Contemporary Pacemaker and Defibrillator Device Therapy
Challenges Confronting the General Cardiologist

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Intracardiac pacemaker and defibrillator implantations were initially described a half century and a quarter century ago, respectively.1,2 Originally conceived as devices for the treatment of symptomatic bradycardia and sustained ventricular tachyarrhythmias, these therapies have subsequently been applied to the management of an increasing host of arrhythmia and other indications, including the treatment of heart failure.3 Although underuse of this technology has occasionally been reported,4 it has become abundantly clear that the number of patients deriving benefit from device therapy has risen exponentially; cardiac electrophysiology and pacing has emerged as a subspecialty within cardiology to address the growing needs of cardiac rhythm management.5 It is, nonetheless, incumbent on the general cardiologist to become proficient in certain aspects of device therapy.6 The goals of the present review are to highlight some of the fundamental areas of knowledge essential for follow-up, to provide an update on newer applications of device therapy, and to address some of the more common challenges and clinical scenarios encountered by the general cardiologist.

Changing Indications for Device Therapy
The implantable cardioverter-defibrillator (ICD) was originally applied to patients surviving life-threatening ventricular tachyarrhythmias—so-called secondary prevention. Subsequently, primary prevention trials identified other patients who were at increased risk for sudden cardiac death but who had yet to manifest such risk,7 thereby expanding the indications for ICD therapy. The most recent iteration of guidelines for device implantation has identified additional conditions in which pacemaker and/or ICD therapy are associated with an enhancement of quality of life and/or survival.5,8 In particular, new ICD indications for primary prevention include prophylactic implantation in patients with ischemic cardiomyopathy and ejection fraction (EF) less than or equal to 30% (MADIT-II [Multicenter Automatic Defibrillator Implantation Trial II] criteria),9 and in patients with Brugada syndrome.10 The potential importance of ICD therapy for patients with nonischemic cardiomyopathy, EF less than 35%, and New York Heart Association class II or III congestive heart failure (CHF) symptomatology was demonstrated by the SCD-HeFT (Sudden Cardiac Death in Heart Failure) trial,11 a primary prevention application published after the 2002 guidelines.

Potential new applications of cardiac pacing have been recognized, such as pacing in patients with neurocardiogenic syncope.3 Ironically, this application has been the subject of more recent debate: No benefit was found in the Second Vasovagal Pacemaker Study.12 Cardiac resynchronization therapy (CRT) has been identified as a class IIA therapy for patients with medically refractory New York Heart Association class III or IV CHF patients with cardiomyopathy, QRS interval of greater than or equal to 130 ms, and EF less than or equal to 35%.13,14 This latter application has allowed for a “marriage” of technologies in 1 device (CRT-ICD) that allows for treatment of CHF and protection against sudden cardiac death in selected patients.

Indications may flourish and, almost as rapidly, languish. The time-honored use of dual-chamber pacing to achieve physiological pacing15 has come under new scrutiny. This concept, as originally proposed, referred to pacing techniques that were designed to allow for normal cardiovascular physiology, preserve atrioventricular (AV) synchrony, and provide heart rate increases (rate adaptiveness) as required by exercise or other physiological demands.16 Although this improved symptoms in many patients and reduced the incidence of atrial fibrillation (AF) compared with single-chamber pacing, no benefit was identified in certain patient subsets,17-19 as in the PASE (Pacemaker Selection in the Elderly)17 and UKPACE (United Kingdom Pacing and Cardiovascular Events)18 trials. Furthermore, potential adverse consequences of right ventricular apical pacing have led to the evaluation of alternative sites for pacing the heart,20 and our definition of physiological pacing continues to evolve.21 All of these points attest to the importance of periodically revisiting and updating implantation indications and guidelines as new data from randomized clinical trials become available.7,8,22

Follow-Up of the Patient After Device Implantation
Much has been written on device follow-up, and training requirements have been established for the selection, implantation, and follow-up of cardiac implantable electronic devic-
Six-month intervals for ICD follow-up seem to be safe;27 the same applies to pacemaker follow-up. These follow-up intervals may be extended as a result of innovations in remote monitoring28,29 and device automaticity. The latter may be defined as the automatic regulation of device function by programmed algorithms (see below) that are based on patient conditions and pacemaker/ICD system conditions, without the need for clinician input.30 Transtelephonic monitoring of pacemakers has been available for nearly 3 decades. The technology has shown that there is a relatively long trouble-free period in the midst of a device’s life; most issues arise early after implantation (wound healing, threshold changes) or toward the device’s end of life.30 These lessons have been applied increasingly to the management of ICDs; depending on the manufacturer, remote monitoring may be enabled through Internet-based systems or through radiofrequency transmission from a transmitter in the ICD via a phone device to a service center.

Nonetheless, there is no substitute for direct patient contact, with an essential role for the physician to uncover device-related issues.24 In addition to periodic routine visits, more urgent examination may be required, as in the case of an ICD patient with increasingly frequent ICD discharges (as opposed to a single ICD shock, which could be addressed through remote monitoring). History taking is crucial to ensure that symptoms prompting the original implantation remain ameliorated and that device therapy has not resulted in the creation of new symptoms, as in the so-called pacemaker syndrome.31 This syndrome, most commonly caused by the loss of optimal AV synchrony in patients with single-chamber ventricular pacing, consists of cardiovascular and neurological symptoms that include neck/abdominal pulsations, palpitation, fatigue, dyspnea, and/or presyncope; the pathophysiology reflects diminished cardiac output and is associated with signs of systemic hypotension, AV valvular regurgitation, unpleasant cannon A waves in the neck, and pulmonary/hepatic congestion.24

Examination of the wound is important early after implantation to address wound-healing as well as potential infection; the latter is particularly serious because it is rare for a patient to overcome device-related infections without extraction of the entire system.24 Pacing-induced complications such as diaphragmatic stimulation or myopectoral stimulation/inhibition also may be identified. Examination of chest radiographs is occasionally necessary to confirm lead integrity and position.32 The importance of 12-lead electrocardiography cannot be understated, not only for assessment of rhythm but also for confirmation of the appropriate QRS complex–paced morphology associated with various pacing lead locations (typically left bundle-branch block morphology and left-axis deviation with conventional right ventricular apical pacing). This examination is particularly important with the advent of alternative-site pacing, where less typical ECG patterns are observed.20,33

Magnet application to a pacemaker generator is a useful adjunct to follow-up and converts the pacemaker to the asynchronous pacing mode (not inactivation).23 The resulting asynchronous rate is manufacturer specific and changes over the life of the battery as it approaches a preset elective replacement indicator rate. Thus, magnet application is useful in following device longevity and in confirming capture at the programmed output. It may be used to temporarily induce asynchronous pacing in cases of electromagnetic interference (EMI), such as electrocautery. With EMI, external signals may be detected by the device and misconstrued as intracardiac, resulting in inhibition of pacing output, unless sensing is eliminated by either magnet application or device reprogramming. In contrast, magnet application to ICD generators does not affect pacing, but it may temporarily or permanently suspend antitachycardia therapies.

The importance of device interrogation and programmability has been appreciated since the inception of pacemaker therapy.34,35 Unfortunately, there is no universal pacemaker/ICD programmer to date; the manufacturer-specific nature of programmers constrains the nonelectrophysiologist who may not be familiar with or have access to the seemingly vast array of programmers required for follow-up. Ironically, allied professionals, often industry employed, may know more about a particular device and its associated programmer than a given physician; guidelines for such assisted follow-up have been established, and the physician must remain responsible for the device management and the patient’s overall care.36

Interrogation of a device provides a wealth of information, including but not limited to programmed parameters, such as the pacing rates and pacing mode (Table 1)37; real-time lead and battery impedance, as well as trends; event counters and histograms detailing the percentage of time sensed/paced, automatic mode-switch episodes, and atrial and/or ventricular arrhythmias; real-time and stored intracardiac electrogams; and marker channels that indicate what a device thinks it has seen or is seeing (Figure 1). Interrogation of programmed settings is essential in explaining phenomena that may be misinterpreted as device malfunction. For example, if a pacing rate is lower than the programmed lower tracking rate in a correctly functioning pacemaker, the phenomenon may be understood if interrogation also reveals that a hysteresis function has been programmed, allowing the patient’s native rhythm to be maintained and to dip down to lower rates before pacing is triggered at a faster rate; this reduces current drain and preserves native rhythm.

The inability to perform telemetry could indicate that the wrong manufacturer’s programmer was used, that the telemetry wand has not been positioned adequately over the generator (some models also require a magnet applied to the wand), or that the generator has reached anticipated (or premature) end of life.38 Elevated lead impedances may suggest a fracture in the conductor coil(s), a loose-set screw site at the lead insertion into the header block, or a problem at the tissue–electrode interface39 (Figure 2). Reduction of lead impedance may indicate a breach in lead insulation, often seen at the site of transvenous insertion—the so-called subclavian crush syndrome.40 Analysis of impedance trends may actually provide historical insight as to when lead disruption may have occurred (Figure 3). Reduction in battery voltage and a rise in battery impedance are characteristic of generators over time, providing a method by which to assess generator longevity.
Device counters may allow for the quantification of events such as high atrial rates, high ventricular rates, and automatic mode-switch episodes, suggesting the interim occurrence of arrhythmias such as AF, supraventricular tachycardia (SVT), ventricular tachycardia (VT), or ventricular fibrillation (VF). Far-field sensing of ventricular depolarization, T waves, or noise by the atrial channel may result, however, in a false-positive incidence of high atrial rate or mode-switch episodes (Figure 4). Thus, pacemaker memory functions do not obviate the need for Holter or event monitoring in all patients with suspected arrhythmias. However, longer durations of individual episodes, on the order of many minutes to days, especially if confirmed by intracardiac electrogroms, are more likely to correspond to true atrial dysrhythmias and an increased association with clinical events such as thromboemboli. 

Although device counters give a rough indication as to true atrial dysrhythmias and an increased association with clinical events such as thromboemboli, certain algorithms have been developed to automatically adjust the gain or sensing threshold; these algorithms warrant some understanding on the part of the general cardiologist.

Automatic Algorithms for Detection and Treatment: A Brief Synopsis

It is beyond the scope of this review to detail all the device-specific algorithms available for sensing, discrimination, pacing, and defibrillation. Certain algorithms have, however, been increasingly used to automatically facilitate device function and follow-up; these algorithms warrant some understanding on the part of the general cardiologist.

Automatic algorithms that are intended to enhance sensitivity and optimize arrhythmia detection invariably encounter challenges regarding under- and oversensing. This is particularly important for ICDs where low-amplitude fibrillatory signals from the ventricle must be adequately sensed while avoiding oversensing in sinus rhythm. ICDs typically have algorithms to automatically adjust the gain or sensing threshold to sense reduced signals; it is, therefore, uncommon for current ICDs to miss VF. Conversely, oversensing may result in spurious discharge or inhibition of pacing output in ICD patients who are pacemaker dependent (Figure 6).

Algorithms that allow for rhythm discrimination have become essential to ICD management. Inappropriate or spurious device discharges may be observed in up to 40% of patients, typically from misclassification of sinus tachycardia or from rapidly conducting supraventricular arrhythmias. For single-chamber devices, ventricular rate has served as the primary determinant of arrhythmia detection. Additional features such as sudden-onset criteria have aided discrimination of sinus tachycardia (gradual in onset) from VT or VF (typically sudden in onset) by comparing the interval at the onset of the tachycardia against an average of the previous intervals. This will not, however, distinguish VT from SVT.

Rate-stability enhancements allow for measurement of beat-to-beat intervals, noting that monomorphic VT is typically regular, whereas AF and polymorphic VT or VF have wider variability when beat-to-beat variability is assessed. This feature proves to be particularly useful in discriminating AF from monomorphic VT. These enhancements reduce the likelihood of delivering spurious shocks for SVT without undersensing or delaying treatment for VT. Beyond this, certain algorithms have been developed to assess morphological characteristics of the electrogram during a patient’s baseline rhythm. The resulting template may then be compared with the morphological characteristics obtained during a tachycardia, thereby aiding in the discrimination of supraventricular and ventricular rhythms. Although it is unpleasant for a patient to receive spurious shocks for misclassified SVT, it is far more hazardous for VT to be undetected; this has led manufacturers to incorporate a programmable time limit for tachycardia duration that, when exceeded, will result in the delivery of VT therapy.

In dual-chamber ICDs, the presence of an atrial lead allows for comparison of ventricular and atrial rates during baseline rhythm and tachycardia, as well as the relative timing of atrial and ventricular activity. Ventricular rates exceeding atrial

### TABLE 1. Pacemaker Mode Code

| Position I | Chamber paced: A indicates atrium; V, ventricle; D, both chambers |
| Position II | Chamber sensed: O indicates none (asynchronous); A, atrium; V, ventricle; D, both chambers |
| Position III | Response of pacer to sensed activity: O indicates none; T, triggered/tracked; I, inhibited; D, both triggers and inhibits output in a particular chamber, depending on the sensed event |
| Position IV | Rate modulation: O indicates none; R, rate adaptive |
| WI | Ventricular demand pacing; inhibits ventricular pacing output if native activity is sensed above lower ventricular demand rate |
| VOO | Asynchronous ventricular pacing (no sensing capabilities) |
| WT | Triggered ventricular pacing (perceives native ventricular activity and triggers ventricular pacing spike to coincide with sensed activity) |
| VVR | Rate-adaptive ventricular pacing |
| AAI, AAIR | Atrial demand pacing, atrial rate-adaptive pacing |
| DDD, DDDR | Dual-chamber pacing, dual-chamber rate-adaptive pacing |
| DDI | DDD pacing without atrial tracking; AV paces at lower demand rate and inhibits atrial pacing output if atrial activity is sensed but does not trigger ventricular pacing in response to sensing atrial activity above the lower demand rate |
| DDIR | DDI Rate-adaptive pacing |
| VDD | Atrial synchronous pacing; senses/tracks atrial activity (to trigger ventricular pacing); senses in ventricle to inhibit if native ventricular activity is sensed; does not pace in atrium |


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rates are typical for ventricular tachyarrhythmias with AV dissociation. Intracardiac electrograms are particularly useful in this regard (Figure 5). The Detect SVT trial reported a reduction of inappropriate SVT detection from 39.5% in the single-chamber detection arm compared with 30.9% in the dual-chamber arm. Arrhythmia detection by dual-chamber ICDs may be enhanced by combining various discriminators.

A recent multicenter trial, Comparison of Empiric to Physician-Tailored Programming of Implantable Cardioverter-Defibrillators (EMPIRIC), reported that standardized empirical ICD programming was as effective as physician-tailored programming as long as the empirical strategy included avoidance of detection of nonsustained tachycardia, avoidance of detection of SVTs as VT, empirical antitachycardia pacing for slow and fast VTs, and high-output first shocks. Nonetheless, misclassification of arrhythmic events is an ongoing concern, and much work remains in the refinement of detection algorithms, particularly in delineating rapid SVTs. For the sake of completeness, it should be acknowledged that automatic sensing algorithms have been applied to a host of sensors unrelated to either atrial or ventricular depolarization, allowing for rate-adaptive pacing that is particularly well suited to patients exhibiting chronotropic incompetence.

For the sake of completeness, it should be acknowledged that automatic sensing algorithms have been applied to a host of sensors unrelated to either atrial or ventricular depolarization, allowing for rate-adaptive pacing that is particularly well suited to patients exhibiting chronotropic incompetence. Thus, activity sensors, accelerometers, minute ventilation, and QT-interval sensing have been used alone or in combination to provide for more physiological rate modulation. Beyond sensing and detection, automatic algorithms have also been increasingly used in the treatment and prevention of arrhythmias. Pacing for bradycardia has been facilitated by automatic capture threshold assessment. The determination of ventricular capture threshold may be made at programmed intervals during the day or on a beat-by-beat basis with corresponding adjustments in output. Periodic and frequent assessments of thresholds are important from a safety standpoint because thresholds may change as a function of multiple factors, including changes in autonomic tone, electrolyte and metabolic abnormalities, and concomitant antiarrhythmic drug therapy. On the other hand, the ability to automatically
down-regulate outputs when improved thresholds are detected extends device longevity.

Automatic modulation of the AV interval may be accomplished in various ways in current pacing systems. It has long been appreciated that optimal hemodynamics are achieved with shorter AV delay intervals after atrial-sensed compared with atrial-paced events, and hemodynamics may be further enhanced by rate-adaptive shortening of the paced AV interval at faster heart rates.

Conventional right ventricular apical pacing may contribute to cardiac desynchronization and foster or exacerbate CHF in pacemaker patients. Thus, in the DAVID (Dual Chamber and VVI Implantable Defibrillator) trial, committed dual-chamber pacing was associated with increased death or hospitalization for CHF in ICD patients with depressed EF compared with patients programmed to an intentionally slow backup ventricular-paced mode. The MOST (Mode Selection Trial) identified an increased rate of CHF hospitalization associated with ventricular-paced rhythm in pacemaker patients with sinus node dysfunction. What options, then, are available to minimize cardiac desynchronization induced by right ventricular apical pacing? Reprogramming to atrial pacing mode is one alternative, recognizing, however, that there is a finite, albeit small progression to AV block in patients with sick sinus syndrome, approximating 0.6% of patients per year. Programming a fixed long AV delay may minimize ventricular pacing, but it is not always feasible. In recognition of this, some manufacturers have incorporated conduction search algorithms that automatically search for intrinsic ventricular events that, if not sensed during an extension of the AV interval, will resume stimulation at the programmed AV delay (Figure 7).

In an effort to mimic naturally occurring circadian rhythms, some devices allow for programming of algorithms that will allow for slower pacing rates nocturnally. Another hysteresis function allows for pacing to kick in only when an abrupt rate drop is detected, applied with mixed results in the management of patients with neurocardiogenic syncope. Rate-smoothing programs are designed to prevent sudden changes in ventricular cycle length that may be responsible for either inducing pause-dependent arrhythmias or exacerbating symptoms of cardiac irregularity that are often encountered in AF.

Automatic mode switching is, undoubtedly, one of the most useful and widely applied algorithms currently available; it provides for the detection of atrial tachyarrhythmias and conversion from dual-chamber to single-chamber ventricular pacing to preempt pacer-mediated tachycardia, with reversion to dual-chamber pacing once the sinus mechanism has reemerged (Figure 8). Oversensing, which results in false-positive automatic mode-switching responses, may be observed, most commonly from far-field sensing of the end of the QRS complex by the atrial channel, especially in the setting of noise and low right atrial implants (Figure 4). An analysis of automatic mode-switching episodes may provide the clinician with an estimate of the incidence of atrial dysrhythmias, prompting clinical decisions such as institution of anticoagulation, antiarrhythmic drug therapy, or even ablation; the above limitations of algorithm analysis regarding sensitivity and specificity must, however, be considered.

Ventricular Pacing Impedance

<table>
<thead>
<tr>
<th>At Implant</th>
<th>680 ohms</th>
<th>Highest</th>
<th>&gt;3000 ohms</th>
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<tbody>
<tr>
<td>Last</td>
<td>&gt;3000 ohms</td>
<td>Lowest</td>
<td>432</td>
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Figure 3. Graph of impedance trends in defibrillator patient, showing intermittent elevations of impedance attributable to make-and-break connections at header block of a pacemaker generator.
Finally, some commentary is warranted regarding the potential utility of pacing algorithms for the prevention and/or treatment of AF. A variety of different methodologies have been developed to increase the atrial pacing rate when the native rhythm emerges and to periodically reduce the search rate for intrinsic atrial activity (dynamic atrial overdrive),\(^6\) transiently use higher rate pacing after mode-switch episodes or spontaneous atrial ectopy, and to pace rapidly alone or in combination with shock therapy to terminate established arrhythmias.\(^6\) In general, evidence-based data in this area are sparse, and much work is needed to clarify whether there is any role for the use of permanent pacing in the prevention or treatment of AF, as recently noted by a joint working group of the American Heart Association and the Heart Rhythm Society.\(^6\)

**Alternative Site Pacing and CRT**

As previously noted, there has been growing evidence that cardiac desynchronization induced by conventional right ventricular apical pacing may have deleterious effects on cardiac function and patient symptomatology, corresponding with an increased risk of heart failure and death.\(^2\) Invasive strategies have been used to minimize right ventricular pacing by lowering the baseline rate, changing to atrial-paced mode, or extending the AV delay.\(^6\) Invasive techniques include upgrading from right ventricular apical pacing to biventricular pacing,\(^7\) alternative or selective site pacing from the His bundle,\(^7\) and primary CRT with left ventricular or biventricular pacing in patients with CHF, as in the MUSTIC (Multisite Stimulation in Cardiomyopathies),\(^7\) MIRACLE (Multicenter InSync Randomized Clinical Evaluation),\(^7\) COMPANION (Comparison of Medical Therapy, Pacing and Defibrillation in Heart Failure),\(^7\) and CARE-HF (Cardiac Resynchronization-Heart Failure) trials,\(^7\) or in non-CHF patients requiring pacing for standard indications (the HOBIPACE [Homburg Biventricular Pacing Evaluation] study).\(^7\)

The general cardiologist may be called on to interpret chest radiographs and electrocardiograms that may become increasingly complex with these alternative pacing modalities. Recently, electrocardiographic demonstration of a ratio of 1 or greater in the R/S wave amplitude in lead V1 has been shown to reliably detect left ventricular capture in patients with cardiac resynchronization devices (Figure 9). Changes in morphology may also be appreciated during assessment of pacing thresholds in biventricular systems (Figure 10).

It is beyond the scope of the present article to detail the burgeoning area of CRT. Selection of optimal candidates for this therapy has typically included New York Heart Association class II or III CHF patients with a widened QRS complex. Although a wider baseline complex (greater than 150 ms versus 120 to 150 ms) in the PATH-CHF (Pacing Therapies in Congestive Heart Failure) study correlated with CRT benefit,\(^7\) other studies have demonstrated that QRS complex duration is a poor predictor of the response to CRT;\(^7\) in addition, the extent of shortening of the QRS complex has not predicted better individual outcomes with CRT.\(^7\) Indeed, attention has been directed increasingly at baseline markers of mechanical dyssynchrony as assessed by echocardiography and tissue Doppler imaging.\(^7\) Thus, patients with a wide QRS complex, that is, electrical dyssynchrony (but without evidence of mechanical dyssynchrony), may not prove to be ideal candidates for CRT. Mechanical dyssynchrony may exist in various forms, including AV dyssynchrony (delay between atrial and ventricular contraction), interventricular dyssynchrony (delayed activation of the left ventricle relative to the right ventricle), and intraventricular dyssynchrony (delayed activation of 1 segment of the left ventricle relative to another).

It remains unclear how the individual forms of dyssynchrony contribute to CHF; equally unclear is the extent to which echocardiographic optimization of these individual forms will ameliorate CHF, and when and how such optimization should be performed. Confounding this problem is the difficult issue of assessing the clinical response to CRT. There is no consensus on what constitutes an adequate response to CRT or on what defines a nonresponder.\(^7\) If, after 6 months of therapy, there is either a lack of symptom-
atic improvement or worsening CHF, a variety of contributing factors may be considered: poor patient selection, development of AF, change in cardiac substrate with worsening cardiomyopathy or ischemia, loss of LV or RV capture, and lack of ideal cardiac resynchronization (nonoptimal AV and VV delays, poor LV lead position). Optimization of the AV delay may increase stroke volume through an increase in preload and may be achieved by guiding programming AV-interval changes through simultaneous echocardiographic assessment of the mitral Doppler inflow pattern. Left bundle-branch block typically delays activation, resulting in fusion of the E and A waves. The optimal AV delay is the shortest possible delay that allows for complete diastolic ventricular filling without interfering with the atrial contribution; the echocardiographic representation of this is separation of the E and A waves without truncation of the A wave (Figure 11). This technique, though widely applied, has yet to be validated in LV-based pacing. Optimization of the VV interval, correcting for interventricular dyssynchrony between RV and LV contraction, is a programmable feature available in some devices that may contribute to hemodynamic and symptomatic improvement.

Certain ICDs have the ability to assess changes in intrathoracic impedance as measured between the pacing lead and generator; an inverse correlation has been demonstrated between intrathoracic impedance and pulmonary capillary wedge pressure, often heralding the onset of fluid overload. Application of this technology may allow the practitioner to institute preemptive changes in medical therapy when such changes are detected, thereby avoiding hospitalization (Figure 12). Thus, management of CHF in patients with CRT devices may well require extensive collaboration among the general cardiologist, electrophysiologist, echocardiographer, and heart failure specialist.

**Drugs, Devices, and EMI**

Pharmacological agents, especially antiarrhythmic drugs, may be required in patients with devices; typical agents include sotalol, amiodarone, mexiletine, and dofetilide. Among secondary prevention patients with spontaneous or
inducible VT who were studied for 1 year in the OPTIC (Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients) trial, shocks were observed in 38.5% of patients on β-blockers alone, in 24.3% of patients on sotalol, and in only 10.3% of patients treated with amiodarone and a β-blocker. In another secondary prevention study, first ICD therapy occurred in patients whose initial indication was sustained monomorphic VT (as opposed to cardiac arrest) and LVEF ≤ 25%; subsequent therapy occurred sooner and unpredictably, suggesting benefit of initiating antiarrhythmic drug therapy soon after the first symptomatic ICD therapy. If frequent nonsustained VT is evident at the time of implantation, such drugs may be considered even sooner. What is less clear is the role of prophylactic antiarrhythmic drugs in primary prevention patients who have yet to demonstrate a sustained arrhythmia.

Although antiarrhythmic drugs such as sotalol have proven useful in reducing the incidence of arrhythmia occurrences and ICD discharges, some antiarrhythmic drugs such as sotalol may reduce defibrillation thresholds, other agents (notably amiodarone and mexiletine) may elevate defibrillation thresholds; the resulting compromise of safety margin between the energy that is required and the energy that is deliverable for defibrillation may have disastrous consequences. Flecainide is particularly noteworthy for elevating pacing thresholds. Thus, the addition of antiarrhythmic agents to the medical program of a patient with a device warrants periodic attention to programmed settings and sensing/pacing/defibrillation thresholds.

Potential sources of EMI are ubiquitous, especially in the hospital environment and workplace; therefore, it is critical for the general cardiologist to be aware of potential risks to the device patient and precautions that may be warranted (Table 2). In the surgical arena, electrocautery may be oversensed by a patient’s intracardiac device, resulting in transient inhibition of pacer output or in triggering of inappropriate ICD shocks (Figure 6). Particularly in the pacemaker-dependent patient, devices should be programmed...
ICD systems may be MRI safe. Alternatively, certain chronic modes and maximal pacing output; selection of reprogramming of the intracardiac device, including asynchrony, inappropriate ICD discharges, rapid pacing, mechanical pull and rotation of the device, and device reversion to asynchronous pacing or result in pacer inhibition. However, it is frequently the case that no significant impact on thresholds, impedances, or device function is observed. Reprogramming a pacemaker to asynchronous mode, inactivating antitachycardia therapies, and reassessing the device after the procedure are recommended approaches for patients undergoing radiofrequency ablation.

Although less common now, patients may have both a pacemaker and a separate ICD device. Clinical interactions between the 2 may include transient failure by the pacer to sense or capture post-ICD discharge; oversensing of the pacemaker stimulus by the ICD, leading to double counting; ICD failure to sense VF, resulting from pacer stimulus oversensing; and pacemaker reprogramming, caused by ICD discharge. In ICD patients who require a separate pacemaker, a bipolar pacing system is indicated to preempt these sensing issues. Other devices may rarely trigger device discharge in an ICD patient, notably, transcutaneous electronic nerve-stimulation units.

In the nonmedical environment, it has been recognized that cellular telephones may interfere with the function of implanted devices, with the potential for any type of interference being 20% in one study with associated symptoms in 7.2% with the phone placed directly over the generator; the most common pacemaker interactions were tracking interference on the atrial channel, followed by asynchronous pacing and ventricular inhibition. Distancing the phone from the generator or using the contralateral ear is the recommended approach. Rarely, antitheft surveillance systems in retail centers may result in ICD discharge or pacemaker triggering. Patients should be advised to walk through the gates without leaning on them or lingering. When in doubt regarding the potential impact of any type of EMI, an inquiry with the manufacturer should be made.

Determinants of Frequent ICD Shocks and the Management of VT Storm

Electrical storm may be defined as recurrent VT or VF occurring 2 or more times in a 24-hour period and usually

![Figure 8. Demonstration of patient with atrial flutter, with atrial electrograms at cycle length of 200 ms, initially tracking every other flutter wave with triggered ventricular-paced beat (2:1 lock-in) (A), then temporary inhibition of pacing by programmer showing flutter waves (B), followed by automatic mode switch to ventricular demand pacing (C); upper tracing is surface recording, lower is atrial electrogram. MS indicates mode switch; AS, atrial sensed; AR, atrial refractory; and VP, ventricular paced.](image-url)
requiring electrical cardioversion or defibrillation.\textsuperscript{104} It is often difficult to determine whether a patient presenting with VT is manifesting a primary arrhythmogenic substrate or a secondary effect of worsening CHF.\textsuperscript{105} Clinical instability manifested as interim hospitalizations for CHF or coronary events, as identified in MADIT-II,\textsuperscript{106} and severe depression, as demonstrated in the Triggers of Ventricular Arrhythmias (TOVA) study,\textsuperscript{107} seem to correlate more strongly with recurrent VT. Also in the MADIT trial, electrophysiological inducibility of VT was associated with increased likelihood of VT, whereas noninducibility predicted a higher VF rate.\textsuperscript{108} Electrical storm typically occurs late after ICD implantation and may correlate with chronic renal failure and depressed EF.\textsuperscript{104,109} What is less clear is whether there is any difference in appropriate or inappropriate device therapy in primary versus secondary prevention ICD patients.\textsuperscript{110} Higher-risk patients may warrant prophylactic administration of antiarrhythmic drug therapy with agents such as amiodarone and \(\beta\)-blockers, as in the OPTIC trial\textsuperscript{87} or sotalol.\textsuperscript{89} When patients are actually manifesting frequent ICD shocks and/or

Figure 9. Twelve-lead electrocardiograms of patient with left bundle-branch block (A) and with biventricular pacing (B); note tall R wave in lead V1 during cardiac resynchronization therapy.
storm, immediate management entails reversal of electrolyte and/or metabolic abnormalities, intravenous administration of amiodarone and β-blockade, and addressing potential acute coronary syndromes in the case of polymorphic tachycardias. Slower VT may be totally asymptomatic or present with symptoms of either palpitations or CHF if the duration is prolonged and below the rate cutoff for device treatment. It may be readily approached by reprogramming the detection rate and incorporating antitachycardia pacing for treatment. Strategies for the management of incessant spurious device discharge include temporarily inactivating the device, rate control or cardioversion in the case of AF or SVT, and reprogramming such detection algorithms and rate cutoffs as previously outlined.

**Device Advisories**

Increased attention has been directed to the safety of implanted devices, particularly because death may result from device failure and/or complications; less commonly, death may result from unintended pulse-generator inactivation (without reactivation). System failure can be associated with lead malfunction, although there is growing concern regarding generator performance. The concept of a national database for tracking devices is not new, but most information regarding device survival and/or malfunction is still limited to industry performance reports of returned products and a meager collection of selected registries. Although pacemaker malfunction replacement rate has declined, ICD malfunction replacement rate has increased. Equally alarming are the risks inherent in replacement, whether warranted or excessive, of devices under advisory; a disturbing incidence of infections and deaths have been associated with system revision. Specific recommendations for clinicians managing device advisory notices are to consider device/lead replacement if the mechanism of malfunction is known and potentially recurrent, if death is a likely result of device malfunction, if the patient is pacemaker dependent, if the risk of replacement is substantially lower than the risk of device malfunction, and especially if the device is nearing its elective replacement indicators.

**Participation in Sports and Driving by the Patient With a Device**

The clinician will often be asked to counsel a patient as to what activities are allowable. Clearly, this will vary as a function of the patient, the patient’s underlying condition, the severity of the patient’s illness, and the potential impact of an activity (such as driving) on others. Physician recommendations to ICD patients regarding sports participation vary widely; one survey of electrophysiologists reported 76% recommending avoidance of contact and 45% recommending avoidance of competitive sports; in the same study, shocks were cited commonly, but adverse effects were rare. Nonetheless, the 36th Bethesda Conference task force strongly suggests that athletes with either primary or secondary prevention ICDs should be disqualified from all competitive sports except for those of low intensity; pacemaker-dependent patients are advised against participating in most competitive sports that potentially involve body trauma. The authors acknowledge that little direct evidence in this area is available; their concern is based on the uncertainties associated with device performance at peak exercise, the likelihood of triggering discharges from sinus tachycardia or

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![Figure 10](image1.png)

**Figure 10.** Assessment of thresholds, with initial capture of both right and left ventricle, followed by loss of capture on left ventricular lead, followed by biventricular loss of capture, with corresponding change in morphologies.

![Figure 11](image2.png)

**Figure 11.** Echocardiographic optimization of atrioventricular (AV) interval for cardiac resynchronization. An AV delay that is too long (160 ms) results in fusion of E and A waves, with reduction in filling time, prolongation of time between the end of the A wave and mitral valve closure, and presystolic mitral regurgitation; an AV delay that is too short (50 ms) results in separation of E and A waves, but the A wave is truncated by mitral valve closure, resulting in an interruption of atrial contribution to ventricular filling. The optimal AV delay should allow for completion of end-diastolic filling before ventricular contraction, resulting in the longest diastolic filling time. The end of the A wave should coincide with complete mitral valve closure; ventricular contraction should begin at the end of atrial contraction. E indicates wave associated with the early filling phase of transmitial flow; A, atrial contraction phase of transmitial flow.
other arrhythmias, and the potential for injury resulting from an ICD shock during competitive activity.

Patients presenting with impaired consciousness while driving a motorized vehicle, in the absence of intoxication or falling asleep, are frequently found to have arrhythmias as the putative cause, as identified by tilt-table testing and/or electrophysiological study. It is not uncommon for patients to have arrhythmia-type symptoms while driving; nonetheless, motor vehicle accidents are infrequent, even when associated with ICD discharge. Thus, in the AVID (Antiarrhythmics Versus Implantable Defibrillators) trial, 55 accidents were reported during 1619 patient-years of follow-up after resumption of driving by patients with documented sustained VT/VF (3.4% per patient-year), and only 11% of these accidents were preceded by symptoms of possible arrhythmia (0.4% per patient-year). Extensive recommendations regarding driving and related activities were published 1 decade ago. ICD patients with sustained VT or VF with structural heart disease were precluded from commercial driving but could resume private driving 6 months after device implantation if they were persistently free from ICD discharge. Pacemaker patients who were pacemaker dependent were only restricted from driving for the first week (private driving) or first month (commercial driving) after implantation. These recommendations predate the era of primary prevention ICD implantation, for which greater leniency may be anticipated; as such, the recommendations warrant updating and are, in any case, subject to the vagaries of local statutes. Of interest, when surveyed more than a decade ago, only 8 states in the United States had specific laws for patients with arrhythmias, and 74% of physician respondents (either general cardiologists or electrophysiologists) did not know their own state’s laws regarding driving by patients with ventricular arrhythmias.

Conclusion

The size and mobility of the patient population with implanted pacemakers and/or defibrillators is steadily increasing, as is the complexity associated with these devices. Recognizing that there are manpower limitations inherent in providing electrophysiological expertise to all these deserving patients, it has become abundantly clear that the general cardiologist must attend to a variety of clinical situations in which such expertise may not be immediately available. The present review addresses some of the nuances associated with device management.

Disclosures

A member of Dr Schoenfeld’s household is a shareholder for Boston Scientific.
References


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