible, then pure “cut” electrosurgery is better than “blend” or “coag,” and ESU should be applied in short bursts (<4 seconds) separated by at least 2 seconds.
The nature of anesthesiology practice requires a wide breadth of knowledge, often encompassing situations that occur rarely throughout a career. Nowhere in medicine does this task seem more difficult than the subject of the cardiac implanted electronic device (CIED). Many factors lead to confusion regarding the behavior of a CIED and the perioperative care of a patient with a CIED. Some patients have old, technologically outdated devices that still appear to function. New developments seem to take place rapidly, and case reports, textbooks, and literature reviews sometimes present incorrect material or do not keep pace with these new developments. Proprietary features, differing magnet behaviors (even among models from the same company), confusion regarding CIED type and available therapies, and the lack of standardization make synthesis of generalizations difficult, with the potential of putting the patient at risk. Finally, preventable adverse events that can cause injury or death in patients are thought to occur “by chance” (see the later section on temporary pacing).

Battery-operated pacing devices were introduced by C.W. Lillehei (a cardiothoracic surgeon) and Earl Bakken (an electrical technician) in 1958, just 4 years after the invention of the transistor. In 1960, Wilson Greatbatch, an engineer, created the first implantable battery-powered device in his barn in Buffalo, New York. The natural progression of pacemaker (PM) developments led to the invention of the implanted cardioverter-defibrillator (ICD) in approximately 1980 by Michael Morchower in Baltimore. First approved by the U.S. Food and Drug Administration (FDA) in 1985, implantation of ICDs required thoracotomy, a significant operation for the patient with poor heart function. Advances in electronic miniaturization, as well as improvements in battery technology, led to the development of very small (10-mL volume), but electronically complicated, programmable pacing devices.

Technologic advances (transvenous lead placement, antitachycardia pacing [ATP] capability, continued miniaturization, advanced pacing capability, and numerous survival benefits) led to increases in ICD implantation. Since 1997, transvenous ICDs (T-ICDs) have been approved to provide permanent antibradycardia pacing.

Sophisticated three-chamber (atrium, right ventricle, and left ventricle) pacing to provide cardiac resynchronization therapy (CRT; also called biventricular [BiV] pacing) from both PMs (CRT-P) and ICDs (CRT-D) became available in the United States in 2001. In 2009, Cameron Health (San Clemente, Calif) received a CE mark (mandatory conformity marking for certain products sold within the European Economic Area since 1985) for their subcutaneous ICD (S-ICD). Boston Scientific/Guidant Medical/CPI (BOS; Natick, Mass) purchased this company in mid-2012 and received US FDA approval for the S-ICD in September 2012.

As noted, these advances and expanding indications for implantation can create confusion when caring for patients with CIEDs. A T-ICD in a pectoral position could be mistaken, by virtue of pacing “spikes” on the surface electrocardiogram (ECG), for a (non-ICD) PM. Additionally, T-ICDs have been inappropriately called “pacemaker/defibrillators” by medical personnel, the press, and patients. Given that pacing functions in T-ICDs respond to external stimuli (magnet placement, electromagnetic interference [EMI]) differently than do PMs, this confusion could harm the patient. For example, failure to disable shock therapy in the presence of EMI, thereby leading to inappropriate shock during a perioperative experience, could actually shorten a patient’s life.

Published data suggest that significant numbers of patients actually receive an inappropriate ICD shock during hospitalization. Distinguishing a conventional PM from a T-ICD can be accomplished by examining the right ventricular (RV) lead system on a
Figure 48-1. A pacemaker, an implantable cardioverter-defibrillator (ICD), and a right ventricular (RV) defibrillator lead are shown to assist in determination of the type of system using chest radiography. A, Conventional pacemaker with one quadripolar lead that provides atrial and ventricular sensing and ventricular pacing. This chest radiograph shows a number of features of a modern pacing system. The generator is located in the left pectoral region. The single lead enters the subclavian vein under the clavicle but superficial to the first rib (a common site for lead problems, although no problem is demonstrated here). This lead has two electrodes positioned within the right atrium to provide sensing to detect intrinsic atrial activity. The ventricular portion of the lead shows the classic bipolar pattern with a ring electrode just proximal to the tip electrode, and these electrodes can be used for sensing intrinsic ventricular activity as well as depolarizing the ventricle. This particular system is a VDD pacemaking system used to provide atrioventricular (AV) nodal activity in patients with a functioning sinoatrial node and atrium because this system cannot be used to depolarize the atrium. Because the surface electrocardiogram often demonstrates ventricular pacing that tracks the atrial activity, inspection of the surface electrocardiogram often produces an erroneous diagnosis of a dual-chamber (DDD) pacemaker. B, Defibrillator system with biventricular pacing capability. Note that three leads are placed: a conventional bipolar lead to the right atrium, a multipolar lead terminating in the right ventricle, and a unipolar lead to the coronary sinus (CS). The presence of a “shock” conductor in the right ventricle (called a “shock coil”) distinguishes a defibrillation system from a conventional pacing system. Many ICDs have an additional shock coil in the superior vena cava (SVC). Typically, the SVC shock coil is electrically identical to the defibrillator case (called the “can”). When the defibrillation circuitry includes the ICD case, it is called “active can configuration.” This particular system is designed to provide “resynchronization therapy” in the setting of dilated cardiomyopathy with a prolonged QRS complex (and frequently with a prolonged PR interval as well). The bipolar lead in the right atrium will perform both sensing and pacing function. Similarly, the tip electrode in the right ventricle along with the shock coil performs RV pacing and sensing functions. When the second RV pacing electrode is merged with the shock coil, the lead is called “integrated bipolar” because unipolar RV pacing is not permitted in an ICD system. The lead in the CS depolarizes the left ventricle (LV). Failure to depolarize one of the ventricles could result in ventricular oversensing (and inappropriate antitachycardia therapy) in an ICD. C, Integrated bipolar RV defibrillator lead. The tip of this lead becomes enmeshed in the RV trabeculae (called a “tined” lead), rather than engaging the myocardial wall with a screw (“active fixation”). This particular lead has an SVC shock coil as well.
chest radiograph (Fig. 48-1). A chest radiograph can also be used to identify the generator manufacturer (Fig. 48-2).

Pacemaking and defibrillation systems have a remarkable record for reliability, but they can, and do, fail. Laskey and associates analyzed FDA records for T-ICD report explantations for the period from 2003 to 2007 (459,000 T-ICDs and 256,000 CRT-Ds implanted) and found an annual explantation rate of 5.0% for T-ICD and 8.3% for CRT-D. A similar study for PMs (1990 to 2002; 2.25 million implants) showed a 0.4% failure rate. Thus, every patient with a cardiac generator should undergo regular in-office follow-up. For appropriately selected devices, remote (i.e., telephone) checks can be used to identify battery depletion, programming, sensing issues, arrhythmia detection (PM and ICD), and arrhythmia treatment (ICD). Some PMs and T-ICDs have automated sensing and capture testing that can report problems, but commanded iterative testing, such as pacing thresholds, cannot be performed. For “stable” PMs, Medicare coverage includes telephone evaluation every 4 to 12 weeks. The Heart Rhythm Society and European Heart Rhythm Association (HRS/EHRA) recommend evaluation (remote or in person) every 3 to 12

![Figure 48-2. Radiographic identifiers for some generator manufacturers. Pacemaker and implantable cardioverter-defibrillator generators can be identified from operative dictations, patient cards, or some chest radiographs. Using digital x-ray equipment with postprocessing zoom capability, corporate x-ray logo identifiers from Biotronik (A), Boston Scientific (B), CPI (C), ELA (D), Guidant (E), Medtronic (F), Pacesetter (G), Sorin (H), and St. Jude Medical (I) are shown. For Sorin devices, all identifiers start with “SP,” and the third character identifies the actual model. *Currently used logo.](image-url)
months. Medicare coverage and recommendations (HRS/EHRA) include at least one in-person evaluation every year. Medicare has no coverage determination for ICD follow-up care; HRS/EHRA recommends evaluation (remote or in person) every 3 to 6 months with at least one in-person evaluation annually.9,10

Issues also develop leading to “notices,” “product advisories,” or “recalls” from the various manufacturers that may affect perioperative care. For example, BOS reported T-ICD magnet switch issues that could prevent delivery of shock. Two separate groups have been identified: (1) certain T-ICDs manufactured before 2005 (~2000 remaining implants) whose magnet switches should be permanently disabled and (2) some T-ICDs manufactured between January 2006 and December 2007 (~34,000 remaining implants) that must be checked for “tones/beeps” following any magnet removal. Any ICD with the magnet switch disabled will deliver a shock even with a magnet in place (Fig. 48-3).11,4 The Medtronic (Minneapolis, Minn) Sprint Fidelis ICD lead series reports ventricular activity in the absence of true electrical systoles (oversensing), and this can result in failure to pace or the delivery of inappropriate shock (Fig. 48-4). Recommended actions included programming changes and use of telephone monitoring.12 In December 2010, St. Jude Medical (Sylmar, Calif) reported that their Riata ICD leads (227,000 implanted) could develop insulation failures, resulting in inappropriate ICD discharge or pacing failures. They recommended in-office follow-up every 3 to 6 months.13

The complexity of PMs and ICDs, as well as the multitude of programmable parameters, limits the number of generalizations that can be made about the perioperative care of the patient with an implanted pulse generator (PG). Aging of the population, continued enhancements in implantable technology, new indications for implantation, and new devices continue to drive the growing numbers of implants. In 2005, the ASA published a Perioperative Practice Advisory, which was revised in 2011.14 Also in 2011, the HRS and the ASA published an “Expert Consensus Statement,”15 in which many experts from several societies tried to balance technical, economic, and safety issues in these patients. The Canadian Anesthesia Society and Canadian Cardiovascular Society (CAS/CCS) jointly published a statement in 2012,16 and the United Kingdom’s Medicines and Healthcare Products Regulatory Agency (MHRA) published guidelines in 2006 in which the use of “surgical diathermy/electrocautery” was anticipated.17 The MHRA document is silent on the CIED patient who is undergoing a surgical procedure without the presence of EMI. All the documents report that care of the emergency patient is more complicated than is care of the elective surgical patient (Table 48-1).

Patients with an implanted cardiac PG often have significant comorbid disease in addition to their cardiac rhythm disturbance. The ability to care for these patients requires attention to both their medical and psychological problems. Anesthesiologists also need an understanding of the patient’s PG, its functions, and likely idiosyncrasies in the operating or procedure room.

Finally, not all electronic generators implanted in the chest are cardiac devices, and devices resembling cardiac PGs have been implanted for indications unrelated to cardiac issues. When implanted in the pectoral position (the usual place for current cardiac PGs), these noncardiac devices can be mistakenly identified as cardiac PGs.18 PG implantation has been approved by the FDA for pain control, thalamic stimulation to control Parkinson disease, phrenic nerve stimulation to stimulate the diaphragm in paralyzed patients, and vagus nerve stimulation to control epilepsy and depression.19 Vagus nerve stimulation is also under consideration to treat heart failure,20 as well as perhaps obesity.21 Thus, when evaluating a patient with any PG, one must now determine whether the PG will be pacing the heart, stimulating the central nervous system, stimulating the spinal cord, or stimulating the vagus nerve.

### PACEMAKERS

PM manufacturers report that more than 3000 generators have been manufactured by more than 26 named entities since the 1960s. Currently, industry experts report that more than 350,000 adults and children in the United States undergo new PM placement each year, and likely 3 million patients have PMs today. Worldwide, the prevalence probably approaches 5 million patients.

<table>
<thead>
<tr>
<th>Magnet</th>
<th>Initial value</th>
<th>Present value</th>
</tr>
</thead>
<tbody>
<tr>
<td>On</td>
<td>Off</td>
<td></td>
</tr>
<tr>
<td>Beeper</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beep during capacitor charge</td>
<td>Off</td>
<td></td>
</tr>
<tr>
<td>Beep on sensed and paced ventricular events</td>
<td>Off</td>
<td></td>
</tr>
<tr>
<td>Beep when ERI is reached</td>
<td>On</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 48-3.** Magnet mode setup from a Guidant (now Boston Scientific) Contak Renewal 3 HE model H179. This implantable cardioverter-defibrillator (ICD) is part of an “Urgent Medical Device Safety Information and Corrective Action” letter to physicians from the Guidant Corporation on June 23, 2005, in which a magnet switch problem was announced. The corrective action was designed to disable the magnet switch permanently by programming. As a result, this ICD will not disable shock therapy in response to magnet placement. According to the notice, approximately 46,000 Guidant ICDs were affected and should have their magnet switch permanently disabled. Industry experts believe that fewer than 10,000 of these devices remained implanted and active after January 1, 2013. Note the line below the magnet switch report called “Change tachy mode with magnet.” Effective with software released by Boston Scientific in October 2009, this setting was removed from all ICDs except the Contak Renewal Series. When present and enabled, this setting allows permanent deactivation of ICD function with the application of a magnet for more than 30 seconds. No Boston Scientific ICD with the “BOS” x-ray logo has this feature. ERI, Elective replacement indicator.
Figure 48-4. Ventricular oversensing from damaged leads. A, Real-time telemetry recording from a Guidant implantable cardioverter-defibrillator (ICD) with a Guidant 0154 right ventricular (RV) defibrillation lead reports a spontaneous ventricular (red up arrow) event that was not present on the surface electrocardiogram (ECG), termed ventricular oversensing. In this ICD, the pacing function was DDD (lower rate, 60 beats/minute; atrioventricular delay, 180 msec), and this patient depended on the ICD pacing function for his ventricular systoles. The top tracing is the surface ECG, the second tracing is the intracardiac atrial electrogram (EGM), and the third tracing is the intracardiac ventricular EGM. Along the bottom is the “marker channel,” which shows the pacemaker’s interpretation of events: AP is atrial pace, AS is atrial sense, VP is ventricular pace, and the inappropriate ventricular sensed event is labeled “PVC” because it occurs after a ventricular event without an intervening atrial event. This oversensing has prolonged the R-R interval between the fourth and fifth ventricular systoles because this patient depends on the pacing function for his ventricular systoles. In a pacing-dependent patient, this type of oversensing can lead to asystole. It can also provoke inappropriate antitachycardia therapy (i.e., shock) in a patient with native ventricular activity because these oversensed events can increase the apparent heart rate count to the ICD. B, A patient with a single-chamber Medtronic ICD and a Medtronic Sprint Fidelis RV defibrillation lead was found to have a significant ventricular oversensing issue at interrogation on April 26, 2005, before a head and neck surgical procedure. In this case the ICD is reporting 875 “short R-R intervals” (note the arrow) since the previous interrogation 15 days earlier (April 11, 2005). These data are consistent with a lead fracture causing intermittent “make-break” contacts. In this case, new counts began accumulating 12 minutes after the previous interrogation was complete. More than 100 counts/month is considered a problem; indications to replace the lead include ventricular oversensing in a pacing-dependent patient or a high rate of detection (frequently resulting in shock) triggered by this inappropriate oversensing. EOL, End of life; ERI, elective replacement indicator.
<table>
<thead>
<tr>
<th>Preoperative Recommendation</th>
<th>Intraoperative Magnet Use</th>
<th>ESU Dispersive Electrode Placement</th>
<th>Postoperative Recommendation</th>
<th>Emergency Procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ASA</strong></td>
<td>“Timely interrogation” before elective surgery</td>
<td>Shuns magnet use in favor of reprogramming</td>
<td>Prevent presumed current path from crossing the chest or CIED system</td>
<td>Interrogation recommended; footnotes added to 2011 revision suggesting that CIED reinterrogation not needed if no monopolar ESU used for most cases involving EMI (especially those inferior to umbilicus and where no preoperative reprogramming was performed), interrogation can take place within 1 mo as ambulatory procedure; for reprogrammed CIEDs, hemodynamically challenging cases, cardiothoracic surgery, RFA, and external cardioversion, interrogation before transfer from cardiac telemetry</td>
</tr>
<tr>
<td><strong>HRS/ASA</strong></td>
<td>PM interrogation within 12 mo, ICD interrogation within 6 mo, and CRT interrogation within 3-6 mo; CIED physician must provide prescription for perioperative care</td>
<td>Magnet use suggested for asynchronous pacing (where needed in PM-treated patients) and disabling ICD high-energy therapy provided that patient position does not interfere with magnet access or observation</td>
<td>Prevent presumed current path from crossing the chest or CIED system</td>
<td>Use 12-lead ECG to identify pacing need; presume dependence if 100% pacing</td>
</tr>
<tr>
<td><strong>CAS/CCS</strong></td>
<td>De novo interrogation likely not needed, but CIED physician must provide prescription for perioperative care</td>
<td>When reasonable, magnet use suggested for asynchronous pacing (as needed in PM-treated patients) and disabling ICD high-energy therapy</td>
<td>No mention</td>
<td>Clear plan for postoperative care established before elective case</td>
</tr>
<tr>
<td><strong>MHRA</strong></td>
<td>Preoperative contact with the PM or ICD follow-up clinic for evaluation and perioperative recommendations</td>
<td>Caution advised because magnet behavior can be affected by programming</td>
<td>“Ensure that the return electrode is anatomically positioned so that the current pathway between the diathermy electrode and return electrode is as far away from the pacemaker/defibrillator (and leads) as possible”</td>
<td>Follow-up clinic to prescribe postoperative follow-up</td>
</tr>
</tbody>
</table>

ASA, American Society of Anesthesiologists; CAS/CCS, Canadian Anesthesia Society and Canadian Cardiovascular Society; CIED, cardiac implanted electronic device; CRT, cardiac resynchronization therapy (any CIED that has right ventricular and left ventricular pacing capability); ECG, electrocardiogram; EMI, electromagnetic interference; ESU, electrosurgical unit (“Bovie”); HRS, Heart Rhythm Society. ICD, implanted cardioverter-defibrillator; MHRA, Medicines and Healthcare Products Regulatory Agency; PM, pacemaker; RFA, radiofrequency ablation.

*Recommendations appear relevant only if electromagnetic interference will be present.
A pacemaking system consists of an impulse generator and leads to carry the electrical impulse to the patient’s heart. Leads are connected to the heart’s chambers through the vena cava (transvenous leads), or they are directly sewn onto the surface of the heart (epicardial leads). Leads can be unipolar (one electrode per lead), bipolar (two electrodes per lead), or multipolar (multiple electrodes and wires contained within one lead with connections in multiple chambers) (see Fig. 48-1, A). Because two electrodes are required to complete a circuit, the second electrode in a unipolar configuration is the metal generator case. Use of the case as an electrode requires that the generator pocket be devoid of gas, and electrical continuity has reportedly been disrupted by the use of nitrous oxide.22

PMs with unipolar leads are more sensitive to the effects of EMI, and these systems produce larger “spikes” on an analog-recorded ECG. Most pacemaking systems (except older Autocapture devices from St. Jude Medical) pace in bipolar mode because bipolar pacing usually requires less energy. Bipolar sensing is more resistant to interference from muscle artifacts or stray electromagnetic fields. Often, bipolar electrodes can be identified on the chest film because they will have a ring electrode 1 to 3 cm proximal to the lead tip (see Fig. 48-1, A). However, generators with bipolar leads can be programmed to the unipolar mode for pacing, sensing, or both. Some PMs automatically switch to unipolar pacing or sensing on detection of a lead problem. Unipolar pacing is absolutely contraindicated in the presence of any ICD.

**CODES**

No discussion of PMs can take place without an understanding of the generic PM code, which was published by the HRS, formerly the North American Society of Pacing and Electrophysiology and Heart Rhythm UK, formerly the British Pacing and Electrophysiology Group. This code (NBG5) was initially published in 1983 and was last revised in February 2002.23 It describes the basic behavior of the pacing device (Table 48-2). PMs also come with a variety of terms generally unfamiliar to the anesthesiologist, many of which are shown in the glossary at the end of this chapter.

The first two positions of this code (chamber[s] paced and chamber[s] sensed) seem relatively straightforward. Although early PMs provided only ventricular support, current models can provide pacing in the atria and ventricles, and these devices can also be programmed to determine intrinsic activity in these chambers. The code does not describe the array of diagnostic data that can be accumulated by these devices.

Probably the most confusing aspect of the NBG code is the third position (response to sensed event). Most PMs are programmed either to the DDD mode (atrial and ventricular [“dual-chamber”] pacing and sensing, both triggered and inhibited mode) or to the VVI mode (ventricular pacing in the inhibited mode). Two other modes frequently found are VDD (ventricular pacing with atrial tracking) and DDI (dual-chamber pacing and sensing, but inhibited mode only). In the United States, atrial-only PM placement (AAI mode) is unusual; however, these devices are implanted in patients with sinus node disease, but intact atrioventricular (AV) conduction, in other countries. The NBG third position describes the following behavior:

D (Dual): DDD and VDD pacing provide AV synchrony. In the DDD setting, atrial pacing takes place in the “inhibited” mode (i.e., the pacing device will emit an atrial pulse if no sensed atrial event [or intervening ventricular event, because any ventricular event will reset atrial timing] takes place within the appropriate time frame). In DDD or VDD devices, once an atrial event has occurred (whether native or paced), the pacing device ensures that a ventricular event follows (up to the upper tracking rate [UTR; see the glossary at the end of the chapter]).

I (Inhibited): The appropriate chamber is paced unless intrinsic electrical activity is detected during the pacing interval. For the DDI mode, AV synchrony is provided only when the atrium is paced. If intrinsic atrial activity is present, then no AV synchrony is provided by the pacing function.

T (Triggered): The pacing device emits a pulse only in response to a sensed event. The triggered mode is used when the device is being tested.

The VDD and DDI modes deserve further comment. VDD pacing is used for the patient with AV nodal dysfunction but intact and appropriate sinus node behavior.

---

5 The NBG code is a joint project of the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG), now known as the Hearth Rhythm Society and Heart Rhythm Society UK, respectively. The “N” is NASPE, the “B” is BPEG, and the “G” stands for generic.
VDD pacing is accomplished with a single lead that incorporates atrial sensing electrodes, as well as ventricular conductors that can both pace and sense (see Fig. 48-1, A). A VDD device has no atrial pacing capability. As a result, in a patient who depends on atrial contraction to augment cardiac output, events that result in VVI pacing (e.g., sinus rate lower than programmed rate, battery depletion) or asynchronous ventricular pacing (e.g., magnet placement in many devices, EMI) can lead to deteriorating hemodynamics.\(^{24,25}\)

DDI pacing is indicated for the patient who has a dual-chamber pacing device and also has episodes of paroxysmal atrial dysrhythmia (e.g., paroxysmal atrial fibrillation). DDI pacing prevents high ventricular rates (i.e., pacing at the UTR) that could result from attempted tracking of the atrial arrhythmia, and it provides AV synchrony only when the atrium is paced. Many DDD programs enter the DDI mode on detection of high (programmable) atrial rates (called Mode Switch, Automatic Mode Switch, or Atrial Tachy Response, depending on the manufacturer). When the mode is switched to DDI, perturbations (e.g., very high \(>400\) minute\(^{-1}\)) atrial rates, EMI from the electrosurgical unit [ESU], or magnet placement and removal) may revert back to DDD pacing, with the resultant appearance of AV pacing or ventricular pacing at the UTR (see the section on magnets later in this discussion of pacemakers).

Rate modulation (the fourth position) also remains a poorly understood concept. Because some patients cannot increase their heart rate in response to increased oxygen demand (chronotropic incompetence), PM manufacturers have devised certain mechanisms to detect “patient exercise,” such as sensors that detect vibration, respiration, and pressure (Box 48-1). As the sensor detects “exercise,” it increases the pacing rate (termed-sensor indicated rate). As the exercise tapers, this sensor-indicated rate returns to the programmed lower rate. The sensitivity of these sensors to their exercise signals and the rates of change in pacing are programmable features in current generators. Activation of rate-response algorithms, resulting in an increased paced rate in the operating room, whether from vigorous chest wall skin preparation, pressure on the generator, or EMI in a minute ventilation device, has led to inappropriate treatment and harm to the patient.\(^{26,27}\)

With the 2002 revision of the NBG, the fifth column describes multisite pacing functionality (it had been used to describe antitachycardia function, but this scheme has been abandoned, and a generic defibrillator code has been established). Atrial multisite pacing may prevent atrial fibrillation,\(^{29}\) and ventricular multisite pacing is an acceptable treatment for pacing the patient with dilated cardiomyopathy (DCM).\(^{29-31}\)

**INDICATIONS**

Indications for permanent pacing are shown in Box 48-2 and are reviewed in detail elsewhere.\(^{12}\) Classically, pharmacologic pacing treats patients with sinus node disease (improper impulse formation) and AV nodal disease (improper impulse conduction). CRT pacing for the patient with DCM requires left ventricular (LV) pacing, usually through a lead placed into the coronary sinus (CS) (see Fig. 48-1, B) or sewn onto the LV free wall. In patients with significant RV-to-LV activation time, loss of capture in a ventricle can lead to ventricular overcounting with resultant inappropriate antitachycardia therapy for the patient with a DCM pacing defibrillator.\(^{33,34}\) Pac-Box 48-1  **Rate Modulation (Activity) Sensors**

<table>
<thead>
<tr>
<th>Currently Approved in the United States</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vibration sensor</td>
</tr>
<tr>
<td>Motion sensor</td>
</tr>
<tr>
<td>Minute ventilation (bioimpedance sensor)</td>
</tr>
<tr>
<td>QT interval (Vitatron only)</td>
</tr>
<tr>
<td>Right ventricular pressure (Biotronik only)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Under Investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right ventricular stroke volume</td>
</tr>
<tr>
<td>Blood pH</td>
</tr>
<tr>
<td>Blood temperature</td>
</tr>
<tr>
<td>Mixed venous oxygen sensor</td>
</tr>
<tr>
<td>Systolic time intervals</td>
</tr>
<tr>
<td>Evoked response</td>
</tr>
<tr>
<td>Intracardiac impedance</td>
</tr>
</tbody>
</table>

*Of the types of sensors used to detect exercise in a patient with a cardiac pacemaker, five are currently approved in the United States, although many others remain under investigation. Some devices have two sensors and can be programmed for cross-checking to prevent increases in heart rate from spurious causes. Minute ventilation sensors are very sensitive to stray electromagnetic interference, and patients have been inappropriately treated for pacemaker-driven tachycardias as a result. Perioperative experience with right ventricular pressure sensors is minimal at this time. Most pacemaker experts recommend that rate modulation be programmed to “off” in the perioperative period to prevent confusion between intrinsic tachycardia and pacemaker-induced tachycardia (e.g., see Schwartzburg and associates\(^{121}\)).

**BOX 48-2  Pacemaker Indications**

| Symptomatic diseases of impulse formation (sinus node disease) |
| Symptomatic diseases of impulse conduction (atrioventricular nodal disease) |
| Long QT syndrome |
| Hypertrophic obstructive cardiomyopathy†* |
| Dilated cardiomyopathy †* |

*Indications for permanent pacing are shown. Most patients with pacemakers currently fall into the first two categories (sinus node or atrioventricular nodal disease).

†Requires 100% ventricular pacing to be effective. Thus, short atrioventricular delays (\(\sim120-150\) msec) are programmed.

Pacing for HOCM and DCM requires careful attention to pacer programming. To be effective in these patients, the PM must provide the stimulus for ventricular depolarization, and AV synchrony must be preserved.\(^{36}\) PM inhibition or loss of pacing (i.e., from native conduction, atrial irregularity, ventricular irregularity, development of junctional rhythm, or EMI) can lead to deteriorating hemodynamics in these patients.
CRT pacing may cause inappropriate lengthening of the QT interval in susceptible patients, and this lengthening has been reported to be associated with torsades de pointes. As a result of this report, the prudent anesthesiologist should ensure adequate access to rapid defibrillation for patients with BiV pacing accomplished with a conventional PM.

MAGNETS

Despite often repeated folklore, most PM manufacturers warn that magnets were never intended to treat PM emergencies or prevent the effects of EMI. Rather, magnet-activated switches were incorporated to produce pacing behavior that demonstrates remaining battery life and, sometimes, pacing threshold safety factors.

Placement of a magnet over a generator may produce no change in pacing because not all PMs switch to a continuous asynchronous pacing mode when a magnet is placed. Moreover, not all models from a given company behave the same way. Possible effects of magnet placement are shown in Table 48-3. In some devices (Biotronik [Berlin, world headquarters; Lake Oswego, Ore, U.S. headquarters], BOS, Pacesetter, St. Jude Medical, Telectronics), magnet behavior can be altered or completely eliminated by programming. For generators with programmable magnet behavior, only an interrogation with a programmer can reveal current settings.

For all generators, calling the manufacturer remains the most reliable method for determining magnet response and for using this response to predict remaining battery life (telephone numbers for the device manufacturers are shown in Appendix 48-1). As battery voltage falls, the magnet response can be used to detect the following:

- Intensified follow-up interval (IFI) or elective replacement near (ERN): The device must be checked frequently (approximately every 4 weeks for most models).
- Elective replacement indicator (ERI) or elective replacement time (ERT): The device is nearing the end of its useful life and should be electively replaced.
- End of life (EOL): The device has insufficient battery power remaining and should be replaced immediately.

On application of a magnet, some devices perform a threshold margin test (TMT). In this test, one of more of the PM pulses is reduced in amplitude, pulse width, or both, in an attempt to gauge the safety margin for pacing voltage. Loss of capture on these TMT pulses indicates an inadequate safety margin for pacing (Fig. 48-5). Some devices from St. Jude Medical (formerly Pacesetter) with the Siemens “Vario” feature reduce the ventricular pacing energy over 16 cycles to demonstrate the pacing threshold. As a result, many pacing cycles can take place at insufficient energy for ventricular capture, and this can produce periods of asystole while the magnet is applied.

Occasionally, PM-mediated tachycardia (PMT) can ensue on removal of the magnet from a dual-chamber PM (Fig. 48-6). These cases of PMT result from P waves conducted in retrograde fashion during asynchronous ventricular pacing, most commonly when the magnet rate is lower than the patient’s intrinsic rate. PMT from retrograde AV nodal conduction also can occur in any DDD or VDD pacing device following EMI, premature ventricular contraction, or a noncaptured atrial pace. When present, retrograde P waves can be “tracked,” typically resulting in ventricular pacing at the UTR. Each paced ventricular cycle results in another retrograde P wave, thereby producing tachycardia. Should this behavior be observed in a conventional PM, it can be treated by application, then removal, of the magnet provided the PM has asynchronous pacing with magnet application. Magnet application does not terminate PMT in an ICD. Most CIED devices can be programmed to recognize these cases of PMT and periodically omit one ventricular pacing pulse, or lengthen one AV delay cycle, during UTR pacing.

In addition, as noted previously, the patient with a dual-chamber device that has detected a high atrial rate and “mode-switched” to prevent UTR pacing could have the mode switch reset on application and removal of the magnet. These patients then undergo UTR pacing until criteria are met to return to the mode-switch mode. Distinguishing PMT caused by retrograde P waves from UTR pacing resulting from high atrial rates (before mode-switch entry) can be very difficult. In general, however, mode-switch secondary to rapid atrial rates takes place within 10 to 15 seconds, and PMT from retrograde P waves is quite persistent.

PREANESTHETIC EVALUATION AND PACEMAKER REPORgramMING

Preanesthetic management of the patient with a PM includes evaluation and optimization of coexisting disease (see also Chapter 39). American College of Cardiology (ACC) guidelines suggest that cardiac testing (stress tests, echocardiograms) should be dictated by the patient’s underlying diseases, medications, symptoms, interval from the last testing, and planned intervention. No special laboratory tests or radiographs are needed for the patient with a conventional PM. Chest radiographs rarely depict lead problems, and a standard chest radiograph may exclude the generator or its markings. Furthermore, most radiologists are unfamiliar with pacing issues. However, a patient with a CRT device may need a chest radiograph to document the position of the CS lead, especially if central line placement is planned. CS leads have no fixation and may be more easily dislodged than a standard PM or ICD lead. Spontaneous LV dislodgment likely occurs in at least 4.7% of patients, at a rate of 2.3% annually.

Important features of the preanesthetic device evaluation are shown in Appendix 48-2. The ASA advisory recommends a recent PM interrogation; the other 3 advisories recommend contact with the patient’s CIED physician and clinic. However, in abstract form, Rozner and associates reported more than 30% noncompliance with in-person follow-up guidelines in 161 consecutive preoperative patients, as well as the need to replace 5% of PMs preoperatively for battery depletion. Remote evaluation provides some assurance of battery longevity, but it does not provide rigorous evaluation of the pacemaking system.
### TABLE 48-3 PACEMAKER MAGNET BEHAVIOR

<table>
<thead>
<tr>
<th>Pacemaker Company</th>
<th>Magnet Mode Designation</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biotronik (except INOS and DROMOS)</td>
<td>AUTO</td>
<td>In normal operation, 10 asynchronous events at 90 beats/min, then returns to original programmed mode, without rate responsiveness. Pacing is at lowest available rate (LRL, sleep rate, or hysteresis rate). If battery at ERI, 10 asynchronous events at 80 beats/min in VOO mode, then either VDD (dual-chamber) or VVI (single-chamber) pacing at 11% lower than lowest available rate. For any dual-chamber mode (DDD, DDI, or VDD), the AV delay shortens to 100 msec while the magnet is in place.</td>
</tr>
<tr>
<td>ASYNCH</td>
<td>Asynchronous pacing at 90 beats/min in normal operation. At ERI, 80 beats/min (single-step change) in VOO mode regardless of original programming. For any dual-chamber mode (DDD, DDI, or VDD), the AV delay shortens to 100 msec while the magnet is in place.</td>
<td></td>
</tr>
<tr>
<td>SYNCH</td>
<td>In normal operation, pacing in original programmed mode, without rate responsiveness. Pacing is at lowest available rate (LRL, sleep rate, or hysteresis rate). If battery at ERI, then either VDD (dual-chamber) or VVI (single-chamber) pacing at 11% lower than lowest available rate. For any dual-chamber mode (DDD, DDI, or VDD), the AV delay shortens to 100 msec while the magnet is in place.</td>
<td></td>
</tr>
<tr>
<td>Boston Scientific/Guidant Medical/CPI (Cardiac Pacemakers, Inc.)</td>
<td>ASYNCH</td>
<td>Asynchronous pacing at 100 beats/min in normal operation, 85 beats/min at ERT (single-step change). Insignia and all &quot;BOS&quot; label devices have an intermediate step (90 beats/min) at ERN. For Triumph and Prelude models, see Medtronic pacemakers, below. Insignia and all &quot;BOS&quot; label devices emit a reduced 3rd pacing pulse (on ventricular lead for dual chamber devices) to demonstrate adequacy of pacing output.</td>
</tr>
<tr>
<td>SO</td>
<td>OFF</td>
<td>No change, magnet is ignored. OFF is the magnet mode after a &quot;power on reset,&quot; which can occur secondary to EMI.</td>
</tr>
<tr>
<td>Medical (not including Sono Medical)</td>
<td>EGM mode</td>
<td>No change in pacing. Magnet application initiates data collection.</td>
</tr>
<tr>
<td>Sorin (formerly ELA Medical)</td>
<td>Asynchronous pacing at 96 beats/min gradually declining to 80 beats/min at ERI. Sorin pacemakers take 8 additional asynchronous pacing cycles (the final 2 cycles are at LRL with long atrioventricular delay) on magnet removal.</td>
<td></td>
</tr>
<tr>
<td>Medtronic</td>
<td>Asynchronous pacing at 85 beats/min in normal operation, 65 SSI regardless of original programming if ERI (single-step change). Most Medtronic pacemakers emit one or more ventricular pulses during the first 3–7 asynchronous events (which may be at a rate of 100 beats/min) at a reduced pulse width or voltage to demonstrate adequacy of ventricular pacing output. A Medtronic dual-chamber pacemaker will revert to single-chamber, ventricular-only behavior on detection of ERI regardless of prior programming. Such behavior could result in hemodynamic embarrassment in a patient with sinus node disease and a bad ventricular lead.</td>
<td></td>
</tr>
<tr>
<td>St. Jude Medical (not including Teletronics)</td>
<td>Battery Test</td>
<td>Asynchronous pacing at 98.6 (100 for Accent/Anthem series) beats/min gradually decreasing to 86.3 or less beats/min at ERI.</td>
</tr>
<tr>
<td>&quot;SJM&quot; x-ray logo</td>
<td>OFF</td>
<td>No magnet response.</td>
</tr>
<tr>
<td>Event snapshots</td>
<td>Event snapshots</td>
<td>No change in pacing. Magnet application causes pacemaker to collect data. Insignia and all &quot;BOS&quot; models lack this feature.</td>
</tr>
<tr>
<td>Battery Test + Battery Test</td>
<td>No magnet response.</td>
<td></td>
</tr>
<tr>
<td>St. Jude Medical (not including Teletronics)</td>
<td>Battery Test</td>
<td>Asynchronous pacing and the rate depends on specific model. In general, a pacing rate of less than 90 beats/min should prompt further evaluation.</td>
</tr>
<tr>
<td>Pacesetter x-ray logo (ϕ)</td>
<td>OFF</td>
<td>VARIO results in a series of 32 asynchronous pacing events. The rate of the first 16 paces reflects battery voltage, gradually declining from 100 to 85 beats/ min at ERI. The next 15 paces are used to document ventricular pacing capture safety margin. The rate will be 119 beats/min with gradually declining pacing voltage. The sixteenth pace of this group is at no output. The next pace restarts the 32-event sequence. The 32-event sequence repeats as long as the magnet remains in place.</td>
</tr>
</tbody>
</table>

AV, Atrioventricular; EGM, electrogram; EMI, electromagnetic interference; the device should be replaced immediately; ERI, elective replacement indicator; the device should be replaced promptly; the FDA requires pacemakers to perform safely for at least 3 months from onset of ERI; ERT, elective replacement time (same as ERI); for Boston Scientific/Guidant/ CPI, at ERT rate-responsive programming is cancelled; at 3 months after ERT, only single-chamber operation continues; the device needs monthly battery checks; LRL, lower rate limit; the programmed lower rate, or set point, of the pacemaker; SSI, single-chamber, inhibited mode; if implanted for ventricular pacing, SSI = VVI; if implanted for atrial pacing, SSI = AAI.

*The effects of appropriately placing a magnet over a pacemaker are shown. Column 1 shows the pacemaker manufacturer. If the magnet response is programmable, then column 2 shows the various programmable modes available. The first mode shown for a company is the default mode. Column 3 shows the effect on pacing therapy for the magnet mode shown in column 2. Unless otherwise specified, asynchronous pacing takes place, without rate responsiveness, in the chambers originally programmed. Thus, a dual-chamber program would result in DOO pacing, and a single-chamber program would result in VOO (ventricular) or AOO (atrial) pacing, and a biventricular, dual-chamber device would be DOOOV.
Figure 48-5. Threshold margin test (TMT) demonstrating an inadequate safety margin for pacing. Application of a magnet to some pacemakers produces asynchronous pacing in which one or more pacing stimuli are emitted with reduced ventricular pacing voltage, pulse width, or both. This sequence is used to determine, without formal pacemaker interrogation, the adequacy of pacing energy settings. In this electrocardiographic strip from an Intermedics device, the patient was being paced in the VVI mode at a rate of 70 beats/minute (equal to 857-msec intervals). On application of the magnet, this pacemaker produced four intervals (five pacing stimuli) of asynchronous pacing at a rate of 90 beats/minute (667-msec intervals), thus demonstrating adequate battery voltage for this device (pacemaker response to magnet placement depends on the manufacturer and sometimes the programming). At the fifth pacing stimulus after magnet application, the pacemaker performed a TMT by reducing the stimulus pulse width to 50% of the programmed value (equal to 50% of programmed energy). Failure of this stimulus to produce ventricular systole (i.e., failure to capture) demonstrates a dangerously low safety margin for ventricular pacing because pacing pulse width should be at least three and generally four times the threshold for capture. After these five initial stimuli, Intermedics pacemakers then pace asynchronously at the programmed lower rate (70 in this case) for 60 additional cycles. On completion of these 64 cycles (65 stimuli), Intermedics pacemakers return to programmed values and ignore the magnet.

Figure 48-6. Pacemaker-mediated tachycardia (PMT) after removal of the magnet from a pacemaker. This patient had a dual-chamber pacemaker implanted for atrioventricular (AV) nodal disease, and she was pacemaker dependent for ventricular activity. She had a sinus rate of 75 beats/minute before application of the magnet with appropriate ventricular pacing (strip not shown). Her programmed AV delay was 200 msec. With magnet application, her pacemaker produced asynchronous AV sequential pacing (DOO mode) at a rate of 60 beats/minute. This strip is from an electrocardiographic recorder that enhances the pacemaker artifact indicated by black, downward arrows. Because the asynchronous “magnet” rate of this device was lower than her intrinsic atrial rate, many of the atrial pacing stimuli were applied during an atrial refractory period (called functional noncapture). A consequence of atrial noncapture can be retrograde AV nodal conduction with depolarization of the atria after depolarization of the ventricle or ventricles. The retrograde P waves are shown with the red upward arrows. While the magnet is applied, this retrograde depolarization of the atria is ignored. Shortly after the magnet was removed (shown), a paced ventricular event occurred, followed by retrograde AV nodal conduction. With the ensuing depolarization of the atria from this retrograde conduction, the pacemaker sensed an atrial event and responded by pacing the ventricle 200 msec later. Yet another retrograde P wave appears, and each ventricular pace in response to a retrograde P wave created yet another ventricular pace. The result is PMT at the upper tracking rate (programmed here to 130 beats/minute) of the pacemaker. PMT from retrograde AV nodal conduction can occur in any DDD or VDD device with magnet removal, a premature ventricular contraction, or a noncaptured atrial pace. Treatment of this PMT should be reapplication of the magnet. Some pacemakers can be programmed to eventually delay one AV cycle when pacing at the upper tracking limit to “break” PMT. Magnet application is ineffective in PMT in a patient with an ICD.
For PMs, interrogation with a programmer remains the most reliable method for evaluating battery voltage, battery impedance, lead performance, and adequacy of current settings. Special attention should be paid to patients from countries where CIEDs may be reused, because of possibly increased malfunction rates. For any PM with a battery voltage less than 2.6 volts or battery impedance (where available) greater than 3000 ohms, the company should be consulted about the possible need for elective replacement of the device preoperatively.

Appropriate reprogramming is the safest way to avoid intraoperative problems, especially for pacing-dependent patients in whom EMI (most commonly monopolar “Bovie” electrosurgery) will be present (Box 48-3). Assistance may be available from the PM manufacturers (see Appendix 48-1 for company telephone numbers). However, interrogation of any generator with or without reprogramming requires a medical prescription and supervision by an appropriately trained physician. Hospital guidelines and policy should reflect the need for a competent physician to review and validate any care delivered by a company representative.

Reprogramming a PM to asynchronous pacing at a rate greater than the patient’s underlying rate usually ensures that no oversensing during EMI will take place, thus protecting the patient. However, in one case report, a Teletronics device opened its output circuitry (i.e., no pacing stimuli were delivered) in the presence of high-voltage EMI. Furthermore, at our institution, two PMs in the DOO mode dropped their rate during use of monopolar coagulation ESU, presumably because of high battery current drain.

Reprogramming a device will not protect the device from internal damage or reset caused by EMI. Additionally, setting a device to asynchronous mode causes the PM to ignore premature atrial or ventricular systoles, which could lead to R-on-T pacing and malignant rhythm, especially in the patient with significant structural compromise of the myocardium.

In general, rate responsiveness and other “enhancements” (e.g., atrial fibrillation suppression, hysteresis, managed ventricular pacing, sleep rate, AV search) should be disabled by programming to prevent misinterpretation of the cardiac rhythm. Older reports of pacing threshold changes during both intrathoracic and nonchest surgical procedures, as well as significant noncardiac acute disease, drive the issue of postoperative pacing threshold testing in nonautomated devices.

Special attention must be given to any device with a minute ventilation (bioimpedance) sensor because inappropriately configured minute ventilation sensors frequently respond to electromagnetic interference (EMI) with pacing at the upper sensor rate. These tachycardias also can be produced by connection to an operating room electrocardiographic monitor because many of these devices inject electrical signals through the electrocardiographic leads to determine respiratory rate or lead disconnection. In 1998, the U.S. Center for Devices and Radiologic Health issued a safety alert calling for the deactivation of any minute ventilation (bioimpedance) sensor before exposing a patient to a potential source of EMI or connection to medical devices.

**INTRAOPERATIVE (OR PROCEDURE) MANAGEMENT OF PACEMAKERS**

Although no special monitoring or anesthetic technique is required for the patient with a cardiac generator, attention must be given to several concerns (see also Chapters 45 and 67).
First, ECG monitoring of the patient should include the ability to detect pacing discharges. Currently, most ECG monitors in both the operating room and the intensive care unit perform digital acquisition and analysis of ECG signals, and they are subject to considerable interference from a variety of sources. In their default settings, these monitors often filter high-frequency signals, thus resulting in the exclusion of pacing artifacts. Disabling this filtering causes the monitor to “paint” pacing spikes onto the display. Even with the filtering disabled, however, pacing artifacts do not always appear because modern leads may accomplish pacing with very low-amplitude pulses. Moreover, given that many digital monitors analyze only one lead for these signals (and then “paint” on artifacts on every lead), placement of the ECG leads can markedly affect the detection axis.

In practice, when a patient with known pacing is monitored, sometimes changing the “analysis” lead on the monitor results in the appearance of pacing signals. Unfortunately, when the high-frequency filter is disabled, EMI, especially from the use of the monopolar ESU, can lead to inappropriate “painting” of pacing artifacts on the monitor (Fig. 48-7).

Second, monitoring of the patient must include the ability to ensure that paced electrical activity is converted to mechanical systoles. Mechanical systoles are best evaluated by pulse oximetry plethysmography or arterial pressure waveform.

Third, published perioperative experience with CRT pacing remains limited. These patients often have ejection fractions less than 30%, and they depend on pacing in both ventricles to improve their cardiac output. Loss of ventricular pacing from any cause (e.g., AV dyssynchrony [atrial fibrillation, atrial flutter, appearance of junctional rhythm], myocardial ischemia, acid-base disturbance, change in pacing threshold, ESU interference) can cause an immediate decrease in cardiac output. With the exception of transesophageal echocardiography, no beat-to-beat monitoring of cardiac output has demonstrated any utility in detecting loss of LV capture. Patients with HOCM pacing can depend on ventricular pacing to limit LV outflow tract obstruction.

---

1 Most monitors manufactured since 2005 have much improved capability to detect and display pacing artifacts, although they still filter these high-frequency signals in their default setup mode.

---

**Figure 48-7.** Disabling the pacemaker artifact filter on a digitally processed electrocardiographic (ECG) monitor results in the “painting” of environmental noise (electromagnetic interference [EMI]) as pacemaker artifacts. **A,** Effects of “cut” monopolar electrosurgical unit (ESU) application. Because the patient’s underlying rate exceeded the pacemaker’s programmed lower rate limit, no pacing took place. However, activation of the ESU in the “cut” mode produced sufficient electromagnetic noise that the monitor began “painting” pacemaker artifacts at a rate of approximately 20 Hz. The top tracing is the ECG lead II, the middle tracing is ECG lead V5, and the bottom tracing is the invasive arterial pressure waveform. **B,** The effects of “Coagulation” ESU application produced ventricular oversensing with pacemaker inhibition and left this patient with compromised cardiac output. Evidence also shows inappropriate monitor “painting” of pacemaker artifacts from the EMI. The top tracing is ECG lead II, the middle tracing is the pulse oximeter plethysmogram, and the bottom tracing is the invasive arterial pressure waveform. (From Rozner MA: Review of electrical interference in implanted cardiac devices, Pacing Clin Electrophysiol 26:923-925, 2003.)
Fourth, some patients may need an increased pacing rate during the perioperative period to meet an increased oxygen demand. This subject is often not addressed. PM-treated patients reportedly have high postoperative morbidity and mortality, and failure to address tissue oxygen demands and cardiac output needs could contribute to this problem.

Fifth, appropriate equipment must be on hand to provide backup pacing and defibrillation if needed. Cardiac generators, although rarely, occasionally perform some untoward maneuver or fail, even in the absence of EMI. Acceptable but inappropriate behavior of a PM or ICD can create an inhospitable situation. Even a properly working, dual-chamber PM can produce R-on-T pacing and ventricular tachycardia (VT), especially in the setting of a junctional rhythm or PVCs (Fig. 48-8).

The medical team caring for the patient with an implanted cardiac PG must understand that the patient has been deemed needy of this device by a physician who is an expert in the diagnosis and management of cardiac rhythm issues. Few anesthesiologists are qualified to contradict this diagnosis, yet some persist in providing anesthesia without appropriate backup pacing and defibrillation equipment on hand.

Monopolar “Bovie” ESU use remains the principal intraoperative issue for the patient with a PM. Between 1984 and 1997, the FDA was notified of 456 adverse events with PGs, 255 from electrosurgery or external defibrillator, and a “significant number” of device failures. Monopolar ESU is more likely to cause problems than bipolar ESU, and PMs with unipolar electrode configurations are more sensitive to EMI than are PMs with bipolar configurations. The most common effect of ESU on pacing function is ventricular oversensing, which causes pacing inhibition (see Fig. 48-7, B). Sometimes, the generator detects significant EMI and begins pacing asynchronously at the programmed lower rate. This behavior is called noise or reversion mode pacing, even though the PM does not actually change modes. Noise reversion is not present in some ICDs and is programmable in others.

Magnet placement during electrosurgery may prevent aberrant PM behavior. Newer generators are believed to be relatively immune to spurious reprogramming from EMI, although they can undergo a reset condition that will likely alter the programmed settings.

If monopolar ESU is to be used, then the electrosurgical dispersive electrode (often misidentified as the “grounding pad”) must be placed to ensure that the ESU current path does not cross the pacemaking system. Some authors recommend placement of this electrode on the shoulder for head and neck procedures or the distal arm (with sterile draping of the wire) for breast and axillary procedures. Procedures using only monopolar ESU or with special EMI pacing ramifications include the following:

**Electroconvulsive Therapy:** This treatment may require nonsensing (asynchronous) mode to prevent myopotential-induced oversensing with resultant PM inhibition.

**Lithotripsy:** The cardiac generator must be excluded from the lithotripter field. If the lithotripter triggers its output on the sensed ECG “R” wave, atrial pacing should be disabled to prevent the lithotripter from inappropriately firing on the atrial pacing artifact (see Chapter 72).

**Magnetic resonance imaging (MRI):** MRI requires special expertise, including the ability to immediately reprogram a CIED. The literature reports that MRI exposure can lead to magnet switch closure (magnet mode activation), CIED reprogramming, inappropriate high-rate pacing, generator damage, myocardial injury, lead failure, or arrhythmia. Except for CIED resets to safety parameters, no significant complications have been reported, even in large series. On February 8, 2011, the FDA approved a Medtronic PM with MRI-conditional labeling; conditions include MRI energy limitations, a requirement for ECG and pulse oximeter plethysmography monitoring, completion of cardiac monitoring and radiology training, and peri-MRI scan reprogramming. Medtronic has introduced a “second-generation” MRI-conditional PM into the U.S. market with similar conditions but with automated testing and reporting features. Only PMs from Medtronic have MRI labeling in the United States at this time, but Biotronik (ICD and PM), BOS (PM), and St. Jude Medical (PM) have received MRI labeling in other countries.

**Nerve stimulator testing or therapy:** Nerve stimulators can cause PM oversensing, with resultant PM inhibition and cardiac arrest. Nerve stimulators have been used intraoperatively to inhibit undesired cardiac pacing or arrest the cardiac rhythm for endovascular stent placement. In patients with an ICD, inappropriate detection of neuromuscular stimulators, transcutaneous electrical nerve stimulation, and chiropractic electrical muscle stimulation as VT or VF has been reported. Nerve stimulators can also interfere with ECG monitoring of PM artifacts. As with ESU current, the electrical path for nerve stimulator current should not cross the generator system or the chest (see also Chapter 53).

**Radiofrequency ablation:** Noncardiac radiofrequency ablation, especially when kept inferior to the umbilicus, behaves primarily like unblended “cut” monopolar ESU. Although problems seem to be rare, the HRS recommends postoperative interrogation before discharge from cardiac monitoring.

**Succinylcholine or etomidate use:** Reports of myopotential problems on administration of succinylcholine or etomidate have been described (see also Chapters 30 and 34). In the succinylcholine case, muscle fasciculations may have caused ventricular oversensing with resultant PM inhibition, but the case is difficult to interpret. No ECG tracings were published, and the PM was damaged during subsequent defibrillation. Using programmers, I have witnessed succinylcholine administration to more than 50 patients with PMs or defibrillators without finding any myopotential oversensing. The etomidate issue is also questionable because the particular PM had other issues. However, myopotential oversensing (in the absence of succinylcholine) has been observed to interfere with PM function or cause inappropriate ICD therapy (see also Chapter 34).

**Transurethral resection and uterine hysteroscopy:** Transurethral resection of the bladder or prostate and hysteroscopic procedures generally use monopolar ESU, and device reprogramming may be needed to
Normal dual-chamber pacemaker timing can produce R-on-T pacing. A, This strip demonstrates functional ventricular undersensing of a premature ventricular contraction (PVC) with resultant R-on-T pace leading to torsades de pointes in a normally functioning, appropriately programmed pacemaker (PM). This patient had a dual-chamber PM in the DDD mode with a programmed lower rate of 70 beats/minute (R-R interval of 857 msec) and atrioventricular (AV) delay of 200 msec. With these parameters, the pacemaker will pace the atrium 657 msec after any previous ventricular event. Atrial pacing is labeled A, and ventricular pacing is labeled V. The top tracing is electrocardiographic (ECG) lead II, the middle tracing is ECG lead V5, and the bottom tracing is the invasive arterial blood pressure waveform. Approximately 660 msec after the first QRS complex (1) on the strip (which was adequately sensed by the pacemaker), an atrial stimulus was emitted. At 200 msec after this atrial pace, a ventricular stimulus was emitted and appeared to depolarize the ventricle (2). Approximately 660 msec later (3), the patient experienced a PVC. Because the pacemaker was preparing to emit the atrial stimulus, it had disabled its ventricular sensing element (called post atrial ventricular blanking, see also Figure 48-13) and failed to sense this PVC (termed functional undersensing). At 200 msec after the atrial stimulus, no ventricular event had been sensed, so the pacemaker emitted a ventricular stimulus on the T wave. Because the ventricle was in a refractory period from the PVC, no depolarization of the ventricle occurred (called functional noncapture). At 660 msec from this attempted V-pace, the pacemaker again paces the atrium (4), and it appears that the next V-pace captures the ventricle. At point 5, a repeat of the events at point 3 occurs (i.e., the pacemaker disabled its sensing elements in preparation to pace the atrium and again failed to detect the PVC). This time, however, the V-pace on the T wave produced torsades de pointes. B, This strip was obtained from a Medtronic programmer during interrogation of a Kappa 700 dual-chamber pacemaker. The top tracing is ECG lead II, and the bottom tracing is the “marker channel,” which shows the pacemaker’s interpretation of events. This pacemaker was programmed to the DDD mode with a lower rate of 60 beats/minute. The AV delay was 200 msec. As a result, after any ventricular event, the pacemaker will emit an atrial pulse at 800 msec if no intervening atrial or ventricular event takes place. This patient had junctional rhythm at 75 beats/minute (corresponding to an R-R interval of 800 msec), so the pacemaker emitted an atrial pulse just as the junctional event occurred. Because the pacemaker disables its ventricular sensing element when emitting the atrial pulse, it failed to detect the ventricular event and emitted the ventricular pulse 200 msec later, on the T wave. This inappropriate pacing takes place every other cycle because every other junctional event is sensed approximately 600 msec after the previous ventricular pace. Decreasing the AV delay diminishes the likelihood of pacing during the vulnerable period of the ventricle. AP is an atrial pace, VP is a ventricular pace, and VS is a ventricular sensed event. The third complex deserves comment. The pacemaker sensed this ventricular event as it re-enabled its sensing element, and the pacemaker cannot tell whether the sensed event is a true ventricular depolarization or an “echo” of the atrial pace. When a signal from the ventricle is sensed within 30 to 90 msec after an atrial pulse, many CIEDs immediately emit a ventricular pacing stimulus. Called a ventricular safety pace, this pacing stimulus is designed to protect the patient from inappropriate sensing of the atrial signal by the ventricular channel, which would then inhibit the ventricular output. The safety pace is emitted at 110 msec to prevent R-on-T pacing. This feature is also called nonphysiologic AV delay by some manufacturers. R-on-T pacing can be appropriate (but not ideal) behavior of a DDD or DDI pacemaker in the setting of PVCs, as well as a junctional rhythm. It can also be seen with atrial or ventricular undersensing.
Prevent PM oversensing with resultant PM inhibition. A resectoscope that includes the current-return electrode on the shaft (the irrigation medium is normal saline) could limit EMI to the generator but has not been carefully tested in this environment.

**PACEMAKER FAILURE**

PM failure has three causes: (1) generator failure, (2) lead failure, or (3) failure of capture. Generator failure is rare in a device that has been previously evaluated and not near the end of its useful battery life, unless the generator (or leads) is struck directly by the ESU. Lead failure, also unusual but reported during patient repositioning, can result in undersensing (intrinsic activity is not detected), oversensing (“detection” of events unrelated to intrinsic activity), or failure to deliver sufficient energy to the myocardium to produce a depolarization (loss of capture). Myocardial changes that lengthen the refractory period or increase the energy requirement for depolarization (failure to capture) can result from myocardial ischemia or infarction, acid-base disturbance, electrolyte abnormalities, or abnormal antiarrhythmic drug levels.

The response to PM failure depends on the clinical situation. A patient with a perfusing rhythm and stable vital signs can be observed while a plan is made to correct the problem. For a patient with inadequate perfusion, the following steps can be tried (while cardiopulmonary resuscitation is in progress when appropriate):

1. A magnet can be applied if the PM is known to revert to an asynchronous mode, which will also eliminate sensing behavior in these devices. Magnet application may add to the hemodynamic embarrassment in the setting of inadequate pacing safety margins or the onset of automated TMT.

2. Temporary pacing can be initiated, and it can be transthoracic (transcutaneous), transvenous, or transesophageal. Transesophageal (atrial) pacing requires a functional atrium and AV node for ventricular activation, so it is contraindicated in patients with atrial fibrillation or flutter. It is also generally contraindicated in the presence of a permanent PM (or ICD). In the setting of external pacing, the ECG can be misinterpreted because the PM artifacts are large compared with the QRS complexes (Fig. 48-9, A). Successful ventricular pacing has been reported with transesophageal PMSs, but it remains unreliable, and it produces inferior hemodynamics compared with atrial pacing (Fig. 48-9, B). Any external pacing may further inhibit PM output at energies that do not produce myocardial capture.

3. Sympathomimetic drugs can be administered to decrease depolarization threshold or increase heart rate, or both. Epinephrine (0.5 to 1 μg/minute) or dopamine (5 to 20 μg/kg/minute) should be considered. Isoproterenol (0.5 to 10 μg/minute) is often recommended, but this drug is not widely available, and its use may produce hypotension. Atropine may be helpful.

4. Causes of myocardial ischemia should be sought and corrected. Myocardial ischemia can substantially increase the energy required for ventricular capture.

5. Disturbances of electrolyte balance, antiarrhythmic drug levels, and acid-base equilibrium should be investigated and corrected. Potassium, calcium, and magnesium abnormalities, as well as amiodarone, can raise depolarization thresholds. Moreover, potassium flux, ionized calcium level, and acid-base equilibrium can be affected by hyperventilation and hypoventilation.

6. If none of the foregoing measures succeed, consider a PM that was reprogrammed for the perioperative period should be reset appropriately. For nonreprogrammed devices, the performance and timing of a postoperative interrogation will depend on the nature of the case and the CIED team caring for the patient. Certainly, interrogation is warranted if any issues appear. Consideration should be given to increasing the lower paced rate in a chronotropically incompetent patient if increased cardiac output will benefit the patient during convalescence.

**POSTANESTHESIA PACEMAKER EVALUATION**

A PM that was reprogrammed for the perioperative period should be reset appropriately. For nonreprogrammed devices, the performance and timing of a postoperative interrogation will depend on the nature of the case and the CIED team caring for the patient. Certainly, interrogation is warranted if any issues appear. Consideration should be given to increasing the lower paced rate in a chronotropically incompetent patient if increased cardiac output will benefit the patient during convalescence.

**IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS**

The development of an implantable, battery-powered device able to deliver sufficient energy to terminate VT or ventricular fibrillation (VF) represents a major medical breakthrough for patients with a history of ventricular tachydysrhythmias. These devices reduce deaths in the setting of malignant ventricular tachydysrhythmias, and they have been long considered superior to antiarrhythmic drug therapy. Initially approved by the FDA in 1985, more than 120,000 devices will be implanted in the United States this year, and industry sources report that more than 300,000 patients have these devices today. Furthermore, results from the Sudden Cardiac Death–Heart Failure Trial (SCD-HeFT, which showed that prophylactic placement of an ICD is superior to drug therapy in any patient with an ejection fraction <0.35 and without evidence of arrhythmic inducibility) led to a significant increase in the number of patients for whom ICD therapy is indicated.

Numerous technologic advances have been applied since the first ICD was placed, including substantial miniaturization (pectoral pocket placement with transvenous leads is the norm), as well as battery improvements that now permit permanent pacing with these devices. Thus, one could easily confuse a pectoral T-ICD with a PM.

Like PMs, ICDs have a four-place generic code (NBD) to indicate lead placement and function, as shown in Table 48-4. The most robust form of identification, the label form, expands the fourth character into its component generic PM code (NBG).

---

5. The NBD code is a joint project from NASPE (the “N”) and BPEG (the “B”). The “D” stands for defibrillator.
The Cameron Health–BOS S-ICD system consists of a PG implanted along the lateral chest wall with a single lead tunneled subcutaneously to the area superficial to the heart (Fig. 48-10). The principal advantage of the S-ICD implant is the ability to implant the device without gaining central venous access. The principal disadvantages include the inability to use the S-ICD to provide bradycardia support, the lack of ATP features, higher energy defibrillation thresholds (DFTs), larger size, and shorter battery life than a single-chamber T-ICD. Approximately 11.8% of all ICD patients have experienced an inappropriate shock.100

**Figure 48-9.** Issues with a transesophageal atrial pacemaker (TAP). A, Improper placement of a TAP demonstrating atrial noncapture. The top recording is electrocardiographic (ECG) lead II, the middle recording is ECG lead III, and the bottom recording is the invasive arterial pressure waveform. Sinus bradycardia with evidence of tissue underperfusion developed in this 72-year-old man. A TAP (CardioCommand, Tampa, Fla.) was placed, and the large ECG artifacts at 75 beats/minute were misinterpreted by personnel and by the ECG monitor as ventricular systoles (capture). This strip shows an underlying sinus rate of 50 beats/minute with a first-degree atrioventricular block (PR interval of 280 msec). The patient’s native atrial (P) and ventricular (R) depolarizations are noted. The arterial pressure waveform confirms pacing noncapture. B, Direct ventricular activation with a transesophageal pacemaker. The top recording is ECG lead II, and the bottom recording (enhanced vertical scale) is the invasive arterial pressure waveform. This strip is from an obese 61-year-old woman with diabetes and hypertension who was undergoing transsphenoidal hypophysectomy. Shortly after induction of anesthesia, sinus bradycardia at 37 beats/minute and mild hypotension developed. A transesophageal pacemaker was placed but was advanced too far into the esophagus. This strip was recorded during “pullback.” The ventricle was directly activated in the first four events, with a resultant wide-complex QRS and average blood pressure of 135/75 mm Hg. On pullback, the atrium was activated, the QRS returned to a narrow complex, and blood pressure increased to 143/80 mm Hg. Except for the bradycardia, this patient had a normal heart and did not have a permanently placed generator.
Table 48-4. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group Generic Defibrillator Code (NBD)

<table>
<thead>
<tr>
<th>Position I</th>
<th>Position II</th>
<th>Position III</th>
<th>Position IV†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock Chambers(s)</td>
<td>Antitachycardia Pacing Chamber(s)</td>
<td>Tachycardia Detection</td>
<td>Antibradycardia Pacing Chamber(s)</td>
</tr>
<tr>
<td>O = None</td>
<td>O = None</td>
<td>E = Electrogram</td>
<td>O = None</td>
</tr>
<tr>
<td>A = Atrium</td>
<td>A = Atrium</td>
<td>H = Hemodynamic</td>
<td>A = Atrium</td>
</tr>
<tr>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
</tr>
<tr>
<td>D = Dual (A+V)</td>
<td>D = Dual (A+V)</td>
<td>D = Dual (A+V)</td>
<td>D = Dual (A+V)</td>
</tr>
</tbody>
</table>

*The NBD code is a joint project from the North American Society of Pacing and Electrophysiology (NASPE) (the "N") and the British Pacing and Electrophysiology Group (BPEG) (the "B"). The "D" stands for defibrillator.
†For robust identification, position IV is expanded into its complete NBG code. For example, a defibrillator programmed to ventricular shock and antitachycardia pacing along with biventricular pacing functionality would be identified as VVE-DDDRV, assuming that the pacing section was programmed DDDRV. Currently, no hemodynamic sensors have been approved for tachycardia detection (position III).

Figure 48-10. The Cameron Health–Boston Scientific subcutaneous implantable cardioverter-defibrillator. A schematic drawing of this system (CE mark 2009; U.S. Food and Drug Administration approval in 2012) is shown. This system consists of a generator implanted along the lateral chest wall with a lead tunneled into position over the heart. (From Hauser RG: The subcutaneous implantable cardioverter-defibrillator: should patients want one? Am Coll Cardiol 61:20-22, 2013.)

All T-ICDs have many programmable features to detect and treat both tachyarrrhythmias and bradyarrhythmias. Essentially, these devices measure each cardiac R-R interval and categorize the rate as normal, too fast (short R-R interval), or too slow (long R-R interval). When the device detects a sufficient number of short R-R intervals within a period of time (all programmable), it begins an antitachycardia event. Depending on arrhythmia characteristics (primarily rate) and device programming, the internal computer chooses ATP (less energy use, better tolerated by patient) or shock. If the ICD chooses ATP, shock may be delayed; each cycle of ATP can delay shock 6 to 15 seconds. If shock is chosen, an internal capacitor is charged. Charging time depends on the desired output, and an additional 6 to 15 seconds may pass during a maximum shock. Charging time is lengthened by lower battery voltage, time from last charge, and lower temperature.

Most ICDs are programmed to “reconfirm” VT or VF after charging to prevent inappropriate shock therapy. All T-ICDs can be programmed to begin ATP immediately on initiating a charge cycle (ATP while charging, which does not delay shock). Typically, T-ICDs deliver 6 to 18 shocks per event. Once a shock is delivered, no further ATP takes place. Despite considerable improvement in detection of ventricular dysrhythmias, more than 10% of T-ICD shocks are for rhythm other than VT or VF, and supraventricular tachycardia (SVT) remains the most common cause of inappropriate shock therapy. Inappropriate ICD therapy, whether shock or ATP, has been shown to injure myocardium, and it appears that any therapy, whether appropriate or inappropriate, shortens a patient’s life expectancy when compared with patients not receiving therapy. The likely results of these reports will be to delay initiation of T-ICD therapy for at least 30 to 60 seconds following onset of arrhythmia.

Programmable features in current ICDs to differentiate VT from SVT include the following:

1. Onset criteria: In general, onset of VT is abrupt, whereas onset of SVT has sequentially shortening R-R intervals.
2. Stability criteria: In general, the R-R interval of VT is relatively constant, whereas the R-R interval of atrial fibrillation with rapid ventricular response is quite variable.
3. QRS width criteria: In general, the QRS complex width in VT is narrow (<110 msec), whereas the QRS width in VT is wide (>120 msec).
4. “Intelligence” in dual-chamber devices attempts to associate atrial activity with ventricular activity.
5. Morphology waveform analysis includes comparison with stored historical templates.

When the R-R interval becomes sufficiently short for VF detection, most ICDs begin a shock sequence. As noted earlier, once the device delivers shock therapy, no further antitachycardia pacing takes place.

The capacitor in an ICD “deforms” during inactivity, thus leading to increased time needed to charge the capacitor. To mitigate the effects of deformation, all ICDs perform nontherapeutic charging of their capacitor (“reforming”) at a programmable periodic interval (usually 1 to 6 months).
Indications for Implantable Cardioverter-Defibrillators

- Ventricular tachycardia
- Ventricular fibrillation
- Brugada syndrome (right bundle branch block, ST-segment elevation in electrocardiographic leads V₁ to V₃)
- Arrhythmogenic right ventricular dysplasia
- Long QT syndrome
- Hypertrophic cardiomyopathy
- Prophylactic use in the patient who has CM without electrophysiologic testing to justify placement: MADIT II (EF <30%, ischemic CM)⁹⁶; MUSTT (EF <40%, ischemic CM with non-sustained ventricular tachycardia)¹⁰¹; SCD-HeFT (EF <35%, and CM of any cause)¹¹

CM, Cardiomyopathy; EF, ejection fraction; MADIT II, Multicenter Automatic Defibrillator Implantation Trial II; MUSTT, Multicenter Unsustained Tachycardia Trial; SCD-HeFT, Sudden Cardiac Death in Heart Failure Trial.

A T-ICD will begin pacing when the R-R interval is too long. Single-chamber T-ICDs implanted into patients with sinus rhythm typically are programmed to VVI pacing mode with a backup rate of 40 to 50 beats/minute. In July 1997, the FDA approved devices with sophisticated single-chamber pacing modes and rate-responsive behavior for patients with T-ICD who need permanent pacing (≈20% of patients with ICDs). Because of the Dual Chamber and VVI Implantable Defibrillator (DAVID) study, which suggested that DDD pacing in a patient without a clear need for dual-chamber pacing decreased survival when compared with single-chamber device placement,¹⁰⁴ many electrophysiologists program long (>250 msec) AV delays in these patients to limit ventricular pacing. As noted earlier, however, long AV delays can lead to R-on-T pacing (see Fig. 48-8, A). In addition, many manufacturers of T-ICDs have designed algorithms to limit RV pacing. Some of these algorithms allow dropped QRS events, which can resemble second-degree Mobitz II block or pacing system malfunction.

Indications

Initially, ICDs were placed for hemodynamically significant VT or VF (Box 48-5). Additional indications associated with sudden cardiac arrest include patients awaiting heart transplantation¹⁰⁵ and patients with long QT syndrome.¹⁰⁶ Brugada syndrome (right bundle branch block, ST-segment elevation in leads V₁ to V₃), and arrhythmogenic RV dysplasia.¹⁰⁷¹⁰⁸ Studies suggest that ICDs can be placed prophylactically (i.e., used for primary prevention) to prevent sudden cardiac arrest in patients with hypertrophic cardiomyopathy,¹⁰⁹ as well as in patients after myocardial infarction with an ejection fraction lower than 30%,⁹⁸ but these devices are of no benefit after coronary artery bypass grafting. Three-chamber T-ICDs have been FDA approved for patients with DCM.

Magnets

As in PMs, magnet behavior in some ICDs can be altered by programming. Most devices suspend tachydysrhythmia detection (and therefore therapy) when a magnet is appropriately placed to activate the magnet switch (Table 48-5). Some devices from BOS, Pacesetter, and St. Jude Medical can be programmed to ignore magnet placement, and some BOS (“GDT” label) ICDs have had their magnet function permanently disabled because of a magnet switch issue (e.g., see Fig. 48-3).¹¹ For BOS devices, if the magnet mode is enabled and the T-ICD is enabled for antitachycardia therapy, the T-ICD will emit beeps synchronized to R waves (older BOS T-ICDs with a CPI or GDT label) or at 1 Hz (most BOS or BSC label T-ICDs) to signify adequate placement of magnet and suspension of tachyarrhythmia detection (thus disabling therapy). If any BOS T-ICD emits a constant tone when a magnet is applied, then antitachycardia therapy will remain disabled, even when the magnet is removed. Many BOS T-ICDs with the GDT label must be auscultated following magnet removal to ensure that tone emission has ceased, thus indicating that the magnet switch has returned to the open position.¹¹

To ensure correct magnet placement on their devices, Medtronic marketed a device called the Smart Magnet. Manufacturing ceased in approximately 2005 because not many of these devices sold. This battery-powered instrument contains a magnet and a radiofrequency (RF) receiver. All Medtronic generators broadcast RF destined for the programmer on magnet placement, and the Smart Magnet detects this RF transmission and illuminates the “found” light when an ICD is under the magnet. Medtronic recommends that the Smart Magnet be taped onto the patient. During EMI (e.g., monopolar ESU), signal transmission from the T-ICD to the Smart Magnet can be interrupted, and the ICD “found” light often turns off. In this setting, however, the T-ICD remains disabled because the physical presence of the magnet, rather than the RF communication, disables the antitachycardia therapy.

No ICD provides asynchronous pacing with magnet placement. T-ICDs from Sorin (Milano, Italy, world headquarters; Arvada, Colo, U.S. headquarters) change their pacing rate (but not mode) to indicate battery status. Interrogating the device represents the most reliable method for determining magnet response. Contact with the CIED clinic or the manufacturer may provide additional information.

Preanesthetic Evaluation and Reprogramming

In addition to evaluating and optimizing any comorbid diseases in the patient with an ICD, the ASA advisory recommends preoperative T-ICD interrogation. The other three statements recommend contact with the CIED clinic and physician. T-ICDs store considerable data on the occurrence of dysrhythmias. Because ATP is well tolerated, most patients remain unaware of this intervention. For any patient scheduled to undergo an elective procedure, the onset of new dysrhythmias likely warrants investigation of the problem before the procedure (Fig. 48-11). HRS/EHRA recommends 3- to 6-month follow-up with either in-office or remote (telephonic at this time) evaluation.⁹ As with PMs, remote follow-up does not allow iterative testing of sensing or pacing thresholds. Medicare has no coverage determination for ICD follow-up.
Determination of the need for elective replacement of an ICD resulting from battery depletion is more complicated than with PMs. Prediction of remaining ICD battery longevity obtained at interrogation is a feature available only in the newest of ICDs. For devices without battery longevity estimates, the capacitor charge time reflects battery status, and the manufacturer should be consulted when charge time exceeds 12 seconds. General statements about battery voltage in ICDs cannot be made because ICDs have different numbers of battery cells.

Most ICDs should have their antitachycardia therapy disabled before the use of any device causing EMI. The use of monopolar ESU can cause inappropriate shocks. Casavant and associates found a stored electrogram sequence in an ICD, a finding suggesting that the device had misinterpreted monopolar ESU during dermatologic facial surgery as VF. Figure 48-12 shows inappropriate VT detection that occurred intraoperatively. Additionally, one report exists of a central line guidewire interacting with the RV lead, thus leading to a short-circuit shock and subsequent failure of the T-ICD.

The comments in the pacing section (including Appendix 48-2 and Box 48-3) apply here for any ICD with anti-bradycardia pacing. Many ICDs have no noise reversion behavior, so ESU-induced ventricular oversensing may lead to nonpacing in the patient who depends on the ICD for pacing.

### INTRAOPERATIVE (OR PROCEDURE) MANAGEMENT OF IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS

At this time, no special monitoring (resulting from the ICD) is required for the patient with an ICD. ECG
Chapter 48: Implantable Cardiac Pulse Generators: Pacemakers and Cardioverter-Defibrillators

Figure 48-11. Unexpected ventricular tachycardia (VT) with antitachycardia pacing was found in this patient during her preoperative visit. A 65-year-old woman with a history of VT had undergone implantation of a Medtronic single-chamber defibrillator approximately 8 months previously. She had not had any dizziness or syncopal episodes since placement of an implantable cardioverter-defibrillator (ICD). Preoperative interrogation of her device revealed VVE-VVI programming, along with an episode of tachycardia at 150 to 162 beats/minute that was detected by the ICD as VT. The ICD delivered a six-beat burst of antitachycardia pacing at 182 beats/minute, which converted the tachycardia back to sinus rhythm. No backup pacing was needed after the VT was terminated. The upper tracing is a digitized ventricular electrogram that was stored in the ICD during the tachycardic event. The lower tracing is the “marker channel,” which reports the interpretation of each event by the ICD. The numbers below the marker channel represent the interval (in milliseconds). The rate is calculated by dividing the interval into 60,000 msec/minute. TD marks the final event that starts therapy. TS represents an interval in the VT zone, TP is an antitachycardia pace event, and VS is an intrinsic ventricular depolarization with a rate that is neither too fast (short interval) nor too slow (long interval). This device was set to detect VT as 16 consecutive ventricular events with a rate between 146 and 200 beats/minute and to deliver antitachycardia pacing at 84% of the last R-R interval (the last interval was 400 msec, so antitachycardia pacing was delivered at a rate of 182 beats/minute (330 msec).

Figure 48-12. Electromagnetic interference (EMI) from the monopolar electrosurgery unit (ESU; “Bovie”) caused an implantable cardioverter-defibrillator (ICD) to detect ventricular fibrillation (VF). This stored electrogram (EGM) was one of 73 found at the end of a 4-hour surgical procedure in which considerable monopolar electrosurgery was used. This patient had a Guidant Medical ICD in the VOE-VVI mode. The patient’s ICD had been placed in a “monitor only” mode before surgery. As a result, the ICD recorded any instance of ventricular dysrhythmia that would have triggered therapy, but it could not actually deliver therapy. From left to right: 1, the EGM demonstrates a ventricular rate of 70 beats/minute, but with considerable noise on the baseline; 2, a VF event was declared for the detected heart rate of 345 beats/minute, and the ICD charged its capacitor; 3, the ICD was programmed to “reconfirm before shock,” and the ventricular rate remained 70 beats/minute with noise on the baseline; 4, again, the noise caused the ICD to believe that the patient remained in VF, and the ICD would have delivered a shock, except that it was programmed to “monitor only”; and 5, because the noise is gone (the ESU had stopped), the ICD declares the event over after “successful” defibrillation.
monitoring and the ability to deliver external cardioversion or defibrillation must be present during the time of ICD disenablement. Should cardioversion or defibrillation be needed, the defibrillator pads should be placed to avoid the PG to the extent possible. Nevertheless, the patient, not the ICD, is being treated. When a magnet is used to prevent ICD discharge, simple removal of a magnet may not produce immediate antiaarrhythmic therapy. The actual rate of the tachycardia versus the lowest treatment zone rate, as well programmed therapy delays, could lead to lengthy treatment postponements. The recommendations in the section on intraoperative (or procedure) management of pacemakers apply here, as well, to T-ICDs (S-ICDs have no permanent pacing at this time).

No special anesthetic techniques have been championed for the patient with an ICD. Most of these patients have severely depressed systolic function, dilated ventricular cavities, and significant valvular regurgitation. Thus, the choice of anesthetic technique should be dictated by the underlying physiologic derangements that are present. Conflicting data have been published regarding the choice of anesthetic drugs and changes to DFT. In 1992, Gill and associates examined DFTs in dogs and concluded that neither halothane nor isoflurane changed DFTs in open chest defibrillation compared with a pentobarbital infusion. However, Weinbroum and colleagues evaluated DFTs in humans during ICD implant and found that halothane, isoflurane, and fentanyl increased DFTs. Even with these increases, however, these increased DFTs were still substantially lower than the maximum energy generally available in ICDs, and these increases would not have been noted under usual testing conditions.

POSTANESTHESIA EVALUATION

Any ICD that was reprogrammed for an intervention must be interrogated and re-enabled, and deaths from failure to re-enable an ICD have been reported. All recorded events should be reviewed, and counters should be cleared. The pacing parameters (T-ICD only) must be checked and reprogrammed as necessary.

SPECIAL SITUATIONS: VENTRICULAR ASSIST DEVICES AND TEMPORARY PACING

Most patients with a ventricular assist device (VAD) also have a T-ICD. Although this subject exceeds the scope of this chapter, several important issues should be highlighted. First, the VAD can interfere with telemetry between the ICD and the programmer, thus resulting in the inability to modify parameters quickly and possibly necessitating T-ICD replacement. Several “workarounds” have been proposed, including changing the LVAD speed, covering the telemetry wand with an iron skillet, or encasing the wand and cord with metallic shielding material. Before placement of a T-ICD in a patient with a VAD, or placement of a VAD in a patient with a preexisting CIED, compatibility issues should be investigated. Second, in many of these patients who then undergo orthotopic heart transplantation, the T-ICD is disabled by programming, and the transvenous leads are cut at the level of the superior vena cava. Unfortunately, any subsequent device reset from cosmic radiation or EMI will restore single-zone shock behavior in all T-ICDs, so a patient may actually receive high-energy therapy to their superior vena cava, with likely disastrous result.

The second special situation is temporary pacing. Temporary pacing devices can be used for bradycardia unresponsive to pharmacologic therapy, bradyarrhythmias complicating myocardial infarction, heart rate support following cardiothoracic surgery, delivery of ATP to the atrium or ventricle in an attempt to treat a tachydysrhythmia, bridge to permanent PM placement, and protection for potential catastrophic failure of a CIED expected to be exposed to strong EMI in a pacing-dependent patient. Pacing can be esophageal, transcutaneous, transvenous, or epicardial. In a patient with a CIED, placement of a temporary pacing device requires CIED reprogramming for temporary pacing testing (e.g., threshold evaluation) because the energy of temporary pacing that fails to depolarize the myocardium could inhibit permanent pacing. A T-ICD must be disabled to prevent inappropriate shock during temporary pacing testing and also if central venous access is planned.

Esophageal pacing requires an intact atrium and AV node. Thus, esophageal pacing remains absolutely contraindicated when a history of atrial dysrhythmia (e.g., atrial fibrillation) or significant AV block exists. This technique requires a specially adapted esophageal probe and a unique generator. Pacing is AOO only, which usually causes no problems, even when the native heart rate exceeds the programmed heart rate. The pacing artifacts typically appear as large, negative “spikes” that can produce overcounting by the ECG monitor and can be confused as QRS events (see Fig. 48-9).

Transcutaneous pacing is accomplished by placing conducting electrodes on the skin, preferably in an anteroposterior position. Device instructions should be followed. Pacing is ventricular only, and this may not produce appropriate hemodynamic support without atrial transport.

Transvenous and epicardial pacing can be atrial, ventricular, AV, or BiV, depending on the catheter or wires and the pacing generator used. Transvenous pacing requires central venous access, and complete sterile barrier technique seems indicated in any patient, especially when a preexisting CIED is present. Three different types of transvenous catheters are available at this time: a simple balloon-tipped bipolar catheter without a hemodynamic channel; a pulmonary artery catheter with one or two extra lumina for placement of bipolar wires into the atrium, right ventricle, or both; and a multielectrode pulmonary artery catheter intended for atrial-RV pacing. Both transvenous pacing and epicardial pacing require a battery-powered external generator. In any patient with only a ventricular lead (whether transvenous or epicardial), the temporary pacing generator should be programmed only to VVI pacing because the use of DDD pacing (in the absence of a functioning atrial lead) can instigate VT secondary to R-on-T pacing (Fig. 48-13). The use of the DDD pacing mode.
Figure 48-13. DDD pacing without atrial lead initiates ventricular tachycardia (VT) by R-on-T pacing. Temporary external dual-chamber pacing without an atrial lead produced several R-on-T paces, one of which initiated polymorphic VT. This patient had undergone elective aortic valve replacement with root repair, three-vessel coronary artery bypass grafting, and a partial Maze procedure 8 hours earlier. His underlying rhythm was atrial fibrillation. Tracings are electrocardiogram lead I (upper) and lead V5 (lower). Pacemaker (PM) settings were DDD mode; pacing rate was 60 beats/minute; and AV delay was 200 msec, resulting in a VV cycle length of 1000 msec and a ventriculotrial time (VA) of 800 msec. AP is presumed atrial pace, BVE is a blanked ventricular event secondary to postatrial ventricular blanking, VP is ventricular pace, and VS is presumed ventricular sense. At the expiration of the VA timer, the PM presumably emitted an AP because no atrial signal had been sensed. The PM then invoked a 20- to 36-msec postatrial ventricular blanking period to prevent any ventricular oversensing resulting from the AP. As a result, the native ventricular event was not sensed (BVE), resulting in a VP 200 msec after the AP. The VP fell on the vulnerable period of repolarization, thus initiating VT. ABP, Arterial blood pressure; HR, heart rate; NBP, noninvasive blood pressure; PVC, premature ventricular contraction; Spo2, peripheral oxygen saturation. (From Schulman PM, Stecker EC, Rozner M: R-on-T and cardiac arrest from dual-chamber pacing without an atrial lead, Heart Rhythm 9:970-973, 2012.)

Glossary

Atrial Fibrillation (AF) Suppression (formerly Dynamic Atrial Overdrive): A programmable rate enhancement for CIEDs that increases the pacing rate in response to the presence of intrinsic atrial activity. AF suppression is designed to pace the atrium at a rate just above the intrinsic rate to prevent atrial fibrillation. Its presence and maximal pacing rate should be identified before an anesthetic regimen because elevated pacing rates may prompt treatment, which will not affect the heart rate but may harm the patient. Consideration should be given to disabling this feature intraoperatively, especially if monopolar ESU will be used superior to the umbilicus, where EMI would be expected.

Atrioventricular Delay: The time that a dual-chamber system waits after detecting (or initiating) an atrial event before pacing the ventricle. Some generators shorten this time as heart rate increases (termed rate-adaptive AV delay or dynamic AV delay). Some generators can be programmed to extend the AV delay to search for intrinsic conduction (search AV delay). Some generators prolong an AV delay after any atrial event in which the last ventricular event was intrinsic (AV delay hysteresis). In a patient with a conducting AV node, the sensed AV delay is slightly longer than the PR interval on the surface ECG (see Fusion Beat and Pseudofusion Beat) because the ventricular sensing element is attached to the apex of the right ventricle and detects the depolarization only after RV activation (typically >60 msec after activation of the AV node).

Automatic Rate. See Lower Rate Limit

Bipolar Lead: An electrode with two conductors. Bipolar pacing typically uses less energy than unipolar pacing, and it produces smaller artifacts on analog monitors.

Summary

Electronic miniaturization has permitted the design and use of sophisticated electronics in patients who need artificial pacing and/or automated cardioversion or defibrillation of the heart. These devices are no longer confined to merely keeping the heart beating between a minimum rate (pacing function) and a maximum rate (ICD functions) because they are now used as therapy to improve the failing heart. Furthermore, the advent of sophisticated pacing algorithms that change heart rate or AV intervals, as well as permit occasional dropped QRS events, can mimic behavior that may be interpreted as “light anesthesia” (increased pacing rates) or pacing system malfunction. Inappropriate treatment of these events can lead, and has led, to injury and death in these patients.

Both the aging of the population and the ability to care for a patient with increasingly complex disease suggest that anesthesiologists will be caring for many more patients with these devices, and we must be prepared for this situation. Safe and efficient clinical management of these patients depends on an understanding of implantable systems, indications for their use, and the perioperative needs they create.
Bipolar sensing is more resistant to oversensing from muscle artifact or stray electromagnetic fields. Most pacing generators (but not ICDs) can be programmed to unipolar mode (separate settings for pacing and sensing), even in the presence of bipolar electrodes.

**EGM Mode (Electrogram Storage Mode):** Passive acquisition and internal storage of ECG data for diagnostic purposes while pacing (or monitoring) with programmed parameters. A PM programmed to enter the EGM mode on application of a magnet will not demonstrate asynchronous pacing behavior.

**Fusion Beat (FB):** A PM spike delivered shortly before a native depolarization of the ventricle that alters the morphology of the QRS, often misdiagnosed as undersensing. With an FB, the pacing stimulus is delivered after the activation of the AV node but before the sensing element detects the PM-induced depolarization. It is similar to preexcitation of the ventricle seen in Lown-Ganong-Levine syndrome or Wolff-Parkinson-White syndrome. Confirmation of appropriate sensing behavior can be made by lengthening the sensing interval (i.e., lengthening the AV delay). Fusion beats suggest, but should not be used to confirm, ventricular capture. See also Pseudofusion Beat.

**Generator:** The device with a power source and circuitry to produce an electrical impulse designed to be conducted to the heart. Typically, cardiac generators are placed in a pectoral pocket, and leads are inserted into the right atrium, right ventricle, or both. Since 1995, ICDs have also been approved for pectoral pocket placement.

**Hysteresis:** If present, the amount by which the patient’s intrinsic rate must fall below the programmed rate before the generator begins pacing. Some pacers periodically decrease the pacing rate to search for resumption of intrinsic activity (called search hysteresis). These functions, when present, can mimic PM malfunction.

**ICD (Implantable Cardioverter-Defibrillator) Mode:** The designation of chambers shocked, chambers paced for antitachycardia pacing, method of tachycardia detection, and chambers paced for antitachycardia therapy. Table 48-4 shows the NASPE/BPEG generic ICD code.

**Lower Rate Limit (LRL, also Automatic Rate or Programmed Rate):** The lowest sustained regular rate at which the generator will pace. Generators convert this value into the maximum allowable R-R interval and emit a pacing pulse at the expiration of this time. Dual-chamber pacing devices subtract the AV delay to determine the maximum allowable interval from a ventricular event to the expected atrial event.

**Mode Switch:** In dual chamber (AV) systems, a feature designed to limit ventricular pacing during high atrial rates (whether atrial fibrillation, flutter, or tachycardia) by temporarily switching to DDI(R) / VVI(R) depending upon the manufacturer. The mode switch detect rate, duration of high rate tracking (pre-mode switch) and return to dual chamber tracking mode upon termination of the atrial high rate can be manufacturer and programming dependent. Often, removal of a magnet in the setting of a high atrial rate will lead to transient ventricular pacing at the upper tracking rate until mode switch occurs. EMI can also cause inappropriate mode switch with resultant loss of atrial transport producing hemodynamic embarrassment.

**Oversensing:** Detection of undesired signals that are interpreted as cardiac activity. Ventricular oversensing can lead to ventricular pause (PM or ICD) or inappropriate antitachycardia therapy (ICD). Atrial oversensing can lead to inappropriately high paced ventricular rates in DDD or VDD devices; these ventricular systoles occur in the absence of true atrial systoles, and hemodynamic compromise can result.

**Pacing Mode:** The designation of chambers paced, chambers sensed, sensing response, rate responsiveness, and antitachyarrhythmia function for a PM system. Table 48-2 shows the NASPE/BPEG generic PM code.

**Programmed Rate. See Lower Rate Limit**

**Pseudofusion Beat (PFB):** A PM spike delivered shortly after a native depolarization without alteration of the QRS morphology. PFBs are often misdiagnosed as undersensing, and they result from the position of the sensing electrode relative to the depolarizing wavefront (see Fusion Beat). Confirmation of appropriate sensing behavior can be made by lengthening the sensing interval (i.e., decreasing the program rate [atrial FB] or lengthening the AV delay [ventricular PFB]). PFB cannot be used to confirm electronic capture.

**Rate Enhancements:** Features such as rate-adaptive AV delay (shortens the AV delay with increasing heart rate), AV search hysteresis (lengthens or shortens the AV delay to produce intrinsic AV conduction), AF suppression (also called dynamic atrial overdrive; increases the lower rate on appearance of native atrial depolarization, thus creating nearly constant atrial pacing but at a rate only slightly higher than the patient’s intrinsic rate), rate smoothing (limits changes in ventricular paced rates resulting from changes in atrial rates; rising and falling rate limits can be programmed separately), sleep rate (see Sleep Rate), ventricular rate regulation (similar to rate smoothing but used only during atrial fibrillation), and hysteresis (see Hysteresis). Each of these enhancements can produce pacing or nonpacing that can mimic PM dysfunction, and consideration should be given to disabling these enhancements before any anesthetic regimen.

**Rate Modulation:** The ability of the generator to sense the need to increase heart rate in response to the patient’s activity. Mechanisms include (1) a mechanical sensor in the generator to detect motion or vibration, (2) electronic detection of the QT interval (shortens during exercise), (3) transthoracic impedance to measure changes in respiration, and (4) sensors for central venous blood temperature or oxygen saturation (see Table 48-3). Some generators now incorporate multiple sensors. The pacing rate determined by the rate modulation algorithm is called sensor indicated rate.

**Rest Rate:** A rate lower than the programmed rate, invoked by the pacing device after some period (manufacturer specific) of patient inactivity, which is determined by the exercise sensor.

**Sensor Indicated Rate:** The pacing rate determined by the sensor in a PM programmed to a rate-responsive mode (fourth character of NBG = R).
Sleep Rate (also Circadian Rate): The rate (lower than the programmed rate) at which the pacing generator will pace during programmed “nighttime” hours. Travel across time zones can lead to maladaptive behavior when using this feature.

Undersensing: Failure to detect a desired event.

Unipolar Lead: An electrode with only one conductor. Some devices with bipolar leads are programmed to the unipolar lead mode. Systems with unipolar leads produce larger spikes on the ECG than bipolar leads. Systems with unipolar leads use the generator case as the second conductor. Unipolar pacing or sensing modes cannot be programmed in an ICD.

Upper Sensor Rate (USR, also Upper Activity Rate or UAR): The maximum rate to which a rate modulated CIED can drive the heart. The USR is not affected by UTR because when USR becomes active, the CIED is pacing the atrium.

Upper Tracking Rate (UTR, also called Upper Rate Limit or URL): In dual chamber (AV) systems, the maximum rate at which the ventricle can be paced regardless of the atrial rate. When the atrial rate exceeds this rate, the observed AV delay will lengthen and some QRS events can be dropped, a behavior that appears similar to Wenckebach rhythms.

Complete references available online at expertconsult.com

REFERENCES

PART IV: Anesthesia Management

Determining the indication for and date of initial device placement
Identifying the type of device (pacemaker, implantable cardioverter-defibrillator) and manufacturer
Determining the patient’s underlying rhythm and rate (if any)
Identifying the number and types of leads
Determining the last generator test date and battery status
Obtaining a history of generator events (if any)
Obtaining the current program information (device interrogation), including mode, rate, and rate enhancements

Ensuring that generator discharges become mechanical systoles with adequate pacing safety margins
Ensuring adequate safety margin for sensing events (if intrinsic events are present)
Ensuring that magnet detection is enabled (magnet behavior and rate should be recorded)
Determining whether the pacing parameter should be reprogrammed, which will depend on, for example, pacing dependency, surgery type and location, and need for increased heart rate

APPENDIX 48-1  Pulse Generator Company Telephone Numbers

<table>
<thead>
<tr>
<th>Company</th>
<th>Phone Number</th>
<th>Company</th>
<th>Phone Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM Pacemaker Corp.</td>
<td>800-227-3422</td>
<td>Diag/Medcor</td>
<td>800-722-3774</td>
</tr>
<tr>
<td>(Guidant Medical)</td>
<td></td>
<td>Edwards Pacemaker Systems</td>
<td>800-325-2518</td>
</tr>
<tr>
<td>Angelon (Sorin)</td>
<td>800-352-6466</td>
<td>ELA Medical (Sorin)</td>
<td>800-352-6466</td>
</tr>
<tr>
<td>Arco Medical (Boston Scientific)</td>
<td>800-227-3422</td>
<td>Intermedics (Boston Scientific)</td>
<td>800-227-3422</td>
</tr>
<tr>
<td>Biotronik</td>
<td>800-547-0394</td>
<td>Medtronic</td>
<td>800-505-4636</td>
</tr>
<tr>
<td>Boston Scientific</td>
<td>800-227-3422</td>
<td>Pacesetter (St. Jude Medical)</td>
<td>800-722-3774</td>
</tr>
<tr>
<td>Cardiac Control Systems</td>
<td></td>
<td>Siemens-Elema (St. Jude Medical)</td>
<td>800-722-3774</td>
</tr>
<tr>
<td>Cardio Pace Medical, Inc.</td>
<td></td>
<td>Sorin</td>
<td>800-352-6466</td>
</tr>
<tr>
<td>(Novacon)</td>
<td></td>
<td>Telecommunications Pacing (St. Jude Medical)</td>
<td>800-722-3774</td>
</tr>
<tr>
<td>Cardiac Pacemakers, Inc.:</td>
<td>800-227-3422</td>
<td>Ventritex (St. Jude Medical)</td>
<td>800-722-3774</td>
</tr>
<tr>
<td>CPI (Boston Scientific)</td>
<td></td>
<td>Viatron</td>
<td>800-328-2518</td>
</tr>
<tr>
<td>Coratonic (Biocontrol Technology)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cordis Corporation</td>
<td>800-722-3774</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(St. Jude Medical)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

APPENDIX 48-2  Preanesthetic Cardiovascular Implantable Electronic Device (Pacemaker, Implantable Cardioverter-Defibrillator) Evaluation

*The preanesthetic cardiovascular implantable electronic device evaluation should consist of a device interrogation. The foregoing statements can be fashioned into a request to the cardiologist or pacemaker service. For implantable cardioverter-defibrillators, the term generator events includes a history of antitachycardia therapy.
REFERENCES


References


49. Crossley GH, Poole JE, Rozner MA, et al: The Heart Rhythm Society (HRS)/American Society of Anesthesiologists (ASA) Expert Consensus Statement on the Perioperative Management of Patients with Implantable Defibrillators, Pacemakers and Arrhythmia Monitors: Facilities and Patient Management: this document was developed as a joint project with the American Society of Anesthesiologists (ASA), and in collaboration with the American Heart Association (AHA), and the Society of Thoracic Surgeons (STS), Heart Rhythm 8:1114-1154, 2011.


Downloaded from ClinicalKey.com at Buddhist Tzu Chi General Hospital JC September 17, 2016.


