Failure to Deliver Therapy or Delay of Therapy

Absent or delayed therapy may be caused by programmed values (including human error), ICD system performance, or a combination of the two. The most common causes are ICD inactivation, VT slower than the programmed detection interval, SVT-VT discriminators, undersensing, and intradevice software interactions. Pacemaker-ICD interactions, once important, are now rare due to incorporation of dual- and triple-chamber pacemakers in ICDs.

ICD Inactivation

If detection is programmed OFF for surgery using electrocautery, reprogramming must be performed at the end of the procedure, a fact easily forgotten, especially in outpatient surgery. One study reported an unexplained 11% annual incidence of transient suspension of detection.1 Medtronic ICDs sound an audible Patient Alert™ 6 hours after the ICD is programmed “off” to alert the patient to the possibility of inadvertent.2

VT Slower Than the Programmed Detection Interval

In most ICD patients, VT with cycle lengths >400–450 ms are tolerated well, while repeated inappropriate therapies are not. However, slow VT may be catastrophic in patients with severe LV dysfunction or ischemia.2 All SVT-VT discrimination algorithms (except ELAs)3–5 deliver less inappropriate therapies if the VT detection interval is programmed to a shorter cycle length so that fewer SVTs are evaluated. To prevent underdetection of irregular VT, the VT detection interval should be set at least 40–50 ms longer than the slowest predicted VT for consecutive-interval counting and 30–40 ms longer for X-of-Y or interval + interval-average counting.5 It should be a longer cycle length if rapidly conducted SVT is unlikely or SVT-VT discrimination is reliable at long cycle lengths (ELA ICDs). A long VT detection interval is important in patients with advanced heart failure, in whom slow VT may be catastrophic.2 (see figure 8 in Part I of this review). The VT detection interval should be increased if antiarrhythmic drug therapy is initiated, particularly with amiodarone or a sodium-channel blocking (Type 1) drug.5,7 It may be prudent to measure the cycle length of induced VT at electrophysiological testing after initiation of drug therapy.5 However, spontaneous VT often is slower than induced VT.8

SVT-VT Discriminators

SVT-VT discriminators may prevent or delay therapy if they misclassify VT or VF as SVT.9–12 Discriminators that reevaluate the rhythm diagnosis during an ongoing tachycardia, such as stability and most dual-chamber algorithms, reduce the risk of underdetection of VT compared with discriminators that withhold therapy if the rhythm is not classified correctly by the initial evaluation, such as onset or chamber of origin. The minimum cycle length for SVT-VT discrimination should be set to prevent clinically significant delay in detection of hemodynamically unstable VT.

Single-Chamber Discriminators

Although spontaneous VT often begins with irregular R-R intervals, stability algorithms classify it as VT as soon as R-R intervals regularize, even transiently. Thus, they rarely prevent detection of VT (~0.5%) if they are programmed to nominal settings and the patient is not receiving an antiarrhythmic drug that decreases cycle length regularity in VT9,13 (see “Antiarrhythmic Drugs”). Previously, we noted that single-chamber stability may be programmed in Medtronic dual-chamber ICDs to reject atrial fibrillation during redetection. In this case, stability is applied before the dual-chamber algorithm. Because stability responds to irregular rhythms by resetting the VT counter to zero, the dual-chamber algorithm does not evaluate rhythms rejected by stability. Thus, stability may delay detection of VT even if the ventricular rate is greater than the atrial rate.

Morphology discriminators also reevaluate rhythm diagnosis during ongoing tachycardias. But if they misclassify monomorphic VT initially, the error usually persists and prevents detection for the duration of the tachycardia. The St. Jude morphology algorithm, which analyzes only the rate-sensing electrode, continuously misclassifies
5–10% of monomorphic VTs as SVT. But, when it is restricted to tachycardias with ventricular rate ≤ atrial rate in dual-chamber ICDs, only 2% of VTs are misclassified.11 The Medtronic morphology algorithm, which usually analyzes high-voltage electrograms, misclassifies 1–2% of VTs as SVTs.14 In contrast, the onset discriminator uniformly misclassifies VT if it either accelerates gradually across the sinus-VT zone boundary or occurs during SVT with cycle length in the VT zone.

**Dual-Chamber Discriminators**

If the atrial lead dislodges to the ventricle, the atrial channel records a ventricular electrogram. Any VT will appear to the ICD as a tachycardia with 1:1 AV relationship and nearly simultaneous atrial and ventricular activation. Some discriminators designed to withhold therapy from SVT with 1:1 AV conduction may then withhold appropriate VT therapy. These include the Medtronic’s PR Logic™, High Rate Discriminator override should be based on clinical factors including the probability that discriminators will prevent detection of VT, the likely consequences of failure to detect VT, and the likelihood of sustained VT in the VT rate zone. For example, override features should be considered whenever a morphology algorithm is programmed without inducing VT at electrophysiologic study. The programmed duration in Guidant Atrial View™ and St. Jude algorithms should be increased from the nominal value of 30 seconds to reduce inappropriate therapies. In most patients, a duration of 2–5 minutes is reasonable.

**Undersensing**

VF may be undersensed due to combinations of programming (sensitivity, rate, or duration), low-amplitude electrograms, rapidly varying electrogram amplitude, drug effects, or post-shock tissue changes. Clinically significant undersensing of VF is rare in modern ICD systems if the baseline R-wave amplitude is ≥5–7 mV.16 Post-shock undersensing was an important clinical problem in older ICD systems using integrated-bipolar leads with closely spaced electrodes.12 Currently, the most common causes of VF undersensing are drug or hyperkalemic effects that slow VF into the VT zone, ischemia, and rapidly varying electrogram amplitude.4,18 ICDs that adjust dynamic range based on the amplitude of the sensed R-wave (Guidant) may be most vulnerable to the latter, extremely rare problem.18 Prolonged ischemia from sustained VT slower than the VT detection interval may cause deterioration of signal quality, resulting in undersensing of VF. Lead, connector, or generator problems may also present as undersensing.

**Pacemaker-ICD Interactions**

Interactions between ICDs and separate pacemakers have become rare since ICDs incorporated dual-chamber bradycardia pacing in the late 1990s. Nevertheless, a few combined systems have not been revised due to vascular access problems or other reasons. The multiple potential interactions have been reviewed and testing protocols to detect them have been developed.19–21 The principal interaction that may delay or prevent ICD therapy is oversensing of high-amplitude pacemaker stimulus artifacts. If this occurs during VF, repetitive automatic adjustment of sensing threshold and/or gain may prevent detection of VF.

**Intravehicle Interactions**

Today, “intra-device interactions”—in which bradycardia pacing features of dual-chamber ICDs interact with and impair detection of VT or VF—pose a greater challenge than pacemaker-ICD interactions.22 During high-rate, atrial or dual-chamber pacing, sensing may be restricted to short periods of the cardiac cycle because of the combined effects of ventricular blanking after ventricular pacing and cross-chamber ventricular blanking after atrial pacing, which is needed to avoid cross talk. If a sufficient fraction of the cardiac cycle is blanked, systematic undersensing of VT or VF may occur. When pacing and blanking events occur at intervals that are multiples of a VT cycle length,
Figure 1. Failure to detect VT due to an intradevice interaction. The rate-smoothing algorithm introduced atrial and ventricular pacing complexes with associated blanking periods that prevented detection of VT during post-implant testing. An external rescue shock was required. Shown from top to bottom are surface ECG, atrial electrogram, ventricular electrogram, and event markers. At top VT is induced by programmed electrical stimulation with drive cycle length 350 ms and premature stimuli at 270, 250, and 230 ms (intervals labeled next to event markers). The first sensed ventricular event occurs 448 ms after the pacing drive (“PVC 448”). The rate smoothing algorithm drives pacing to prevent a pause after the “premature ventricular complex” (PVC), labeled AP↓1638. A ventricular-paced event does not follow the first AP↓ because a ventricular event is sensed (VT 415). Subsequent rate smoothing generated atrial and ventricular pacing pulses (indicated by AP↓ and VP↓ markers, respectively). The resultant post-pacing blanking periods are shown in the figure as horizontal bars. PAB denotes cross-chamber (post-atrial-pace) ventricular blanking period. VBP denotes same-chamber (post-ventricular-pace) blanking period. Together, they prevent approximately 4 of every 6 VT complexes from being sensed. Since the VT counter must accumulate 8 out of 10 consecutive complexes in the VT zone for detection of VT to occur, VT is not detected.

Ventricular complexes are repeatedly undersensed, delaying or preventing detection (see Fig. 1).22–24 Although uncommon, intradevice interactions have been reported most frequently with the use of the Rate Smoothing™ algorithm in Guidant ICDs.22–24 This algorithm is intended to prevent VT/VF initiated by sudden changes in ventricular rate.25 It prevents sudden changes in ventricular rate by pacing both the atrium and ventricle at intervals based on the preceding (baseline) R-R interval. As an unintended consequence, it may prevent sensing of VT/VF in some patients because it introduces repetitive post-pace blanking periods. The algorithm applies rate-smoothing to baseline intervals independent of their cycle length, including intervals in the VT or VF zones. Intra-device interactions that result in delayed or absent detection of VT/VF are most common and most dangerous when VT is fast. The parameter interrelationships that result in delayed or absent detection of VT/VF are complex and difficult to predict, but they usually elicit a programmer warning. Generally, aggressive rate-smoothing (a small allowable percentage change in R-R intervals), a high upper pacing rate, a long and fixed AV interval favor undersensing and should be avoided. If rate-smoothing is required, parameter settings that result in warnings should be avoided by programming a dynamic AV delay, limiting the upper rate to less than 125 beats/min, and shortening ventricular blanking after atrial-paced events to maximize the sensing window for VT/VF. This programming reduces, but does not eliminate, the risk of undersensing.22–24,26

Unsuccessful Therapy

Because defibrillation success is probabilistic, occasional shocks fail, but failure of more than two maximum-output shocks is rare if the safety margin is adequate.27 If an ICD classifies a shock as unsuccessful, stored electrograms must be reviewed.
to determine both if the shock was delivered for true VT/VF and if the shock actually failed to terminate VT/VF. Shocks from chronic ICD systems that defibrillated reliably at implant may fail to terminate true VT or VF because of patient-related or ICD system-related reasons. The problem of high-implant DFTs has been reviewed. In chronically ICD system-related reasons. The problem of high-implant DFTs has been reviewed.3,4 In chronically implanted systems, most patient-related causes of unsuccessful shocks can be reversed, but most system-related causes require operative intervention.

Misclassified Therapy

ICDs misclassify effective therapy as ineffective if VT/VF recurs before the ICD determines the VT/VF episode as terminated and reclassifies the post-therapy rhythm as sinus. Decreasing the duration for redetection of sinus rhythm (St. Jude) might correct this classification error. However, this type of misclassification usually does not constitute a clinical problem, while post-shock detection of nonsustained VT does. An exception occurs when one iteration of antitachycardia pacing is programmed for the first therapy and a shock for the second. If antitachycardia pacing terminates VT, but it recurs before the rhythm is classified as sinus, the recurrent VT will receive a shock instead of potentially effective antitachycardia pacing. Programming multiple iterations of antitachycardia pacing can mitigate this problem. However, the “Smart Mode” in Medtronic ICDs (nominally “on”) disables antitachycardia pacing if it is classified as unsuccessful on four consecutive episodes. Thus, if successful antitachycardia pacing is misclassified repeatedly, Smart Mode will disable it. In this setting, Smart Mode should be programmed off.

ICDs may also misclassify shocks as ineffective if the post-shock rhythm is SVT in the VT rate zone (catecholamine-induced sinus tachycardia or shock-induced atrial fibrillation). Solutions include applying SVT-VT discriminators to redetection of VT, increasing shock strength to prevent shock-induced atrial fibrillation, or administering β-blockers to slow post-shock SVT.

Patient-Related Factors

Factors that raise DFTs reversibly at the cellular level include hyperkalemia, antiarrhythmic drugs, and ischemia. Pleural or pericardial effusions raise the DFT by forming parallel intrathoracic current paths that shunt current away from the heart. Progressive cardiac enlargement or new myocardial infarction may also raise the DFT and are less easily reversed. Usually, defibrillation testing should be performed to confirm reliable defibrillation after the suspected cause has been reversed.

A few patient-related causes that would otherwise require operative revision may be resolved by programming shock pathway and waveform parameters. For ICDs with fixed-tilt waveforms, waveform duration depends on output capacitance and pathway resistance.28 ICDs with programmable waveform duration or tilt (St. Jude) permit optimization of waveform parameters independent of pathway resistance. Shortening the entire waveform might reduce DFT.29 Shortening phase-two might reduce DFT after amiodarone therapy is started.30 Migration of an active-can pulse generator low on the chest wall can increase DFT by altering the shock vector. This may be resolved by excluding the pulse generator from the shock circuit, which may be done noninvasively in Medtronic ICDs.

ICD System-Related Reasons

ICD-related causes include insufficient programmed shock strength, battery depletion, generator component failure, lead failure, device-lead connection failures, and lead dislodgment. In evaluating data from the ICD interrogation, attention should be paid to arrhythmia duration, electrograms from the shocking electrodes, charge time, battery voltage, the relationship between programmed and delivered shock strength, impedance of the high-voltage lead at the time of therapy is started.30 Migration of an active-can pulse generator low on the chest wall can increase DFT by altering the shock vector. This may be resolved by excluding the pulse generator from the shock circuit, which may be done noninvasively in Medtronic ICDs.

Problems Identified at Routine Follow-Up

Battery Depletion

Battery Discharge Curves—Relationship to Elective-Replacement and End-of-Service Indicators

ICDs use batteries composed of lithium anodes and silver vanadium oxide (AgV4O11) cathodes31 in which reduction at the cathode occurs in multiple steps. The corresponding discharge characteristics and battery behavior during capacitor charging have been a source of confusion among clinicians familiar with the slow, constant voltage decline typical of lithium-iodine pacemaker batteries. Initially, the cathodal contribution to battery voltage is due to two simultaneous reactions corresponding to reduction of $V^{5+}$ and $Ag^{+}$, where $V^{5+} + e^{-} \rightarrow V^{4+}$ and $Ag^{+} + e^{-} \rightarrow Ag$ (unloaded). Unloaded cell voltage falls from about 3.2 to 3.1 V at about 30% battery depletion. In most ICD batteries, voltage then falls rapidly to about 2.6 V, where it reaches a plateau until the battery is 80–90% depleted. This
voltage decrease is caused by a combination of factors, including build-up of inert material at the cathode and depletion of the $V^{+5}$ valence state of vanadium so that the cathodal half-cell voltage reflects reduction of $V^{+4} (V^{+4} + e^{-} \rightarrow V^{+3})$. The 2.6-V plateau is nominal performance for most (but not all) ICD batteries. End-of-service indicators correspond to either an unequivocal reduction in unloaded voltage about 0.1 V below this plateau or an excessive increase in charge time.

Premature Battery Depletion

The most common cause is excessive external power drain due to pacing or capacitor charging. Pacing problems include unnecessary ventricular pacing, high pacing outputs, and lead insulation failures. The most common cause of asymptomatic premature battery depletion related to capacitor charging is repeated aborted shocks due to repetitive nonsustained VT or oversensing due to lead-connector problems. Repeated shocks due to VT storm might also rapidly deplete the battery. Some diagnostic features have high power consumption. In Medtronic ICDs, “prestorage” of electrograms prior to each VT/VF episode results in continuous amplification of the unfiltered electrogram, substantially reducing longevity. In contrast, prestorage in Guidant and St. Jude ICDs, which store only filtered electrograms, does not cause significant battery depletion. ICD component failure is a rare cause of battery depletion.

Lead Problems

Ventricular lead problems are the most common cause of ICD system malfunction.32–35 They usually present either as oversensing, resulting in inappropriate shocks, or as abnormal diagnostic measurements at routine follow-up. Presentation as undersensing during VF or unsuccessful therapy is uncommon but often fatal. Clinical and lead-design factors identify patients at high risk for ventricular lead problems and merit intensified follow-up. Noninvasive assessment is usually diagnostic.36 The introduction of remote ICD monitoring,37,38 which permits transmission of complete ICD interrogation via telephone or the internet, may enhance noninvasive identification of subclinical lead failure (Fig. 2). Patients with Medtronic ICDs may present with audible “Patient Alert” warnings (see below).

In ventricular ICDs, atrial lead malfunctions may present as failure to discriminate VT from SVT. Atrial lead dislodgment to the ventricle might also result in inappropriate shocks caused by ventricular oversensing of noise generated by contact between the leads (Fig. 3).

Diagnosis of Lead Failure

A systematic approach to the patient with suspected lead failure requires evaluation of electrograms from all electrodes on the lead, pacing threshold, pacing impedance, and painless high-voltage electrode impedance; system radiography; stored episode electrograms, data logs, and flash-back memory (extended recording of R-R intervals in Medtronic ICDs).36 The yield of these tests is shown in Table I. The Sensing Integrity Counter™ (Medtronic) displays the number of nonphysiologic short intervals detected in the 120–140-ms range. With intact, true-bipolar leads, the count remains at zero. With intact, integrated-bipolar leads, the large proximal sensing electrode (the distal ICD coil) permits occasional oversensing of diaphragmatic myopotentials, but a count >300 over 3 months suggests a lead problem.39

Pacing Impedance

The normal value for pacing impedance is a function of lead design; but for any lead (except Y-adapted lead combinations), impedance $<200 \Omega$ indicates an insulation defect and impedance $>2,000 \Omega$ suggests a conductor failure if a loose set screw or faulty adapter are excluded.40,41 Modern ICDs perform serial impedance measurements automatically. The range of variability of pacing lead impedances has been studied for coaxial pacemaker leads,42 but corresponding data for coaxial and multi-lumen defibrillation electrodes are limited. Generally, conductor failures do not present with moderate changes in impedance. Either the impedance exceeds 2,000 Ω (continuously or intermittently), or it is normal, and conductor failure is diagnosed by identification of a characteristic pattern of oversensing. Pacing lead impedance is insensitive to inner insulation failure of Medtronic 6936 leads.41 Isolated pacing insulation failure of multi-lumen leads is sufficiently rare that the sensitivity of pacing lead impedance is unknown. Inner insulation failure in Medtronic 6936 or 6966 leads may be detected by a fall in ring-coil impedance with normal pacing impedance.41 Review of ring-coil impedance may require sending the programmer’s “save-to-disk” file to the manufacturer for analysis by a specialized software.

High-Voltage Impedance

Shocking-pathway impedance normally is 25–75 Ω in transvenous systems. Values outside this range suggest a conductor defect (high impedance) or insulation failure/short circuit
Asymptomatic malfunction in coaxial defibrillation lead (Medtronic model 6936) detected by internet-based, remote patient monitoring (Medtronic CareLink®). The Sensing Integrity Counter provides a cumulative count of nonphysiologic short intervals caused by oversensing. Multiple episodes of nonsustained VT with cycle lengths 140–210 ms also suggest oversensing. In the absence of exposure to electromagnetic interference, this is highly suggestive of an inner-insulation failure. The pacing and defibrillation lead impedances are insensitive to early insulation failure. Analysis of ring-coil impedance from the programmer “save-to-disk” file is often diagnostic. See text. Courtesy of Physician Assistant Sheila Reynolds and Dr. Dwight Reynolds.

Electrogram Amplitude (R-Wave, P-Wave) and Pacing Threshold

The amplitude of the R-wave during sinus rhythm should be at least 5–7 mV for assurance that the low-amplitude electrograms during VF will be sensed reliably. Causes of reduced electrogram amplitude include antiarrhythmic drugs, myocardial infarction, shocks, fibrosis at the lead
Figure 3. Atrial lead dislodgment causing inappropriate shock. Top right box shows the episode summary, indicating an episode detected as VF (via the combined count), for which the first VF therapy was delivered. The interval summary plot (middle panel) demonstrates a regular “atrial” rate (boxes) of 640 ms, with a highly variable ventricular cycle length (black circles), leading to detection and delivery of a 34.6-J shock. The bottom panel shows the atrial electrogram (Atip to Aring), near-field ventricular electrogram (Vtip to Vring), calculated A-A intervals, event markers, and calculated V-V intervals. Each ventricular electrogram is associated with an atrial-channel electrogram that has large amplitude and slope. The sensing of ventricular events on the atrial channel in the absence of sensed atrial events suggests atrial lead dislodgment to the ventricle. The presence of smaller deflections on the ventricular electrogram (the first is sensed as “VS 550”) is consistent with oversensing of “noise” signals caused by contact with the atrial lead. This oversensing results in detection of VF and delivery of shocks during normal rhythm. AR = atrial refractory sense; VS = ventricular sense; TS = tachycardia sense (in VT zone); VF = fibr sense (in VF zone).

Radiography

Abnormal radiographic features are identified in less than half of patients with ICD lead malfunctions. Lead dislodgment is diagnosed when the lead tip is in a different position than in the post-operative radiograph, but variability due to cardiac and respiratory motion may prevent diagnosis of minor dislodgments. Typically, RV apical leads dislodge toward the tricuspid valve; atrial leads dislodge into the RV; and coronary venous leads of resynchronization ICDs dislodge proximally into the coronary sinus or right atrium.

Subclavian crush occurs when a lead is compressed in the narrowly confined space between the first rib and clavicle; this common site of fracture requires careful examination if lead failure is suspected (Fig. 4). Radiography may also detect conductor defects, inadequate pin insertion into the header, abandoned or incompletely removed leads, or torsion due to Twiddler’s syndrome. However, some intact electrical connections in leads and adapters appear radiographically to have conductor discontinuity (“pseudo fracture,” Fig. 5). Fluoroscopy of an unopened lead or adapter (if available) can be used to determine the expected radiographic appearance.

Real-Time Telemetry

Real-time display of intracardiac electrograms is used to assess the effect of body position and
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TABLE I.
Detection of Malfunction in Transvenous ICD Leads

<table>
<thead>
<tr>
<th>Test Sensitivity*</th>
<th>Asymptomatic Patients (n = 11)</th>
<th>Patients with Inappropriate Shocks (n = 7)</th>
<th>All Patients with Lead Failures (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stored electrograms</td>
<td>27%</td>
<td>100%</td>
<td>56%</td>
</tr>
<tr>
<td>X-ray</td>
<td>27%</td>
<td>29%</td>
<td>28%</td>
</tr>
<tr>
<td>Pacing threshold</td>
<td>36%</td>
<td>14%</td>
<td>28%</td>
</tr>
<tr>
<td>R-wave amplitude</td>
<td>55%</td>
<td>0%</td>
<td>6%</td>
</tr>
<tr>
<td>Pacing impedance</td>
<td>9%</td>
<td>0%</td>
<td>6%</td>
</tr>
<tr>
<td>High-voltage lead impedance</td>
<td>29%</td>
<td>14%</td>
<td>43%</td>
</tr>
<tr>
<td>Defibrillation threshold</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Combination of tests</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All pacing/sensing tests</td>
<td>82%</td>
<td>14%</td>
<td>78%</td>
</tr>
<tr>
<td>Pacing/sensing, stored electrograms and x-ray</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Sensing and detection of induced VF</td>
<td>29%</td>
<td>14%</td>
<td>43%</td>
</tr>
</tbody>
</table>

Modified from Luria et al.36

maneuvers on recorded signals. The appearance of noise during Valsava or inspiration may be caused by myopotential oversensing49 or a structural lead defect. Abnormalities of pacing function, R-wave amplitude, pacing and shocking impedance, and stored evidence of oversensing39 usually indicate lead defect. Simultaneous surface ECG recording with telemetry of the sensing signal, the far-field electrogram (between shocking electrodes), and sensing/pacing markers may permit localization of the malfunction to a specific component of the lead. Medtronic ICDs permit use of special telemetry Holter monitors for troubleshooting.50 They provide a simultaneous, 24-hour recording of a surface ECG lead with two intracardiac electrograms and an extended Marker Channel™ (Fig. 5).

Figure 4. Subclavian crush injury to defibrillation electrode. (A) Note the sharp bend on the lead at the site where it passes between the subclavian vein and the first rib (arrow). (B) Lead extracted from a patient with subclavian crush. Note the disrupted insulation and the disarray of the fillers (the circumferentially coiled conductor) at the site of crush. Reprinted with permission from Lloyd et al.46
Figure 5. Oversensing identified by telemetry Holter monitor. Telemetered ECG and ventricular integrated-bipolar and high-voltage electrograms are recorded with extended marker channel showing usual markers (top line) and multiple other device parameters. The sixth line shows the VF counter which increments from 0 to 3 during oversensing. Saturation of the sensing electrogram and intermittent oversensing are characteristic of electrode problems, in this case intermittent conductor failure. The Holter was performed because the Short Interval Counter was abnormal. In this case, the lead impedance was normal.

Stored Episodes

Lead, adapter, or set screw malfunction often presents as oversensing, resulting in inappropriate detection of VF and aborted or inappropriate shocks. This results from make-break contact noise or oversensing of extracardiac signals due to insulation defects. The pattern of oversensing often includes extremely short nonphysiological intervals (<140 ms, Fig. 6). In Medtronic ICDs, the combination of a high Sensing Integrity CountTM of nonphysiological intervals, nonsustained tachycardia episodes with nonphysiological intervals, and abnormal lead impedance triggers a lead alert warning on interrogation.39

Patient Alerts

Medtronic ICDs emit patient-alert tones for pacing or high-voltage lead impedances out of range, low battery voltage, long charge time, three shocks delivered per episode, or all therapies in a zone delivered. Most serious problems identified by alerts are lead-related40 (Fig. 6). Unfortunately, ICD recipients may not hear alert tones or may not recognize their significance. Alerts are a useful adjunct to, but not a substitute for, periodic device interrogation and follow-up.

Risk Factors for Lead Failure

Clinical factors predictive of lead failure include epicardial leads,34,51 abdominal pulse generator location,36,47 coaxial defibrillation leads,36,41 subclavian venous access,48,52 and dual-chamber (as opposed to single-chamber) ICD systems.52 In epicardial systems, sensing and defibrillation are performed by different leads, eliminating oversensing as an early warning for failure of defibrillation leads. Thus, periodic assessment of shocking lead impedance is essential. Currently, epicardial
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Figure 6. Conductor failure identified by lead alert. Top panel shows marked, abrupt increases in all measured impedance values triggering a lead alert on July 28, 2 days after the patient was seen in clinic. The patient did not hear the alert tones, probably because the ICD was implanted submuscularly. Middle panel shows episode summaries from inappropriately detected episodes of VF during intense stationary bicycle exercise in “spinning” class (August 1). Bottom panel shows the corresponding stored electrogram from the true-bipolar sensing lead. Signals are probably caused by mechanical motion at the fracture site. Saturation of rate sensing electrogram is characteristic of conductor failure; but persistence of oversensing throughout the cardiac cycle is not and is probably caused by intense exercise.

systems are used only in patients with a univentricular heart or mechanical tricuspid valve. Subcutaneous patches, which have similar construction to epicardial patches and have similar high failure rates, also warrant careful observation. Abdominal pulse generator placement, independent of epicardial or transvenous approach, may cause lead stress associated with tunneling and mechanical friction of the pulse generator against the lead. Cardiac resynchronization leads have a higher dislodgment rate than other electrodes.

Adapters increase the number of mechanical connections and stress points and may increase the risk of system malfunction. A loose set screw (in the header or an adapter) can mimic lead fracture, with make-break contact noise and elevated impedance.

Coaxial leads have higher failure rates than multi-lumen leads, in which conductors are longitudinally arrayed along the length of the lead. Medtronic Transvene coaxial leads (models 6936 and 6966) are prone to failure caused by metal ion oxidation of a middle insulation layer. A drop in the ring-to-coil impedance is an early marker of this failure, which results in oversensing of nonphysiological signals. Use of ring-to-coil impedance, the nonsustained episode log, and Sensing Integrity Counter may permit identification of early failure before oversensing causes inappropriate shocks.

Protecting ICDs from High-Voltage, Short Circuits

Low impedance on the shock electrodes indicates an insulation failure that diverts shock current from the heart. It may produce sufficient peak current that the ICD’s output circuit fails by “electrical overstress.” For example, an insulation failure in the pocket may produce a short circuit from the high-voltage coil to the can with a resistance of 8 Ω. A maximum-output (~800 V) shock would result in a peak current of 100 A in the high-voltage output circuit, causing it to fail catastrophically.
Figure 7. Post-shock oversensing. Panels show recordings of true-bipolar sensing and marker channel during VF induced at replacement of an ICD pulse generator attached to a chronically implanted Medtronic model 6936 Transvene™ coaxial defibrillation electrode. Top and middle panels are continuous. At left of top panel, T-wave shock (CD) induces VF, which is reliably sensed, detected, and terminated by a programmed 20-J shock (CD 19.6 J) in the middle of the middle panel. Post-shock intermittent oversensing saturates electrogram signals, resulting in failure to identify sinus rhythm and repetitive inappropriate redetection of VF, with multiple shocks aborted using the programmer. The lower panel shows one aborted shock with detection of VF (FD). The short charge time between FD and CE markers is caused by high residual voltage output capacitor after previous aborted shocks. Post-shock oversensing is the typical early sign of inner insulation failure in this coaxial defibrillation electrode.

intracardiac electrodes of opposite polarity can cause the same effect. To prevent electrical-overstress component failure, modern ICDs protect the output circuit by aborting the shock pulse immediately if the current in the defibrillation pathway is excessive. Depending on the manufacturer, this corresponds to a series lead impedance of 15–20 Ω. The presumption is that the short circuit diverts current from the heart, preventing shocks from terminating VT/VF. In addition to preserving the generator, this feature has a safety benefit. Simultaneous insulation failure of the rate-sensing and high-voltage components of RV leads may present as oversensing. Diverting the resultant inappropriate shock prevents unnecessary pain. Further, it may prevent fatal proarrhythmia from a weak shock delivered in the vulnerable period with no effective means to rescue the patient60 (see Fig. 8).

Pulse Generator Failure
Implantable ICD pulse generator failure is rare, although the incidence is largely unknown due to limitations in current reporting methods.61 Pulse generator failure might result from primary semiconductor component failure caused by exposure to extreme voltages or currents (electrical overstress). It can occur due to inappropriate internal arcing, commonly associated with the hybrid circuit.58 In older ICDs without shorted high-output protection, pulse generators fail if shocks are delivered into a short circuit resulting from lead insulation failure.
Figure 8. Protection for shorted output of high-voltage circuit. Text therapy summary indicates sequence of therapies after induction of VF by a 0.6 J T-wave shock (T-shock) during replacement of an ICD pulse generator attached to a chronically implanted Guidant Model 00125 defibrillation electrode. The measured high-voltage lead impedance is $< 20 \Omega$. Despite programmed shocks strengths of 15, 20, and 30 J, delivered energies are 0.2, 0.3, and 0.3 J, respectively, because of shorted output protection feature. Short charge times for second and third shocks correspond to high residual energy on capacitors at end of previous shock. Top left panel shows interval plot. Top right panel shows third unsuccessful shock (followed by minimum distortion of post-shock, high-voltage electrogram due to low voltage) and external rescue. An insulation failure in the pocket shorted the high-voltage lead to the generator can.

Pulse generator failure may manifest as absence of telemetry, emission of device tones, inappropriate shock, premature battery depletion, display of fault codes upon interrogation, inability to interrogate or program, failure to charge the capacitors or retain a charge, early battery depletion, or failure to deliver therapy. Failed pulse generators should be explanted and returned to the manufacturer for analysis, and the incident should be reported to the Food and Drug Administration's Manufacturer and User Facility Device Experience database (http://www.fda.gov/cdrh/maude.html). Rigorous reporting and analysis may distinguish random component failure from a systemic failure, correctable by design or manufacturing modifications before many patients are affected.

Considerations for Bradycardia Pacing in ICDs

To prevent intradevice interactions (see “Pacemaker-ICD Interactions”), dual-chamber ICDs have interlocks between pacing and VT/VF detection parameters that limit the fraction of the cardiac cycle affected by pacing-related blanking periods. These interlocks limit combinations of the upper rate limit and AV delay based on the VT detection interval. Adaptive, rate-related shortening of the post-pacing ventricular blanking period permits higher pacing rates with less ventricular blanking, but might increase the risk of T-wave oversensing.

In patients with LV dysfunction, RV apical pacing promotes ventricular dyssynchrony, atrial fibrillation, and heart failure. Dual-chamber ICDs should be programmed to minimize ventricular pacing in this population. One approach is to use a nontracking pacing mode (DDI) and a long AV interval. However, this may introduce parameter interlocks or—in conjunction with ratesmoothing—intradevice interactions resulting in undersensing of VT/VF. Specific algorithms that promote intrinsic conduction periodically determine whether intrinsic conduction is present during dual-chamber pacing. They either prolong the AV interval (Search Hysteresis™ in Guidant ICDs; AutoIntrinsic Conduction Search™ in St. Jude) or change the pacing mode to a modified
SWERDLOW AND FRIEDMAN

Figure 9. Algorithm for maximizing intrinsic AV conduction. (A) Hospital telemetry strip. The first four complexes demonstrate atrial pacing with intrinsic ventricular conduction in a patient with MVP\textsuperscript{TM} Mode programmed in a Medtronic ICD. The fifth atrial-paced beat is not conducted, followed by AV pacing on the sixth beat. This algorithm paces in the AAI mode with ventricular surveillance, to minimize ventricular pacing. Ventricular pacing occurs only if a nonrefractory sensed or paced atrial event is not conducted (allowing for a maximum pause of two times the lower rate limit plus 80 ms). Nominal performance of this algorithm may be confused with pacemaker malfunction. (B) From top to bottom: surface ECG, electrogram markers, and near-field ventricular electrograms. The first three complexes depict DDDR pacing mode with MVP\textsuperscript{TM} Mode programmed on. The algorithm periodically checks for the return of AV conduction to minimize ventricular pacing (seen in the fourth complex, AS-VS). AS = atrial sense; VS = ventricular sense; VP = ventricular pace.

AAI mode with ongoing assessment of AV conduction (AAI+ or AAIR+ mode, Medtronic MVP\textsuperscript{TM}). The AAIR+ mode eliminates interlocks between the pacing upper rate limit and VT zone, permitting paced rates in the VT zone. In the AAIR+ mode, early ventricular events reset the VA interval and ventricular pacing does not occur at a fixed or dynamic AV delay. The AAIR+ mode thus eliminates interlocks to permit rate-responsive pacing in the VT zone without the risk of intradevice interactions that may prevent detection of VT by competitive bradycardia pacing\textsuperscript{22–24} (see “Pacemaker-ICD Interactions”). Because the PR interval is not limited, a single nonconducted P-wave may occur (see Fig. 9). A potential risk of this pacing mode is ventricular proarhythmia caused by pause-dependent VT.

Troubleshooting Cardiac Resynchronization Therapy in ICDs

The vast majority of cardiac resynchronization pacing in the United States is performed using ICDs.\textsuperscript{65–67} The two widely spaced ventricular leads in resynchronization ICDs introduce novel intradevice interactions between resynchronization pacing and detection of VT/VF. We address their unique troubleshooting implications. Optimizing pacing for hemodynamic benefit\textsuperscript{68} is beyond the scope of this review.

Ventricular Sensing Problems

Early, resynchronization systems utilized adapters that have multiple modes of pacing and sensing malfunction.\textsuperscript{68,69} The first dedicated system (Guidant Contak CD\textsuperscript{TM}) sensed between the LV and RV electrodes to determine R-R intervals. This “extended bipolar” sensing configuration “merges” events sensed in the LV or RV into a single recording channel. During pacing, RV and LV depolarizations are synchronized and refractory periods prolonged, preventing double-counting. But during conducted rhythms or VT, R-wave double-counting occurs whenever the interval between local electrograms of conducted beats
Figure 10. Multiple shocks in a patient with a cardiac resynchronization ICD with extended bipolar sensing (Contak C™) due to double counting initiated by a pacemaker-mediated tachycardia prevention algorithm. Electrograms from top to bottom are atrial, ventricular near-field, and ventricular far-field. The initial rhythm is sinus tachycardia, with P-synchronous pacing at the maximum tracking rate (“VP-MT”). This algorithm periodically extends the post-ventricular atrial refractory period during upper-rate limit pacing to abort a pacemaker-mediated tachycardia (indicated by “PMT-B”). The following atrial-sensed event falls in the extended post ventricular atrial refractory period (indicated by the parenthesis, “(AS)”) and is therefore not tracked. Cessation of ventricular pacing permits double counting of the intrinsic-wide QRS complex by extended bipolar sensing, leading to inappropriate detection of VF. VS = ventricular sense in sinus zone. Other abbreviations as in Figure 11.

R-wave double-counting may result in shocks for SVT slower than the programmed VT detection interval because alternate device-detected intervals correspond to (1) the interval between the two sensed components of the ventricular electrogram and (2) the difference between the true ventricular cycle length and the double-counting interval. See Figure 6 for details. Because alternate “R-R” intervals in any conducted rhythm increment the VF counter, all detected VT or SVT episodes are classified as VF and treated with shocks, regardless of cycle length. Because there are two detected intervals for each ventricular electrogram, transient nonsustained tachycardias may satisfy the VF detection criterion, resulting in aborted shocks. Pacemaker algorithms—such as those designed to terminate pacemaker-mediated tachycardia—may promote inappropriate shocks by temporarily suspending ventricular pacing and permitting double-counting (Fig. 10).

Extended bipolar ventricular sensing also increases the risk of oversensing P-waves, most commonly when the LV lead dislodges into the coronary sinus. This may inhibit pacing, preventing resynchronization therapy for heart failure, or cause ventricular asystole in patients with complete heart block. Oversensing of atrial arrhythmias may cause inappropriate shocks despite a slow ventricular rate. Oversensed events may have complex and unanticipated interactions with detection enhancements (Fig. 11).

LV Threshold and RV Anodal Capture

Even if they use RV sensing, most ICDs use dual-cathodal (“extended bipolar”) pacing in which the LV and RV tip electrodes serve as the combined cathode. The common anode may be either the RV coil (integrated-bipolar pacing configuration) or the RV ring (true-bipolar pacing configuration). Some ICDs permit simultaneous (or temporally offset) true-bipolar pacing in both the LV and RV (Fig. 11).
Figure 11. Extended and true bipolar left LV pacing configurations (available in the Contak Renewal TM ICD). The top two panels depict extended bipolar pacing, in which pacing occurs between LV and RV electrodes. This configuration has the advantage of requiring only a single LV electrode, but the disadvantage of permitting anodal capture (Fig. 13). The ability to select either the distal or proximal LV electrode as cathode for pacing functionally permits "moving" the LV lead via noninvasive reprogramming. This can be advantageous postoperatively if the pacing threshold increases or phrenic-nerve stimulation occurs. The bottom two panels depict true-bipolar LV pacing, which eliminates the possibility of anodal RV capture, but requires a bipolar LV lead.

Determining whether the LV lead is capturing can be difficult. At least seven possible states of ventricular pacing can occur in cardiac resynchronization systems that use an extended bipolar pacing configuration. These are no capture (intrinsic conduction) and capture from six specific pacing configurations: RV cathodal + LV cathodal (the intended state); LV (cathodal) only; RV cathodal only; RV anodal only; RV anodal + LV cathodal; and RV (cathodal + anodal) + LV ("triple site" pacing). Most of these can be distinguished by analysis of intracardiac electrograms combined with a 12-lead ECG, which is invaluable for follow-up of resynchronization pacing systems.

In early cardiac resynchronization systems, dual-cathodal pacing applies the same voltage to the commonly wired LV and RV cathodes. Since the local LV and RV thresholds usually differ, the pacing pulse may be supra-threshold in one chamber and subthreshold in the other, leading to loss of resynchronization. In these ICDs, a change of ventricular-electrogram morphology during a threshold test indicates loss of capture at one electrode; the corresponding change in the surface ECG usually indicates which pacing configuration is capturing (see below). Current systems permit independent programming of RV and LV outputs, facilitating determination of the RV and LV threshold.

ECG changes associated with biventricular pacing have been reviewed. During determination of the LV pacing threshold, the QRS complex usually widens with loss of LV capture. It becomes less negative in lead I and more negative in lead III as ventricular excitation originates from the inferior RV as opposed to the lateral LV (Fig. 12).
During dual-cathodal pacing, a ventricular electrogram that follows the pacing stimulus by 50–200 ms may assist in determining the site of capture. It occurs when one ventricular electrode senses the wavefront propagating from the site of capture in other ventricle, giving rise to the second event. “Anodal capture” occurs when the delivered voltage captures at the RV anode either in isolation or in conjunction with the LV cathode. When anodal capture results in “triple-site” pacing (LV cathode, RV cathode, and RV anode), the QRS duration may decrease by 10–20 ms and QRS amplitude of leads I and aVL may increase.71 If anodal RV capture is mistaken for LV capture, the pacing output may be programmed to a subthreshold value in the LV, resulting in loss of cardiac resynchronization (Figs. 13 and 14). It is much more common if the RV anode is a ring electrode (true-bipolar pacing), which has a small surface area (high current density) and may be in direct contact with endocardium than if the RV anode is a defibrillation coil (integrated-bipolar pacing), which has a large surface area (low current density). Thus, anodal capture is extremely rare in Medtronic and Guidant ICDs, which use integrated-bipolar pacing. St. Jude ICDs use true-bipolar pacing if the LV lead is unipolar and the RV lead is true-bipolar. Anodal capture is more common in this pacing configuration.

Ensuring Delivery of Effective Resynchronization

In order to provide effective cardiac resynchronization, a high frequency of biventricular pacing must be delivered. A practical goal is to resynchronize 90% of R-R intervals. Figure 15 shows a systematic approach to confirming resynchronization. Loss of resynchronization occurs if LV capture fails. Kay provides a comprehensive review of the differential diagnosis of loss of resynchronization for reasons other than loss of LV capture.72 The most common causes are intrinsic AV conduction due to a long programmed AV

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Figure 12. QRS morphology during biventricular pacing. The morphology of the paced QRS complex depends on lead positions and the distribution and properties of diseased myocardium. Left panel: LV capture is suggested by a markedly negative QRS in lead I. Middle panel: Loss of LV capture is associated with a positive QRS compels in lead I and a more negative deflection in lead III. Right panel: With loss of capture, intrinsic conduction returns. In this case, the intrinsic QRS is narrow.
Figure 13. Possible modes of anodal capture during biventricular pacing. In all examples, an extended bipolar paces between LV and RV leads. The top left panel demonstrates biventricular pacing using an integrated-bipolar RV lead, in which the common anode is the distal defibrillation coil. Since the RV coil has a large surface area and thus a low current density, anodal capture is rare. The remaining panels depict biventricular pacing between a LV electrode and a true-bipolar RV lead, in which the common anode is the RV ring electrode. The top right panel depicts normal function, with capture only at the LV tip during LV-only pacing. The bottom left panel depicts capture only at the anode. This occurs if the anodal RV-ring threshold is lower than the LV threshold and the pacing output is between the RV anodal threshold and LV cathodal thresholds. The bottom right panel depicts anodal and cathodal capture. This may confound the determination of LV pacing threshold. See Figure 14.

delay and loss of atrial tracking at fast atrial rates. Others include frequent premature ventricular complexes, sensing malfunction (see above), programming considerations and/or specific pacing algorithms (see below). Interruption of cardiac resynchronization pacing is common, but it can usually be restored by noninvasive or invasive intervention.73

ICD Programming and Resynchronization Algorithms

Any parameter setting that minimizes ventricular pacing or permits ventricular fusion will adversely affect cardiac resynchronization. Such settings include a long AV delay, a prolonged post-ventricular atrial refractory period or extension of the post-ventricular atrial refractory period after a premature ventricular complex, a low maximal-tracking rate, AV search hysteresis, rate-smoothing up or down, or use of a DDI pacing mode. Atrial undersensing and ventricular oversensing may similarly minimize ventricular pacing and limit resynchronization.

Resynchronization ICDs have features designed to maintain LV stimulation. To prevent LV T-wave oversensing, Guidant Renewal™ ICDs incorporate an LV refractory period, which may inhibit LV pacing. Additionally, they include an LV protection period after a sensed or paced event during which pacing will not occur. Although designed to prevent pacing in the LV vulnerable period, this parameter reduces the maximum LV pacing rate and may inhibit cardiac resynchronization (Fig. 16).

Resynchronization ICDs also employ algorithms designed to maximize ventricular pacing during potentially disruptive events such as premature ventricular complexes or rapidly conducted atrial arrhythmias. In Medtronic resynchronization ICDs, the Ventricular Sense Response™ feature triggers pacing in one or both ventricles after each RV-sensed event.74 Both Medtronic and Guidant systems include algorithms that temporarily shorten the post-ventricular atrial refractory period to regain atrial tracking and restore resynchronization after premature ventricular complexes or during sinus tachycardia faster than the nominal upper rate limit. Medtronic’s Conducted AF Response™ resynchronizes conducted beats in atrial fibrillation up to a minimum R-R interval without increasing ventricular rate. Guidant’s Ventricular Rate Regularization™ algorithm is intended to restore resynchronization and ventricular regularity by pacing the ventricle during irregular conduction of atrial fibrillation. Concealed conduction into the AV node from the paced events may slow AV nodal conduction, thereby limiting the pacing-induced increase in ventricular rate. These and other algorithms designed to maximize cardiac resynchronization therapy may result in pacing after QRS onset on surface ECGs and pacing at “unexpected” times (Fig. 17).

Phrenic-Nerve Stimulation

Left phrenic-nerve stimulation by an LV electrode may make cardiac resynchronization intolerable. It may become manifest only after implantation due to postural changes or minor lead migration. In Guidant ICDs, daily impedance measurements performed to assess lead integrity temporarily increase the pacing amplitude. They may need to be programmed “off” in patients who have phrenic stimulation at the higher output. Decreasing the pacing output noninvasively eliminates phrenic stimulation if there is a sufficient safety margin between the LV and phrenic-nerve thresholds. Data are insufficient to determine how
Figure 14. Anodal capture during testing of LV capture threshold. LV pacing occurs between a LV cathode and a RV ring anode. (A) Upper left panel: From top to bottom: surface ECG, electrogram markers and intervals, atrial electrogram, and ventricular near-field electrogram. At left, pacing output is 2.5 V and anodal capture is present, with capture occurring at both the LV electrode and the RV ring. No electrograms are visible on the LV channel due to the simultaneous RV and LV capture. With a decrement in pacing output to 2.25 V, the QRS morphology changes on the ECG (circled complex); and electrograms appear on the ventricular channel, representing local RV depolarization. Inset at lower right shows that the time between the pacing stimulus and the RV electrogram represents the interventricular conduction time during LV pacing. (B) With further decrement in LV pacing output from 0.75 to 0.5 V, true loss of LV capture occurs, with widening of the surface ECG due to left bundle branch block. The local RV depolarizations are similar during LV pacing (in A) and intrinsic AV conduction (panel B).

Troubleshooting Interactions with ICDs

Antiarrhythmic Drugs

Antiarrhythmic drugs are prescribed commonly to ICD patients. Serious drug-ICD interactions associated predominantly with the use of sodium- or potassium-channel blocking (Vaughn-Williams class IA, IC, or III) drugs have been reviewed and are summarized in Table II. These interactions must be considered when antiarrhythmic drug treatment is initiated or the dose is altered.

Sensing, Detection, and Pacing

Drug effects on the amplitude and slew rate of local electrograms may impair sensing. This may delay or prevent detection of VT/VF. Antiarrhythmic drugs that slow the rate of VT below the programmed VT rate cutoff can prevent initial detection of VT or divert appropriate therapy during reconfirmation (Fig. 18). Class IC drugs bind sodium channels in the open state, resulting in greater drug effect during tachycardias. They may alter ventricular-electrogram morphology sufficiently in SVT to invalidate a morphology-algorithm template acquired at a normal sinus rate. Because VT displays increased cycle length variability in patients taking Class IC drugs, interval-stability detection enhancements may misclassify VT as atrial fibrillation. The effect of Class IC drugs to increase pacing...
Figure 15. Systematic approach to insuring delivery of cardiac resynchronization therapy. CRT = cardiac resynchronization therapy; PVC = premature ventricular complex; VTns = nonsustained VT; AVN = AV node.

Figure 16. Inhibition of LV pacing by the programmable LV protection period in a Contak Renewal 3TM. Shown from top to bottom are ECG lead II, RV electrogram, and LV electrogram. A programmer command for LV pacing was issued during atrial fibrillation. Since the LV protection period prevented pacing at the selected rate, markers indicating inhibition of pacing appear ("inh-LVP"). Programming the protection period off permitted assessment of the LV threshold. RVS = right ventricular sense.
Figure 17. Algorithms designed to maintain a high percent of cardiac resynchronization pacing during atrial fibrillation may be confused with inappropriate pacing. (A) Hospital telemetry shows ventricular pacing occurring after the QRS onset due to the Medtronic Ventricular Sense Response™ algorithm, which introduces a triggered biventricular pacing pulse after each RV-sensed event. (B) Surface ECG lead II, event markers, and the RV tip to LV tip electrogram are displayed during atrial fibrillation. The first three complexes represent biventricular pacing (“BV”). The last complex represents a conducted complex that was sensed (“VS”). The split marker associated with the “VS” indicates that a triggered pace pulse resulted. (C) Hospital telemetry shows pacing during rapidly conducted atrial fibrillation due to an algorithm designed to maximize cardiac resynchronization pacing. This pacing, with the patient at rest, is distinct from rapid, rate-responsive pacing that is triggered by a sensor.

thresholds is greatest at rapid rates, so that antitachycardia pacing could be subthreshold even if bradycardia pacing is supra-threshold at slower rates. This rarely presents a clinical problem because the pulse amplitude for antitachycardia pacing nominally is higher than that for bradycardia pacing. When Class IA, IC, or III antiarrhythmic drugs are started in an ICD patient, the slowest VT zone detection interval should be increased by ≥40 ms, SVT-VT discriminators should be active to prevent inappropriate therapy of SVT with rate overlapping that of drug-slowed VT, the interval-stability discriminator should be programmed to a less specific value, a new morphology template should be acquired during atrial pacing at a rapid rate, and a sustained-duration override should be considered. Noninvasive programmed stimulation may be appropriate to determine if VT/VF is detected accurately and terminated promptly.

Defibrillation

Antiarrhythmic drugs may cause potentially life-threatening rises in DFTs. Drug-defibrillation interactions are complex. Studies are confounded by anesthetic effects, heterogeneity in ICD leads and waveforms, and inter-species variability (e.g., human vs canine vs porcine). Drugs that block the fast inward sodium current (such as lidocaine) elevate the DFT, whereas agents those that block repolarizing potassium currents (such as sotalol and dofetilide) lower the DFT slightly. Improved defibrillation safety margins have mitigated these adverse pharmacologic effects, and potent sodium-channel blocking drugs are rarely prescribed for chronic oral therapy in ICD patients. However, chronic oral amiodarone increase DFTs to a clinically relevant degree in some patients. When treatment is initiated with amiodarone or another drug that often elevates the DFT, the defibrillation safety margin should be confirmed if it is both
TABLE II.
Adverse Interactions Between Antiarrhythmic and ICDs

<table>
<thead>
<tr>
<th>ICD Function</th>
<th>Effect</th>
<th>Drugs</th>
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<tr>
<td>Undersensing (rare)</td>
<td>Diminished electrogram slew rate/amplitude</td>
<td>1A, 1C</td>
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<tr>
<td>Underdetection of VT</td>
<td>VT slowed to rate &lt; detection rate</td>
<td>IA, IC, III</td>
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<td></td>
<td>Increase in cycle length variability of VT results in</td>
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<td></td>
<td>misclassification of VT as SVT by stability enhancement</td>
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<tr>
<td>Inappropriate detection of SVT</td>
<td>Organization of atrial fibrillation into atrial flutter with 1:1</td>
<td>1C</td>
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<td></td>
<td>AV conduction</td>
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<td></td>
<td>Use-dependent altered morphology of ventricular electrogram causes</td>
<td>1C</td>
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<tr>
<td></td>
<td>misclassification of SVT as VT</td>
<td></td>
</tr>
<tr>
<td>Increase in pacing threshold</td>
<td>Baseline</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rapid pacing rates for antitachycardia</td>
<td>Amiodarone, 1C</td>
</tr>
<tr>
<td>Bradyarrhythmias</td>
<td>Battery depletion from more bradycardia pacing</td>
<td>1C, β-blockers,</td>
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<td></td>
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<td>calcium antagonists</td>
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<tr>
<td>Increase in defibrillation threshold</td>
<td>Unsuccessful shocks/death</td>
<td>Amiodarone, 1B</td>
</tr>
<tr>
<td>More aborted or delivered shocks</td>
<td>Proarrhythmia</td>
<td>IA, IC, III</td>
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marginal (e.g., safety margin <10 J) and if an insufficient safety margin would result in a change in therapy (revision of the defibrillation system or discontinuation of the drug). Similar considerations apply to substantial increases in amiodarone dose.

Metabolic Effects

Hyperkalemia is the metabolic abnormality with the greatest clinical effect on ICD function. In addition to increase in pacing threshold, it can present as T-wave oversensing, R-wave double-counting, or failure to detect VT or VF (Fig. 19). Hypokalemia and hypomagnesemia may contribute to repetitive episodes of VT (“VT storm”).

Electromagnetic Interference

Ubiquitous environmental electromagnetic energy can generate electrical potentials on ICD sensing electrodes. Such electromagnetic interference (EMI) can result in inappropriate detection of VT/VF, failure to sense VT/VF, and inappropriate pacing or inhibition of pacing. A static magnetic field causes reversion to magnet mode, which usually disables detection of VT/VF but does not alter pacing. Electromagnetic interference with frequencies less than ~2 Hz may be

Figure 18. Underdetection of VF caused by antiarrhythmic drugs (amiodarone and mexiletine). Displayed from top to bottom are the atrial, ventricular near-field and ventricular far-field electrograms, and event markers. VT degenerates to VF. The left panel shows detection of VF. Despite persistence of VF (seen in far-field ventricular electrograms), slowing in local near-field electrogram results in four events at cycle length 378–388 ms, slower than the VT detection interval (“VS”) resulting in diversion of therapy (“Dvrt Chrg”). Detection occurred eventually, and the resultant ICD shock terminated VF. Atrial asystole occurs during VT/VF.
detected as R-waves and prevent automatic adjustment of sensitivity from sensing VT/VF. Oversensing of higher frequency electromagnetic interference may cause inappropriate detection of VT/VF. Pacemaker function may respond as tracking (atrial channel) or inhibition (ventricular channel). These effects have been reviewed.86,87 With the exception of industrial and military sources, clinically significant interference by nonmedical sources is rare.88,89 Interference from sources in the medical environment has been studied specifically, including magnetic resonance imaging90,91 radiofrequency catheter ablation92 and surgical electrocautery.93 We address limited issues relevant to troubleshooting.

Electromagnetic interference is a lesser problem for true-bipolar sensing than for integrated-bipolar sensing. It is diagnosed based on history of exposure and characteristic high-frequency, non-physiological signals that are larger in far-field than near-field electrograms (fig. 3 in part I of this review). Diminishing programmed sensitivity may mitigate interference. VT/VF detection may be disabled during planned, unavoidable exposure (e.g., surgical electrocautery) if an external defibrillator and trained personnel are present. Field strength in the patient’s environment may be measured if external electromagnetic interference is suspected. Static magnetic fields > 5 G should be avoided. ICD detection of time-varying, external magnetic fields
two distinct rates of VT and antitachycardia pacing for monomorphic VT that overlaps in rate with polymorphic VT. Other experts recommend two rate zones with the slowest at cycle length 340–320 ms for patients whose only spontaneous arrhythmia is VF. This approach lowers the risk of inappropriate therapy but increases the risk of not treating VT. In the largest primary-prevention trial, ICDs were programmed to a single detection zone at cycle length 320 ms, without SVT-VT discriminators. Approximately half of shocks were inappropriate; the incidence of sudden death from undetected VT has not been reported.

The sinus-VT rate boundary should be slow enough to ensure detection of all hemodynamically compromising VT. The boundary between the two VT zones should be based on the cycle length at which different types or fewer trials of antitachycardia pacing are preferred. The VT-VF rate boundary is based on the cycle length below which antitachycardia pacing should not be delivered. In Medtronic ICDs, which use consecutive-interval counting above the VF Interval and X-of-Y counting below it, this boundary should be set to prevent underdetection of irregular, polymorphic VT by consecutive-interval counting.

**Duration for Detection and Redetection**

Detection duration prior to antitachycardia pacing should not be decreased from nominal values because therapy is immediate after detection and undersensing of monomorphic VT is rare. It should be increased in patients who have long episodes of nonsustained VT. Substantial increases in duration for detection of VT probably are safe in St. Jude and Guidant ICDs, which use counting methods that are insensitive to occasional long ventricular intervals or undersensing: Guidant ICDs use X-of-Y counting, and St. Jude ICDs count based on the interval and average of the previous three intervals. In contrast, consecutive-interval counting used by Medtronic ICDs in the VT zone may underdetect VT if occasional long ventricular intervals or undersensing occur. Unless VT is known to be highly regular, the number of intervals to detect VT usually should not be more than 50% greater than the nominal value in Medtronic ICDs. Duration for detection of VF may be reduced from nominal only if there is no alternative method to ensure reliable detection. Redetection time for VT should be increased from nominal if VT is well-tolerated, but antitachycardia pacing is not. Redetection time for VF should be decreased if post-shock undersensing occurs.
SVT-VT Discrimination

Some SVT discriminators should be programmed at implant in all patients with intact AV conduction. See Section III D.

Programmed Therapy

Antitachycardia pacing should be programmed in all patients unless it is known to be ineffective or proarrhythmic. See Section IV C. The first shock strength for VF should be set either in relation to an established implant safety margin or to maximum.16,27 In modern ICDs with short charge times, we recommend that shocks for hemodynamically stable VT be programmed to the same strength as those for VF, rather than to a low energy. This maximizes the likelihood that a ventricular shock will terminate (inappropriately detected) rapidly conducted, atrial fibrillation. It also minimizes both the risk that a ventricular shock will accelerate VT and the risk that it will be weaker than the atrial upper limit of vulnerability (and thus initiate atrial fibrillation if it is delivered during the atrial vulnerable period). But we recognize that practice differs with regard to this issue.

Evolving Trends

The ICD has expanded from a single-purpose “shock box” to a multi-purpose platform for electrical cardiac therapies; ICDs under development include sensors for monitoring heart failure and other comorbidities. This increasing functionality and complexity is likely to expand the domain of troubleshooting and to result in yet unknown, unintended consequences, including novel intradevice interactions.

Present ICDs have approximately 500 programmable features. Despite simplified user interfaces that might reduce operator errors, both nonlethal100,101 and lethal4,96 programming errors occur. The automatic, self-checking, and self-adjusting functions in today’s ICDs are a welcome addition given the time pressures of present clinical practice and the fact that troubleshooting is both time consuming and poorly reimbursed. However, automated features should be disabled in some conditions. Examples discussed in this review include the need to disable automatic updating of morphology-algorithm templates in patients with rate-related aberrancy and the need to disable Medtronic Smart Mode when VT recurs rapidly after effective antitachycardia pacing. Future automated features may also introduce unforeseen consequences that require novel forms of troubleshooting.

A potential risk of simplifying the user interface is loss of programming flexibility and troubleshooting tools. ICDs are required to perform multiple functions based on input signals and clinical conditions that vary widely from patient to patient. Features optimized for one condition do not perform optimally for another condition. Consider familiar clinical trade-offs: The preferred solution to rejection of far-field R-waves depends on the relative sizes and timing of the P-wave and far-field R-wave as well as the likelihood that the patient will develop atrial fibrillation or flutter. The preferred solution to discriminating SVT from VT with a 1:1 AV relationship depends on various features such as the reliability of morphology discrimination with available electrograms, the difference between VA and AV times during VT and SVT, and the specific responses of VT and SVT to antitachycardia pacing. Prophylactic implantation of ICDs without prior electrophysiological evaluation increases the conflicting requirements for reliable detection of all VTs while rejecting unanticipated SVTs.

ICD manufacturers are working to provide a simplified user interface that provides sufficient options for common clinical situations. At the same time, they seek to maintain and increase the wide range of flexible options that permit optimal programming for outlier patients or clinical conditions. Manufacturers must also provide physicians with adequate technical references for troubleshooting. Reference manuals are no longer packaged with ICDs, although some manufacturers provide online manuals.

Present ICDs provide troubleshooting alerts to the patient and physician for specific risk conditions.40 Most relate simply to a parameter “out-of-range,” but at least one lead-integrity alert that integrates multiple parameters has been verified and released.39,102 The capability to download ICD data remotely over the Internet37,38 permits development of automated troubleshooting algorithms that require computing power beyond that available to implanted ICDs or even ICD programmers. One application would be high-level, automated analysis of stored arrhythmia episodes to evaluate the accuracy of SVT-VT discrimination and recommend programming changes. Physician-initiated or automated ICD reprogramming via internet could both simplify troubleshooting and introduce novel problems.

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ADVANCED ICD TROUBLESHOOTING

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