Optimal Programming of Implantable Cardiac-Defibrillators

Malini Madhavan, MBBS; Paul A. Friedman, MD

Implantable cardioverter-defibrillators (ICDs) reduce sudden cardiac death risk and improve survival in patients with a history of life-threatening arrhythmia or cardiac arrest (secondary prevention) and in high-risk patients without such a history (primary prevention).[1–3] Patients with ICDs, however, may receive unnecessary shocks, which have been associated with proarrhythmia, anxiety, depression, poor quality of life, and possibly increased mortality.[4–8] In contrast to drug therapy, in which a limited number of parameters (such as dose) are adjustable, hundreds of programmable parameters affect device therapy delivery. Moreover, selection of nonnominal parameter settings (ie, changing the out-of-the-box factory default settings) reduces morbidity and mortality compared with nominal settings in many clinical situations because of publication and regulatory delays.[4,9,10] Thus, optimization of programming to prevent unnecessary shocks is paramount to minimize morbidity and mortality and is dependent on clinician proactivity.

Inappropriate Shocks and Unnecessary Shocks

Delivery of ICD therapy for rhythms other than ventricular tachycardia (VT) or ventricular fibrillation (VF) is termed *inappropriate therapy*. It may result from inappropriate detection of supraventricular tachycardia (SVT) such as atrial fibrillation (AF) or sinus tachycardia or from oversensing of physiological (eg, T wave) or nonphysiological (eg, lead fracture noise) signals. Inappropriate shocks have been reported in 16% to 18% of ICD recipients and constitute 30% to 50% of all shocks.[11–13] Although therapy delivered for a ventricular tachyarrhythmia is considered appropriate, a growing body of evidence has shown that programming antitachycardia pacing (ATP) or delaying shocks to permit nonsustained episodes to terminate significantly reduces the frequency of shock delivery. Koneru et al[14] have used the term *unnecessary shocks* to refer to inappropriate shocks delivered for rhythms other than VT/VF and to include shocks delivered for VT that would have responded to ATP or terminated spontaneously if given time. Optimal programming minimizes unnecessary shocks.

ICD Shock–Reduction Strategies: Overview

A comprehensive approach to shock reduction includes appropriate patient selection, management of heart failure, ICD programming, and in some cases, use of antiarrhythmic drugs and catheter ablation to prevent VT/VF occurrence. Here, we focus on optimal ICD programming.

The detection and treatment of ventricular tachyarrhythmias by an ICD involves a series of sequential steps, each of which provides an opportunity to minimize unnecessary shocks (Figure 1). A sense amplifier processes local intracardiac electrograms or subcutaneous electrograms to create a series of discrete cardiac events. The time interval between events defines the heart rate. Errors in event detection (oversensing or undersensing) may lead to errors in rate calculation and absent or inappropriate therapy. Once a rate threshold has been crossed, a tachycardia must persist for a programmed duration or number of intervals before is detected. Although there are differences in how this process is performed across manufacturers, the fundamental concept of rate and duration triggering tachycardia detection is similar across all devices and is therefore hardware independent.

After tachycardia detection, algorithms may be applied to distinguish SVT from VT. These algorithms are hardware dependent and differ markedly between single-chamber, dual-chamber, and subcutaneous ICDs. Single-chamber detection enhancement algorithms use V-V timing intervals and comparison of ventricular morphology during tachycardia to baseline in single-chamber ICDs to distinguish SVT from VT. Dual-chamber ICDs also assess A-A intervals, A-V relationships, and chamber of onset for SVT-VT discrimination, whereas the subcutaneous ICD (s-ICD) processes the surface ECG equivalent from 1 of 3 possible vectors to identify the rhythm. If classified as ventricular, a tachycardia may receive ATP, which, if successful, prevents shock delivery. After therapy delivery, redetection or reconfirmation (abbreviated redetection at end of charge) determines whether tachycardia is ongoing and additional therapy is needed. Each of these steps affords an opportunity to optimize programming and is discussed in further detail.

Rate and Duration for Initial Detection

ICDs detect VT/VF if the RR intervals are shorter (ie, the heart rate is faster) than the detection interval for a programmable number of intervals or duration. Up to 3 programmable zones are available, each with independently programmable rates and durations, with therapies (ATP, shock) individually programmed within each zone. The zones are defined strictly by heart rate, so that a rapid monomorphic VT is labeled a VF episode if it falls within the VF zone. In the VT zone, the counter may be cumulative or consecutive (ie, reset if 1 interval falls outside of the VT zone). In the VF zone, a percentage of

From the Division of Cardiovascular Diseases, Mayo Clinic, Rochester, MN.
Correspondence to Paul A. Friedman, Division of Cardiovascular Diseases, Mayo Clinic, 200 First St SW, Rochester, MN 55905. E-mail friedman.paul@mayo.edu

(*Circulation*. 2013;128:659-672.)

***Circulation* is available at http://circ.ahajournals.org** **DOI: 10.1161/CIRCULATIONAHA.112.000542**

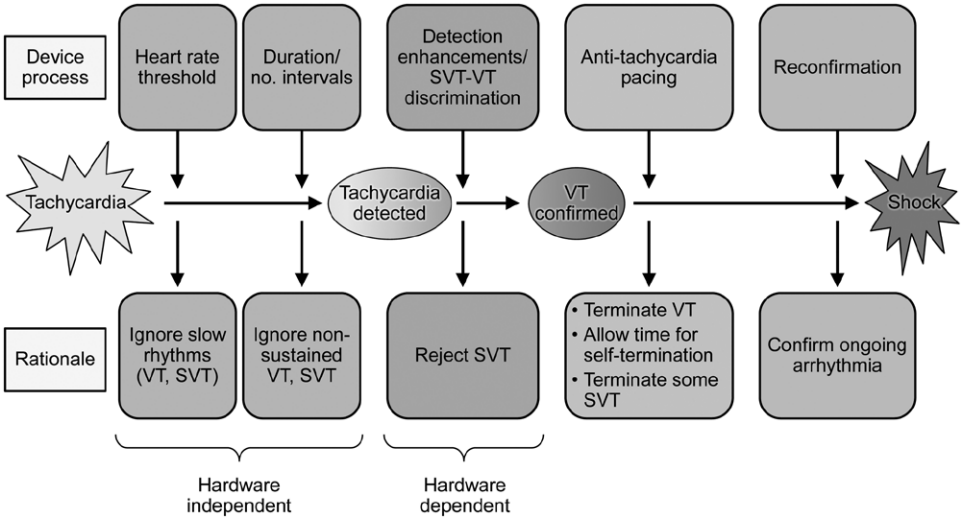


Figure 1. Overview of detection and treatment of ventricular arrhythmia by implantable cardioverter-defibrillator (ICD). The detection and treatment of ventricular arrhythmia by an ICD involves a sequence of events that provide opportunities to prevent unnecessary shocks using appropriate programming. SVT indicates supraventricular tachycardia; and VT, ventricular tachycardia.

intervals must be shorter than the rate cutoff; this strategy permits some of the highly amplitude-variable VF electrograms to drop out without hampering detection. Detection duration is shorter in the VF zone because of potential hemodynamic instability with delay to therapy.

Recognition of population-based arrhythmia characteristics informs optimal programming. In contrast to patients with a secondary prevention indication, recipients of an ICD for primary prevention with predominantly ischemic or dilated cardiomyopathy have a shorter VT cycle length (303±54 versus 366±71 ms) and a longer inappropriately detected supraventricular arrhythmia cycle length (363±70 versus 323±75 ms; Figure 2A).[15] The observation that primary prevention patients experience faster VTs with rates less likely to overlap SVT than secondary prevention patients permits the programming of faster VT rate cutoffs, which minimizes the number of

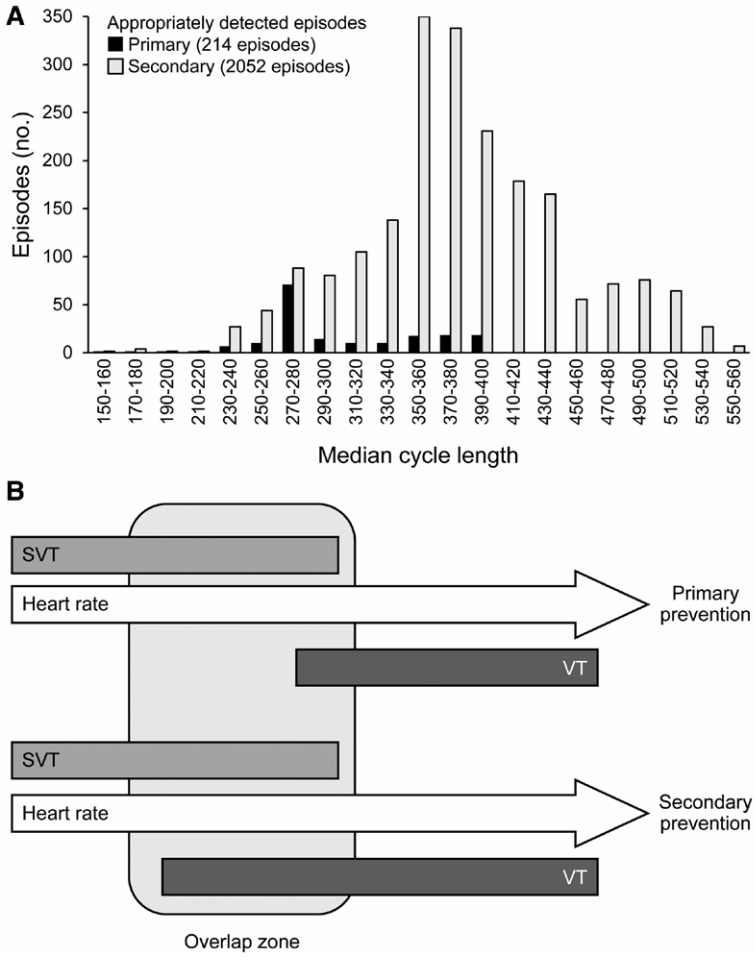


Figure 2. Programming of therapy zones based on arrhythmia characteristics in patients in whom an implantable cardioverter-defibrillator (ICD) was implanted for primary and secondary prevention. **A**, Rates of ventricular and supraventricular arrhythmias detected in patients in whom an ICD was implanted for primary and secondary prevention. Reproduced from Wilkoff et al[15] with permission of the publisher. Copyright © 2004, Wiley. **B**, Programming of therapy zones in primary and secondary prevention patients. Device programming in patients with an ICD is guided by indication-based arrhythmia characteristics. Patients with a primary prevention indication have faster ventricular tachycardia (VT) and benefit from programming high-rate zones only. Secondary prevention patients have slower VT and greater overlap between VT and supraventricular tachycardia (SVT); hence, they may require programming of slower detection zones and benefit more from SVT-VT discrimination algorithms.[15]

tachycardias that the ICD must adjudicate and consequently minimizes unnecessary shocks in primary prevention patients (Figure 2B).[15,16] A number of studies have investigated this strategy in combination with prolonged detection time in primary prevention patients.

The PREPARE (Primary Prevention Parameters Evaluation) study was a cohort controlled study that analyzed 700 recipients of biventricular and nonbiventricular ICDs with a primary prevention indication.[9] In the active cohort, detection rates were set to 182 bpm, with the duration set for 30 of 40 intervals (≈9 seconds). Compared with a historical control with slower rate cutoffs and faster detection, PREPARE study patients were less likely to receive a shock (9% versus 17%, $P<0.01$), with no difference in the rate of arrhythmic syncope between groups. The RELEVANT study (Role of Long Detection Window Programming in Patients With Left Ventricular Dysfunction, Non-ischemic Etiology in Primary Prevention Treated With a Biventricular ICD) randomized nonischemic cardiomyopathy patients with a primary prevention ICD indication to short (12/16 interval) or long (30/40 interval) detection. Ninety percent of VT and SVT terminated within 30 beats in the long-detection group, which resulted in a significant reduction in number of shocks (relative risk 0.36) without an increase in syncope or death.[17]

The MADIT-RIT study (Multicenter Automatic Defibrillator Implantation Trial to Reduce Inappropriate Therapy) prospectively randomized 1500 primary prevention ICD recipients to confirm the safety and efficacy of high-rate and prolonged duration to detection programming.[4] Patients were randomized to 1 of 3 groups: (1) High-rate therapy (detection at ≥200 bpm with a 2.5-second delay to therapy), (2) delayed therapy (60-second delay at 170–199 bpm, 12-second delay at 200–249 bpm, and 2.5-second delay at ≥250 bpm), and (3) conventional therapy (2.5-second delay at 170–199 bpm and 1-second delay at ≥200 bpm). The high-rate and delayed-therapy groups had a lower risk of inappropriate therapy for SVT (shock and ATP, hazard ratio 0.21 and 0.24, respectively) and death (hazard ratio 0.45 and 0.56, respectively) with a similar incidence of syncope compared with conventional programming. These data indicate that programming primary prevention ICDs with a detection zone ≥200 bpm or with delayed therapy at >170 bpm is a preferred strategy. Furthermore, mortality was lower in the high-rate therapy group than with conventional therapy, which raises the possibility that the reduction in appropriate and inappropriate shocks in the trial could have led to better survival. The remarkable reduction in shocks and improved survival in this trial suggest that an increased detection rate or prolonged duration is the most robust programming tool for prevention of unnecessary ICD therapy in primary prevention patients. Similar studies in secondary prevention are lacking. In the only currently available s-ICD, rate is programmable but not duration. The 25% to 75% time from detection to shock is 13 to 16 seconds,[14] so that the use of a detection rate of 200 bpm results in a therapy delay slightly longer than that in the delayed-therapy arm of MADIT-RIT. Recommended programming parameters for the primary prevention indication at our institution are presented in the Table.

SVT-VT Discrimination

Rapidly conducted AF and other SVTs that exceed rate and duration thresholds account for 80% of inappropriate shocks.[11] SVT-VT discriminators are ICD algorithms used to withhold inappropriate therapy. The algorithms are hardware dependent, differing between single-chamber ICDs, dual-chamber ICDs, and s-ICDs. Because patients with secondary prevention indications for ICDs and those receiving antiarrhythmic drugs are more likely to experience overlap between SVT and VT rates (Figure 2A), these patients are most likely to benefit from SVT-VT discrimination algorithms.[15,16,18]

Single-Chamber Algorithms

Single-chamber ICDs use interval- and morphology-based ventricular electrogram information to distinguish SVT from VT. The individual algorithms are discussed below.

Onset

The sudden-onset algorithm distinguishes the abrupt change in RR intervals with the initiation of VT from the gradually shortening intervals during sinus tachycardia.[19,20] Because sinus tachycardia is detected at the lower boundary of VT detection, the algorithm is applied in slower VT zones. The development of VT during sinus tachycardia or in the presence of ectopy immediately preceding VT onset may lead to misclassification of VT as SVT; AF that begins abruptly will be misclassified as VT. Additionally, in contrast to other single-chamber SVT-VT discriminators that continuously reassess the rhythm, the onset algorithm makes its determination only once, so that initial errors cannot be corrected. Thus, onset is best used in conjunction with other algorithms for SVT discrimination to improve accuracy.

Stability

Variability in beat-to-beat RR intervals is used to differentiate AF (variable, unstable intervals) from VT (regular/stable intervals). Stability is applied during ongoing arrhythmia, so that a VT that begins with interval variability and stabilizes will be classified correctly. Stability misclassification arises from (1) rapidly conducted AF (approximately >175 bpm), during which the variability of RR intervals is diminished; (2) regular SVTs such as atrial flutter; and (3) irregular VT (as may occur in the presence of class 1C antiarrhythmic drugs) and polymorphic VT.[21–23] As with onset, the stability algorithm is also best applied in conjunction with other algorithms.[19,20,24]

Morphology

The morphology criterion is the only non–interval-based single-chamber SVT-VT discriminator. Morphology algorithms compare electrograms during tachycardia with a template acquired during normally conducted rhythm. A tachycardia morphology that differs from the stored template by a programmable threshold is classified as VT. The approach for aligning and comparing a tachycardia electrogram with a baseline template varies by manufacturer, but all have similar efficacy and failure modes. Misclassification occurs with (1) SVT with rate-related aberrancy, (2) errors in electrogram alignment, (3) electrogram truncation (ie, signal clipping caused by improper amplifier setting), (4) electrogram distortion caused by myopotentials, (5) changes in morphology

Table. ICD Programming for Primary Prevention of Sudden Death

Zone	Rate	Detection	Therapy	Discriminators
Therapy zone	200 bpm (300 ms)	5- to 9-second delay	ATP during charge, maximum-energy shocks	Morphology: On below the SVT limit* Stability, onset, dual-chamber SVT discriminators: Off
Monitor-only zone	170–199 bpm (353–301 ms)	9- to 60-second delay	None	SVT discriminators: On SVT discrimination timeout: Off

ATP indicates antitachycardia pacing; ICD, implantable cardioverter-defibrillator; and SVT, supraventricular tachycardia.
*Applicable only to Medtronic ICDs.

over time caused by lead maturation or bundle-branch block, and (6) electrogram distortion in the minutes immediately after shock delivery.[25] A number of programming steps can minimize the risk of misclassification. In patients with rate-related aberrancy, a template can be acquired during atrial pacing at a rapid rate (eg, 120 bpm) and template updates disabled. Alignment and truncation errors may be prevented with appropriate electrogram source selection and gain adjustment (Figure 4). Template autoupdate features correct for lead maturation and electrogram evolution over time. Morphology discrimination is not applied during redetection after shocks to prevent misclassification; however, SVT that develops shortly after a completed, shocked episode may be misclassified.

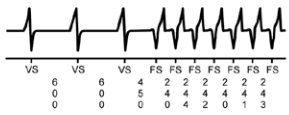
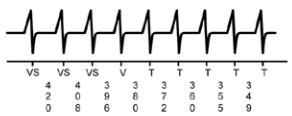
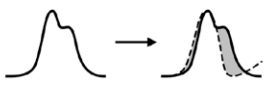
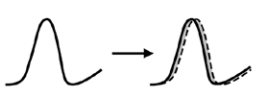
Algorithm	Function	Strengths	Limitations	Performance	Suggested parameter settings
Onset Inhibit therapy if gradual onset *Rationale:* Sinus tachycardia has gradual onset compared to abrupt onset of VT	Abrupt onset of VT  Slow warm up of sinus tachycardia 	High sensitivity for distinguishing sinus tachycardia from VT	Misclassify • AF, SVTs with abrupt onset as VT • Exercise induced VT that follows sinus tachycardia under detected • PVCs before VT may misclassify VT as gradual onset • Applied only once at initial detection - misclassification cannot be corrected	• Accuracy in rejecting sinus tachycardia – 98% • VT under detection in 0.5%[24] • Specificity – 64%[19]	• Medtronic 84 – 88% • Boston Scientific 9% • St. Jude 100 ms
Stability Inhibit therapy if ventricular rate is variable *Rationale:* RR intervals are irregular in AF compared to regular in VT	Stable RR intervals in VT  Irregular RR intervals in AF 	High sensitivity for discriminating VT from AF at rates < 170bpm	Misclassify • Stable SVT (e.g. atrial flutter) as VT • Rapid AF where RR is less variable as VT • Irregular VT (such as in the setting of anti-arrhythmic drugs) as AF	• Sensitivity 95%, specificity 77 – 88% at rate < 170bpm[19, 23] • Sensitivity and specificity ↓ at rates >170bpm. Best applied to tachycardia <170bpm[23]	• Medtronic 40 – 50 ms • Boston Scientific 24 – 40 ms, 2.5s • St. Jude 80 ms
Morphology Inhibit therapy if morphology of intracardiac electrogram matches template stored in normal rhythm *Rationale:* Discriminates SVT from VT based on comparison of morphology, independent of RR intervals	VT – morphology does not match template  SVT – morphology matches template 	• Discriminate abrupt onset stable SVT (e.g. atrial flutter) from VT • Applied continuously, permits correction if initial misclassification occurs • Can be applied at rapid rates > 200bpm	• Misclassify SVT with aberrancy • Misclassification due to electrogram mal-alignment, truncation • Cannot be applied to redetection post shock	• Most accurate of the single chamber algorithms • Medtronic wavelet™ sensitivity 100%, specificity 78%[26] • Boston Scientific Rhythm ID™ sensitivity 99-100%, specificity 92-97%[27, 28] • St. Jude Morphology Discrimination™ in conjunction with dual chamber discriminators sensitivity 100%, specificity 84%[29]	Medtronic - 3/8 electrograms >70% match Boston Scientific - Rhythm ID™ 'ON"* St. Jude - 5/8 electrograms > 60% match S-ICD: conditional zone 200-229 bpm (primary prevention)

Figure 3. Supraventricular tachycardia–ventricular tachycardia discriminators in a single-chamber implantable cardioverter-defibrillator. AF indicates atrial fibrillation; PVCs, premature ventricular contractions; S-ICD, subcutaneous implantable cardioverter-defibrillator; SVT, supraventricular tachycardia; and VT, ventricular tachycardia.

In general, the morphology algorithm provides greater sensitivity and specificity than the onset and stability algorithms, with reported sensitivity for detection of VT on the order of 92% to 99% and specificities of 90% to 97% (Figure 3).[26–29] Algorithms that are applied continuously during tachycardia, such as morphology and stability, are preferred over the onset algorithm, which can be applied only once. SVT-VT discriminators are more specific and more liberally applied in slower (VT) zones, in which a short delay in detection is generally well tolerated. The interval-based algorithms (onset and stability) are unreliable at higher rates (>180–200 bpm); morphology is the only SVT-VT discriminator reliably applied at rates >200 bpm. Frequently, the individual single-chamber algorithms are applied jointly with Boolean logic (AND/OR). The logic with which they are combined significantly affects overall performance. Manufacturer-specific recommended programming in single-chamber ICDs is presented in Figure 3.

Dual-Chamber SVT-VT Discriminators

Dual-chamber algorithms vary significantly between manufacturers, but all use information collected simultaneously from the atria and ventricles to improve specificity. They add atrial rate information to standard ventricular rate–only algorithms, compare the relative timing of atrial and ventricular events during arrhythmia, or mix interval and morphology algorithms, incorporating atrial information. For example, stability is applied only after the atria are confirmed to be in AF (Boston Scientific) or only when the atrial rate is faster than the ventricular rate (St Jude Medical). A ventricular rate greater than the atrial rate (V>A) is a highly specific marker of VT. Because the ventricular rate is faster than the atrial rate in 80% to 90% of VTs detected in the VT zone, the application of V>A as the initial step in some algorithms eliminates the need for single-chamber discriminators in these patients, which reduces the potential for misclassification.[25,30] Some manufacturers also use measures of atrioventricular (AV) association to distinguish regular SVT from VT. When AV association is operational, stable tachycardias with a constant AV interval will not be detected as VT despite their cycle-length regularity. Proper atrial and ventricular sensing is essential for correct diagnosis. Thus, it is important to prevent both far-field R-wave (FFRW) oversensing and the undersensing of small fibrillation waves on the atrial channel. A brief description of dual-chamber algorithms and programming recommendations is presented in Figure 5.

Atrial sensing is the Achilles' heel of dual-chamber algorithms. Atrial undersensing may be caused by low-amplitude electrograms (particularly during AF) or lead dislodgement, or it may be functional, resulting from the atrial blanking that occurs during and immediately after ventricular sensed or paced events. Atrial undersensing can lead to misclassification of SVT as VT. Oversensing is most commonly caused by large FFRWs (Figure 6), which may lead to misclassification of VT as SVT and inappropriately withheld therapy. During implantation, ensuring an adequate sensed P-wave amplitude and absence of sensed FFRW (with an atrial to FFRW electrogram ratio of at least 4:1) minimizes sensing errors. Atrial leads with small interelectrode distance (1.3 mm) reduce the risk of sensing far-field signals because of their smaller antenna effect.[31,32] Experimental data suggest totally intramyocardial electrodes (with both cathode and anode embedded within the myocardium) may increase tissue-sensing

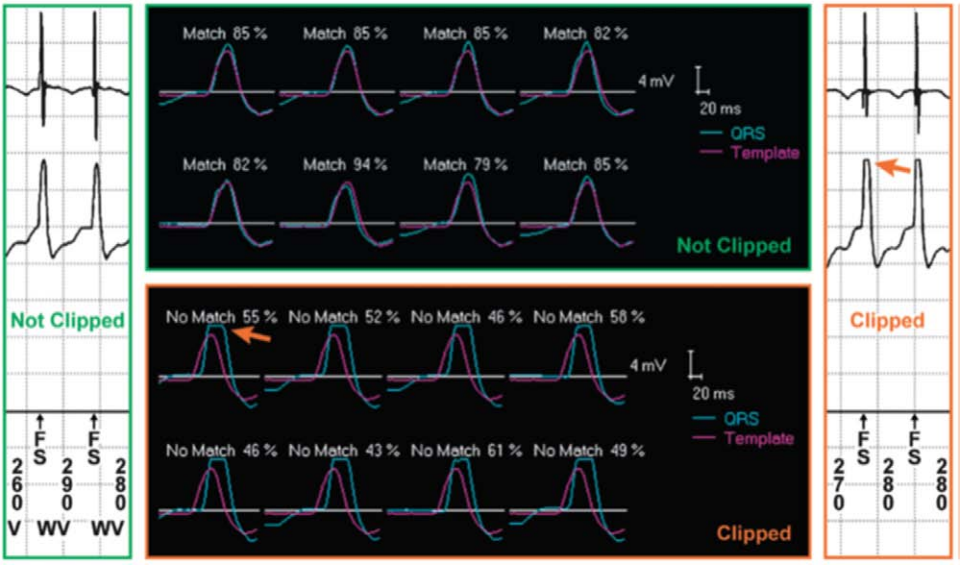


Figure 4. Failure of the morphology algorithm because of electrogram truncation. Electrogram truncation can lead to failure of morphology algorithm. If the electrogram amplitude exceeds the maximum range of dynamic sensing, the peaks may be clipped. Variable truncation can lead to morphology mismatch caused by malalignment and distortion of the electrogram. **Top** and **Left**, Appropriate rejection of a supraventricular tachycardia by the Wavelet morphology algorithm (Medtronic). **Bottom** and **Right**, The gain of the far-field electrogram was too high for the signal, which led to clipping or truncation of the peak. Because the Medtronic implantable cardioverter-defibrillator used the peak to align the electrogram, this led to misclassification by 2 mechanisms (electrogram distortion and misalignment). This can be prevented by programming the amplitude scale such that the sensed electrogram occupies 25% to 75% of the dynamic sensing range. Reproduced by permission of Mayo Foundation for Medical Education and Reseatch. All rights reserved.[25a]

specificity and eliminate FFRW oversensing.[33,34] When present, FFRW oversensing can be addressed by decreasing atrial sensitivity and extending the postventricular atrial blanking period; however, this increases the risk of undersensing AF and rhythm misclassification. All manufacturers use dynamic sensing or algorithmic signal processing to minimize the risk of atrial undersensing or oversensing. Algorithm performance can be enhanced by screening for FFRW oversensing at routine follow-up and adjusting atrial dynamic sensing to eliminate it when present.

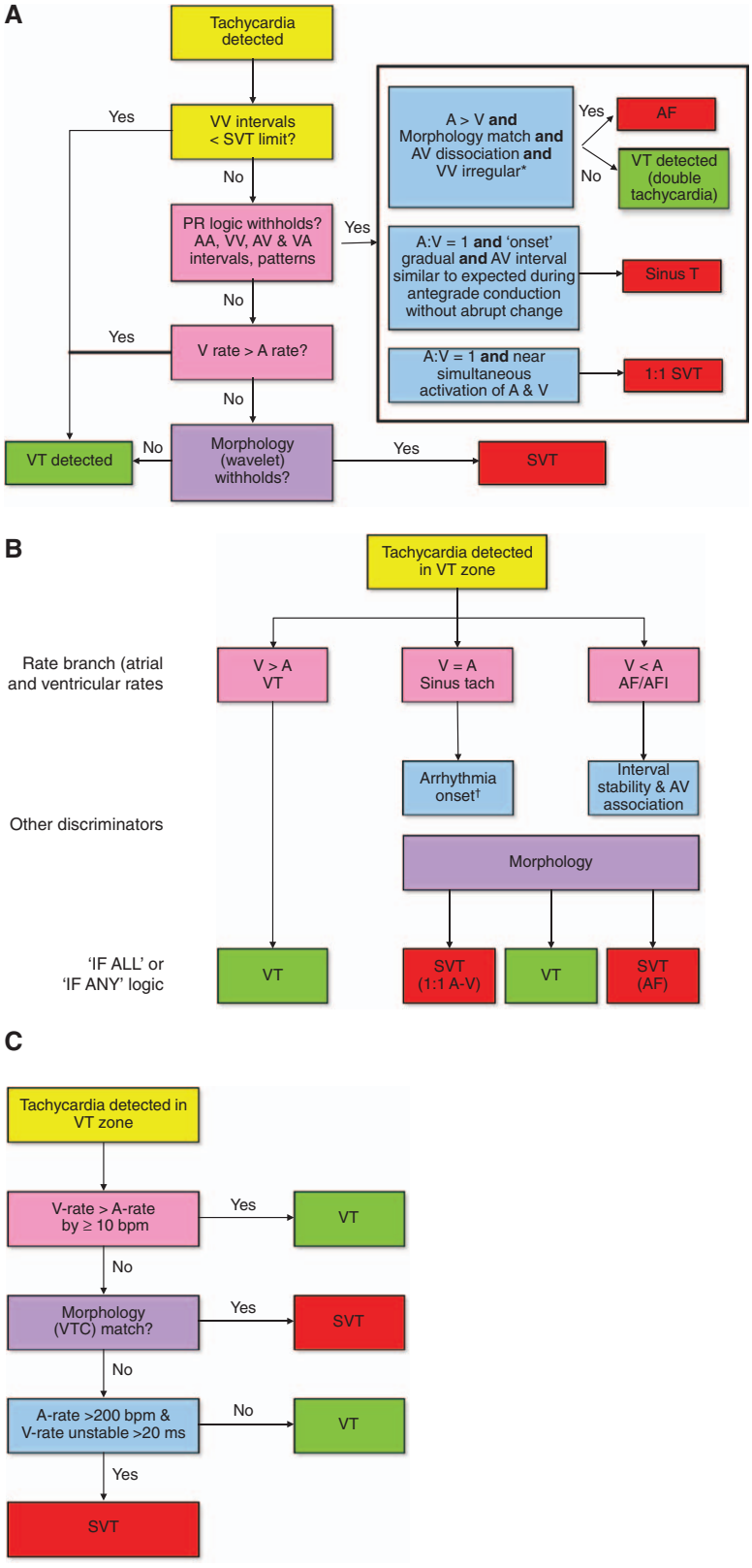


Figure 5. Dual-chamber supraventricular tachycardia–ventricular tachycardia discrimination algorithms. **A**, Medtronic PR Logic uses the AA and VV intervals, patterns of AV and VA intervals, and VV regularity and does not have an atrial blanking period after a sensed ventricular event. It has 3 distinct features that are individually programmable: Atrial fibrillation (AF)/atrial flutter (AFl), sinus tachycardia (Sinus T), and Other 1:1 supraventricular tachycardia (SVT). If the logic determines that there are ventricular and atrial tachyarrhythmias with dissociation, it is classified as ventricular tachycardia (VT), and therapy is delivered. The device applies Wavelet morphology if the sensed event is classified as VT by PR Logic. We recommend programming all features except for Other 1:1 SVT at the time of implantation. The Other 1:1 SVT feature can be turned on after atrial lead maturation to prevent misclassification of VT in the event of atrial lead dislodgement into the ventricle. We program off the onset and stability algorithms in dual-chamber devices. *Regularity of VV intervals in PR logic is applied only in the VT zone. **B**, The St Jude Medical implantable cardioverter-defibrillator uses the rate branch algorithm to classify the tachycardia into 3 groups based on the V and A rates: (1) If V>A, VT is detected; (2) if V=A, other discriminators, including morphology and arrhythmia onset, are applied; (3) if V<A, discriminators to identify AF, including stability plus AV association and morphology, are applied. Each discriminator is individually programmable. The results of each discriminator are then applied by use of ALL or ANY logic: (1) If the ALL logic is programmed, VT is detected only if all the programmed discriminators are classified as VT; if not, SVT is detected. (2) If the ANY logic is programmed, VT is detected even if 1 of the discriminators classifies as VT. We recommend the following programming: (1) rate branch on, (2) morphology on in V=A branch with sudden onset programmed to passive mode and (3) stability and morphology on in V<A branch using ALL logic. †Arrhythmia onset incorporates sudden onset and the chamber of onset of the arrhythmia to distinguish SVT from VT. **C**, The Boston Scientific Rhythm ID algorithm applies the following discriminators in sequence: (1) V>A rate, (2) morphology criteria (vector timing and correlation [VTC]), and (3) AF detection using a combination of atrial rate (A-rate) over the threshold and ventricular rate (V-rate) instability. Undersensing in the atrium can lead to misclassification of SVT as VT. During redetection, the morphology criterion is not used. Rhythm ID can be programmed on or off without any other programmable features. We recommend programming the feature on at implantation.

Single- Versus Dual-Chamber ICD Selection

In patients not in permanent AF who do not have a requirement for dual-chamber pacing to treat bradycardia or to provide resynchronization, the choice between a single- or dual-chamber ICD remains controversial. Intuitively, dual-chamber algorithms, which use atrial and ventricular intracardiac information for rhythm classification, should be superior to single-chamber, ventricular-only detection enhancements. However, early nonrandomized and small randomized studies failed to show any benefit of dual-chamber over single-chamber diagnosis.[35–37] Some studies used first-generation algorithms, which have since been refined; however, the single most common failure mode for dual-chamber algorithms has been atrial sensing malfunction.[38]

Subsequent prospective studies have demonstrated some advantage of dual-chamber over single-chamber discrimination, although the clinical benefit has been absent or modest. In the DETECT SVT trial, Friedman et al[38] randomized 400 dual-chamber ICD recipients to dual- or single-chamber detection. Dual-chamber discrimination reduced the odds of inappropriate detection by 53% (P=0.03). This in turn led to a reduction in the rate of inappropriate therapy (ATP or shock) from 33% to 25%; however, because therapy was not controlled and patients in the single-chamber study arm ended up being more likely to have ATP programmed on, there was no difference in the rate of inappropriate shock. The DATAS trial (Dual Chamber and Atrial Tachyarrhythmias Adverse Events Study)[18] randomized 354 patients to dual-chamber, single-chamber, or simulated single-chamber (dual chamber programmed as single chamber) ICDs and found a modest reduction in the number of patients with >1 inappropriate shock using dual-chamber discrimination (3% versus 12%) and a statistically significant reduction of a composite end point of clinically significant adverse events. Of note, 89% of patients in DATAS had a secondary prevention indication, which suggests greater overlap in rate between SVT and VT and thus a greater reliance on SVT-VT discriminators for adjudication of therapy delivery. In a meta-analysis, Theuns et al[39]

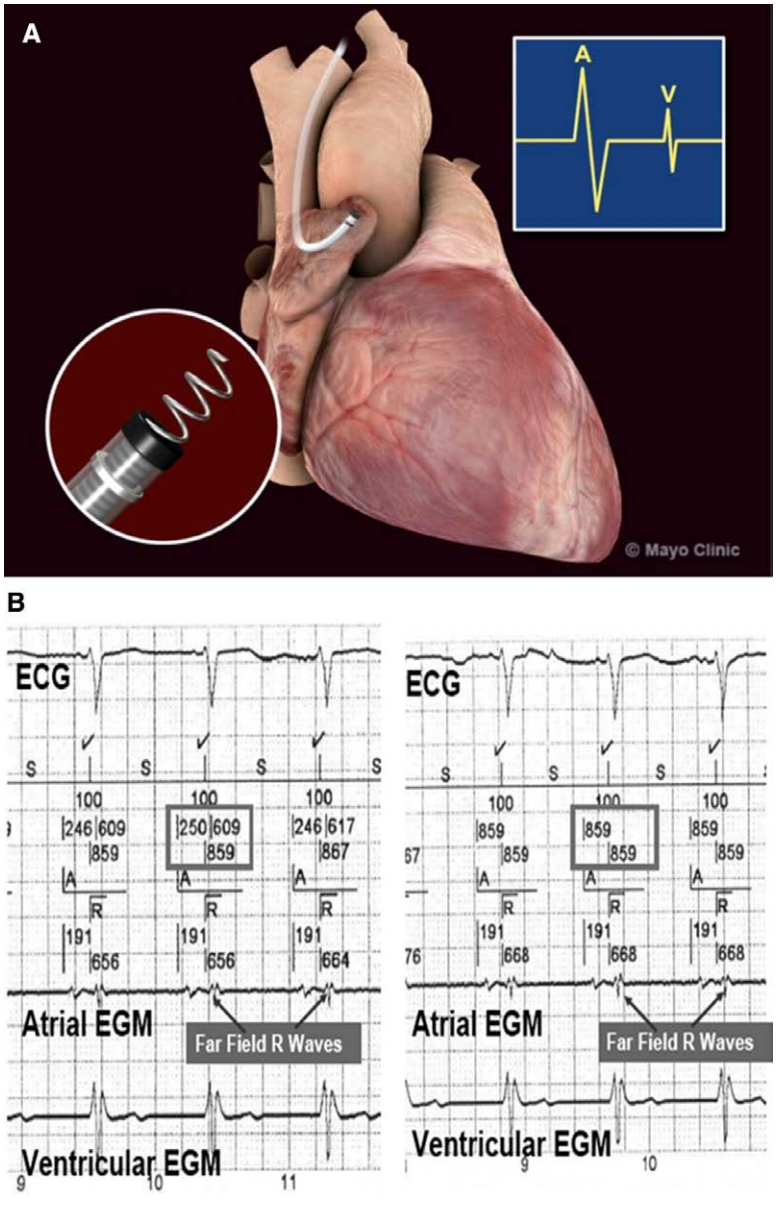


Figure 6. Far-field R-wave oversensing in dual-chamber implantable cardioverter-defibrillator: Recognition and troubleshooting. **A**, The close proximity between the atrial lead in the right atrial appendage and the right ventricle increases the likelihood of sensing far-field R waves on the atrial channel. Reproduced with permission from *Circulation*. 2011;124:477–486. **B**, **Left**, Far-field R waves sensed on the atrial channel. These are counted toward atrial tachyarrhythmia detection and may lead to inappropriate withholding of therapy for ventricular tachycardia. **Right**, Far-field R waves are still present but are no longer detected by the atrial channel after atrial sensitivity is reprogrammed. EGM indicates electrogram.

observed that dual-chamber ICDs reduced the number of inappropriately treated episodes per patient but not the number of patients who received an inappropriate therapy. In contrast to the DATAS study, which enrolled predominantly secondary prevention patients, the recently concluded RAPTURE trial (Reduction and Prevention of Tachyarrhythmias and Shocks Using Reduced Ventricular Pacing With Atrial Algorithms) randomized 100 primary prevention patients to single- or dual-chamber ICDs and used a high rate cutoff (>182 bpm) and a delayed time to therapy (≈17 seconds).[40] Inappropriate shocks were rare in both study arms (2% at 1 year), with no difference between groups. The cost of implantation was $2330 higher in the dual-chamber arm, with no difference in quality of life between groups.

Although controversy remains, taken in aggregate, the data indicate that modern dual-chamber discrimination is superior to single-chamber discrimination when overlap exists in the VT and SVT heart rates (Figure 2B). This favors dual-chamber ICDs for secondary prevention patients and for patients with slow VT. In primary prevention patients, in whom there is little overlap between SVT and VT heart rates, the benefit of using high rate cutoffs and prolonged detection appears to overwhelm any modest additional contribution of dual-chamber SVT-VT discrimination.

Detection Zones and SVT-VT Discriminator Programming

SVT-VT discriminators are linked to the programmed therapy zones. In Boston Scientific ICDs, SVT-VT discriminators are applied in the 2 VT zones but not in the VF zone. In St Jude Medical ICDs, the discriminators can be programmed independently in the 2 VT zones but not in the VF zone. Medtronic ICDs use an SVT limit that is programmed independent of therapy zones, with discriminators applied to all tachycardias with cycle lengths longer than the SVT limit; however, the discriminators function differentially in the VT and VF zones. In the VF zone, the stability counters in PR Logic are not applied (because VF is irregular), in contrast to the Wavelet morphology algorithm. The boundary between zones is selected on the basis of the heart rate at which different ATP cycle lengths or a different number of ATP trials are preferred.

ICD discriminator timeout can override inhibition of therapy for an SVT if the tachycardia persists longer than a programmed time period; however, this can lead to inappropriate shocks for sustained SVTs. When timeouts were programmed off in the PREPARE study, it was not associated with inappropriate inhibition of therapy, and we recommend programming this feature off.[9]

Rhythm Discrimination in the s-ICD

s-ICDs use 3 sensing vectors obtained from 3 subcutaneous electrodes to record electrograms that closely resemble surface ECGs. Cameron Health's s-ICD allows programming of 2 rate zones: A shock zone and a conditional shock zone. Rate alone is used to determine therapy in the shock zone. Morphology discriminators are applied in the conditional shock zone. When a tachycardia is detected in the conditional shock zone, a series of discriminators are applied. First, the waveform is correlated to the stored template obtained in normal rhythm. If the degree of correlation is high (>50%), SVT is detected. If the correlation is low, a beat-to- beat comparison of the tachycardia morphology is performed to identify polymorphic VT. If this detects a monomorphic tachycardia, the algorithm proceeds to compare QRS width between the tachycardia and stored template. If the tachycardia QRS width is wide, VT is detected. The conditional shock zone and shock zone are nominally programmed at 170 and 200 bpm, respectively. We recommend programming the conditional shock zone at 200 bpm and the shock zone at 220 to 230 bpm in primary prevention devices based on studies that used transvenous ICD and s-ICD trial data.

The START (Subcutaneous Versus Transvenous Arrhythmia Recognition Testing) study compared the performance of SVT-VT discriminators in transvenous ICDs with that of s-ICDs using cutaneous electrograms obtained during induced arrhythmias in supine sedated patients.[41] The accuracy of detection of VT/VF was excellent for both devices (100% versus >99%). The s-ICD, however, was more specific in accurately classifying SVTs (98% versus 68%, P=0.001). The superiority of the s-ICD, which uses morphology only in correctly identifying SVTs, compared with transvenous ICDs, which first use interval criteria before applying morphology, could be the result of the greater sensitivity and specificity of morphology discriminators. Furthermore, the s-ICD uses a far-field electrogram that resembles a surface QRS and compares up to 41 points on the electrogram to achieve greater resolution. Importantly, the study was not a real-world comparison, because recorded electrograms were run through devices for the analysis; therefore, additional data are required.

Antitachycardia Pacing

ATP is rapid pacing at a cycle length shorter than VT that terminates reentrant VT by penetrating the circuit and depolarizing the excitable gap to block reentry. ATP can reduce painful shocks, improve quality of life, and lengthen pulse generator life. ATP has been used traditionally to terminate slow VT (<188–200 bpm) with a success rate of 85% to 90% and a 1% to 5% risk of acceleration.[42] Analysis of stored ICD electrograms reveals that the majority of fast ventricular arrhythmias (>180–200 bpm) detected in primary prevention patients are monomorphic VT, which extends the potential application of ATP. The PainFREE Rx II trial (Pacing Fast Ventricular Tachycardia Reduces Shock Therapies) randomized patients to ATP followed by shock or shock alone for the treatment of fast VT (188–250 bpm).[43] In the ATP arm, a single 8-pulse burst of ATP was programmed, followed by shocks for ongoing arrhythmia. ATP was successful in terminating fast VT in 72% of episodes and resulted in a significant reduction in shocks (28% versus 64%). The rate of acceleration of VT, syncope, or mortality was unaffected by ATP use. Quality of life, on the other hand, was significantly better in the ATP arm. The PREPARE study, in addition to programming prolonged detection duration (30 of 40 intervals) and a higher-rate detection threshold (182 bpm) with systematic SVT-VT discrimination (SVT limit 200 bpm), applied ATP for fast VT (182–250 bpm) and used a high-output first shock. The PREPARE cohort had fewer shocks without an increase

in the rate of arrhythmic syncope, although the relative contribution of ATP versus other parameters used is not known.[9] Early delivery of ATP overestimates its efficacy, because up to 40% of episodes may terminate spontaneously with more time.[30] Overall, ATP terminates up to 90% of fast VT with a low risk of acceleration (1% to 5%).[44,45]

Factors that affect the efficacy of ATP include (1) tachycardia cycle length (faster VTs are less responsive), (2) rate and coupling interval of pulses, (3) number of pulses in the drive train, and (4) temporal proximity of the pacing site to the reentrant circuit. ATP can be applied as either burst (the interstimulus interval in the train remains constant) or ramp (each subsequent stimulus in a train is decremented). Burst and ramp pacing have similar efficacy and safety for slow VT; however, for fast VT, burst pacing may offer greater efficacy and smaller probability of acceleration and is generally preferred.[46–48] The rate of ATP is typically programmed at 69% to 88% of VT cycle length, with similar efficacy and safety in this range. Each drive train typically has 8 stimuli, with a rapid decline in efficacy beyond 10 stimuli.[49] The first sequence of ATP is the most effective, with 95% of responsive fast VTs terminated successfully. Only a small minority of patients are responsive to >3 or 4 ATPs. ATP is also efficacious in patients implanted with a cardiac resynchronization device. Although the efficacy and safety of right ventricular, left ventricular, and biventricular ATP are similar in most studies,[50,51] a few observational studies have reported greater success and fewer accelerations with biventricular ATP when treating slow VT.[52] The ADVANCE-CRT trial reported greater efficacy of biventricular ATP in patients with ischemic cardiomyopathy. Thus, in patients with coronary artery disease undergoing implantation of a cardiac resynchronization device, programming biventricular ATP is reasonable.[50]

ATP during capacitor charging for fast VT was introduced to reduce the time to shock if ATP fails and was demonstrated to be successful in terminating 69% of episodes.[53] The shock is aborted if ATP is successful and may lead to early depletion of battery. The Charge Saver feature (Medtronic) may prevent battery depletion by providing ATP before charging, followed by reconfirmation of tachyarrhythmia before initiation of capacitor charging.

Analysis of the return cycle length after delivery of unsuccessful ATP can be used to optimize programming. If the tachycardia is reset, the paced beats are able to penetrate the circuit without causing bidirectional block. The cycle length of the drive train can be shortened or an extrastimulus added at the end of the drive train. If the VT cycle length is unaffected, increasing the number of paced beats facilitates penetrating the circuit by peeling away refractoriness.[14] Analysis of the return cycle length also provides the electrophysiologist with a measure of temporal proximity of the pacing site to the VT circuit. Analysis of the response to ATP can also help in the differential diagnosis of SVT and VT. The occurrence of transient ventriculoatrial (VA) and AV block during ATP is diagnostic of VT and SVT, respectively. The atrial and ventricular sequence at the end of unsuccessful ATP that accelerates the atrial rate can aid in the diagnosis of tachycardia with a 1:1 AV relationship: An AAV response is diagnostic of atrial tachycardia, and a VVA response is diagnostic of VT. A comprehensive analysis of the response to ATP can be found elsewhere.[54]

Medtronic ICDs have a programmable SMART mode for optimization of ATP. If a particular ATP sequence is ineffective in 4 consecutive episodes, SMART mode will cancel that therapy to reduce the delay to treatment of subsequent episodes. However, if misclassification of successful ATP occurs, this feature should be disabled.

Redetection and Reconfirmation

Although first-generation ICDs delivered committed shocks regardless of arrhythmia termination before charge end, contemporary devices reconfirm the persistence of arrhythmia during and after charging. The shock is thus noncommitted, and the charge is painlessly dissipated if the arrhythmia terminates spontaneously. The reconfirmation algorithm is less specific than initial detection, and the rate varies by manufacturer and is programmable in some devices (St Jude Medical). Shocks should be programmed to be noncommitted if possible, although programmability varies by manufacturer: All shocks can be noncommitted in St Jude Medical devices, whereas Medtronic devices allow all shocks to be noncommitted if the Confirmation+ algorithm is turned on. This is in contrast to Medtronic's earlier ICDs that allowed only the first shock to be noncommitted. In Boston Scientific devices, every other shock can be noncommitted; however, the confirmation algorithm before shock is less specific and has been reported to result in shock despite successful termination of tachyarrhythmia.[53,55]

Programming Therapy Zones

Tiered-therapy ICDs have 2 to 3 zones defined by the longest RR interval in each zone. The primary determinants of the boundaries of therapy zones are programming of specific therapies and SVT discrimination. The lower boundary between sinus and VT zone is programmed at approximately 180 to 188 bpm in primary prevention patients[9,28] and at 30 to 60 ms greater than the cycle length of the slowest observed VT (150–160 bpm in most trials) in secondary prevention patients.[16,45,56] Programming in the VT zone is focused on preventing therapy for SVT and nonsustained VT. If 2 VT zones are programmed, the rate at which fewer trials or different cycle lengths of ATP therapy are desired forms the boundary between the 2 zones. We routinely program 3 zones in primary prevention patients, using the slowest VT zone for monitoring only at 160 to 166 bpm (Table). In secondary prevention patients, the slower VT zone is programmed with 3 to 4 sequences of ramp/burst ATP compared with 1-burst ATP followed by shocks in the faster VT zone. SVT discriminators are programmed on in VT zones.

The boundary between the VT and VF zones is determined by the rate at which ATP before charge is no longer desired. SVT discriminators are either not programmable in the VF zone (Boston Scientific and St Jude Medical) or are applied with some limitations (Medtronic). Medtronic ICDs allow programming of SVT limit within the VF zone, but SVT with AV dissociation or irregular VV intervals is no longer classified as AF because of the RR variability that occurs during VF.

Shock Strength, Polarity, and Defibrillation Threshold

Implant testing assesses VF sensing and defibrillation effectiveness. Some form of defibrillation testing has been performed in all of the major trials that have informed guideline-based ICD use,[1–3] but studies to determine whether defibrillation threshold (DFT) testing improves outcomes are lacking. With advances in technology, ≈95% of patients will pass safety-margin implantation criteria, which has led some to question the need for routine testing.[57,58] The SAFE-ICD observational study (Safety of Two Strategies of ICD Management at Implantation) revealed very low and comparable rates of ICD implantation complications and long-term sudden death in patients who did and did not undergo routine DFT testing.[59] Although the role of routine DFT testing requires further investigation, it should be strongly considered in special circumstances, such as nonstandard lead positioning, right-sided devices, amiodarone therapy, pediatric implantations, and use of an s-ICD.

The relative efficacy of incremental shock strength compared with maximum-energy shocks is unknown. If the DFT is known, the first shock in the VT zone is programmed ≥10 J above it, and subsequent shocks are set to maximal energy. The advantage of lower-energy shocks (conservation of battery life, diminished postshock stunning or block) is minimal with current biphasic technology. High-energy shocks are more likely to be successful for VT/VF and SVT (in case of inappropriate therapy) without any appreciable increase in patient discomfort or increase in charge time in contemporary devices.[60] Low-energy shocks also convey a risk of conversion of VT to VF. Hence, maximal shock strength is generally preferred.

ICD manufacturers have different nominal settings for the shock polarity. A meta-analysis by Kroll et al[61] showed that configuring the right ventricular coil as the anode resulted in slightly lower DFT in some patients and a theoretically lower propensity for induction of VF. However, in contemporary ICDs that use biphasic shocks, the shock coil polarity does not significantly alter the DFT in the majority of patients. Reverse-shock polarity, however, can be tried in patients with high DFTs. Our practice is to program 1 shock with reversed polarity in each zone, with the rest programmed per nominal settings.

Do ICD Shocks Increase Mortality?

Whether ICD shocks increase mortality is controversial. Patients who received 1 or more appropriate or inappropriate ICD shocks in the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) had a 5- and 2-fold increased risk of death, respectively.[12] Similar trends were observed in the MADIT II trial: The hazard ratio for mortality was 3.4 and 2.3 in patients who received appropriate and inappropriate shocks, respectively.[11,62] However, whether shocks are an independent predictor of mortality or a marker of disease severity is unknown. Shocks delivered for direct current cardioversion for AF in the AFFIRM study (Atrial Fibrillation Follow-up Investigation of Rhythm Management), DFT testing after ICD implantation, and inappropriate shocks related to oversensing have not been associated with increased mortality,[63,64] which suggests that poor outcomes after shocks may be related to the substrate. Shocks have also been associated with worsening heart failure, electromechanical dissociation, and electrical storm in 14% to 20% of patients.[65–67] ICD shocks are associated with poor quality of life, anxiety, and depression.[68,69]

Optimization of Sensing to Prevent Shocks

Sensing of ventricular electrograms by ICDs has the following, often conflicting requirements: (1) Reliably sense every QRS complex during normal rhythm and small fibrillatory waves during VF; (2) do not sense T waves; and (3) ignore extracardiac and nonphysiological signals. ICDs use dynamic sensitivity or gain to achieve these sensing requirements (Figure 7). After a sensed or paced ventricular event, sensitivity is reduced as a function of the amplitude of the R wave after the postventricular blanking period expires. Then, sensitivity is increased progressively until a subsequent event is sensed or

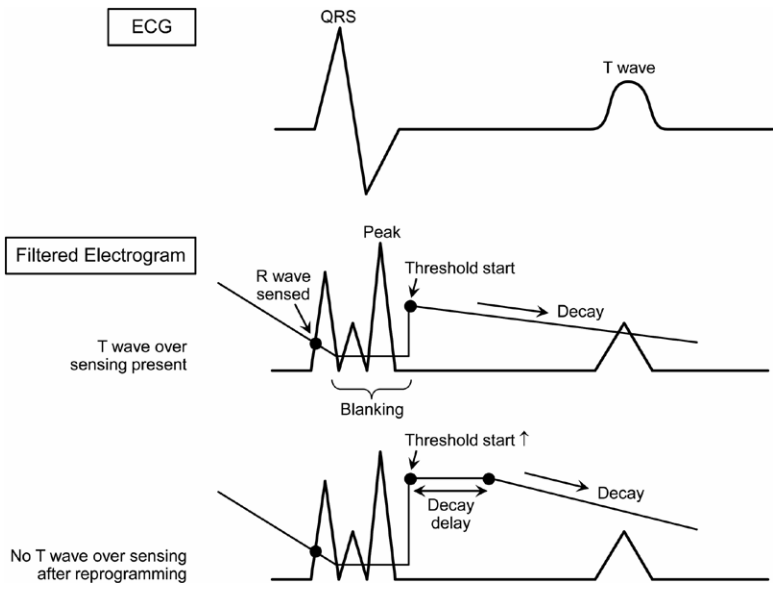


Figure 7. Ventricular dynamic sensing in St Jude Medical implantable cardioverter-defibrillator and programming to prevent T-wave oversensing. Dynamic sensitivity settings are used to prevent T-wave oversensing. The sensitivity is adjusted each cardiac cycle based on the amplitude of the sensed ventricular electrogram. After the ventricular blanking period, the sensitivity is adjusted to a percentage of the peak electrogram amplitude, called the threshold start. The sensitivity is then held constant for a programmable period of time (decay delay), after which it is increased gradually (decay) until another sensed event occurs or the maximum sensitivity is reached. T-wave oversensing can be prevented by increasing the threshold start or decay delay (**bottom**).

the maximum programmed sensitivity is reached. This strategy reduces the risk of T-wave oversensing while maintaining sensitivity for the small deflections of VF. Oversensing of physiological and nonphysiological signals accounts for a small but significant proportion of inappropriate therapy.[70]

Preventing T-Wave Oversensing

Low-amplitude R waves (<3 mV), large T waves, or long QT intervals (sensitivity is maximal at the end of diastole) promote T-wave oversensing, which can result in double counting and inappropriate therapy. Hypertrophic cardiomyopathy, long-QT syndrome, and Brugada syndrome may predispose toward T-wave oversensing. Decreasing ventricular sensitivity reduces the risk of T-wave oversensing but requires a sufficiently large R- to T-wave ratio and may risk VF undersensing. St Jude Medical ICDs allow programming of the decay of sensitivity by programming the starting sensitivity as a percentage of the QRS amplitude (threshold start) and the time delay before the onset of the decay (decay delay; Figure 7). Boston Scientific ICDs use a band-pass filter to modify the sensed signal to ignore low-frequency signals such as T waves and very high-frequency signals such as noise. The T-wave discrimination algorithm in Medtronic ICDs compares the time derivative of the electrogram (which reduces the amplitude of the low-frequency T waves) with a standard sensed electrogram to identify T-wave oversensing. This strategy does not risk VF undersensing. Additionally, in Medtronic ICDs, the sensing vector can be programmed to either true bipolar (tip to ring) or integrated bipolar (tip to distal coil), which can affect the R- and T-wave pattern.

Noise-Detection Algorithms

Algorithms for the discrimination of VT from myopotentials and nonphysiological noise such as electromagnetic interference are manufacturer specific. These algorithms are based on the fact that noise tends to have higher frequency with very short intervals that are unlikely to represent ventricular activation. The Boston Scientific Dynamic Noise Algorithm divides the sensed frequency range into 2 halves and examines the upper half for high-frequency noise. If noise is detected, the device raises the floor of the dynamic sensitivity above the level of the noise to prevent oversensing. This is most useful to prevent myopotential detection.

Surveillance of Lead Fracture

The voluntary withdrawal of the Medtronic Sprint Fidelis lead because of high rates of lead fracture and inappropriate detection of noise led to the development of the Medtronic Lead Integrity Alert algorithm for early detection of lead fracture based on sudden changes in lead impedance and noise detection.[71] The lead-integrity alert is triggered if 2 of 3 events occur: (1) Sudden increase in lead impedance; (2) ≥2 nonsustained tachycardia events (≥5 beats) with intervals <220 ms; and (3) sensing integrity counters with ≥30 short RR-interval counts <140 ms within 3 consecutive days indicative of nonphysiological signals. If criteria are met, the device sounds an audible alarm and extends detection intervals to 30/40. The lead-integrity alert algorithm has been shown to be effective in detecting most Sprint Fidelis lead fractures and reduces the incidence of inappropriate shocks related to lead fracture.[72] The algorithm is downloaded to Medtronic ICDs at the time of programming.

Both the Medtronic and St Jude SecureSense right ventricular lead noise discriminations detect noise caused by lead fracture or a loose set screw. Noise caused by lead defects is more likely to be seen on the near-field electrogram (tip-ring) than the far-field electrogram (coil-can). During a tachycardia, the presence of short intervals on the near-field but not the far-field signal indicates lead noise; short intervals on both the near- and far-field electrograms indicates a true tachycardia.

Programming Bradycardia Pacing Parameters in ICDs: Significance of Short-Long-Short Sequences

Short-long-short (S-L-S) sequences are an important cause of ICD proarrhythmia and can initiate both polymorphic and monomorphic VT. Sweeney et al[73] noted that 8% to 15% of all VT/VF episodes in ICD patients were initiated by an S-L-S sequence that was facilitated by pacing. Furthermore, pacing modes (eg, ventricular pacing avoidance modes such as Managed Ventricular Pacing [Medtronic]) and lower-rate programming (in both VVI and DDD modes) that passively permitted S-L-S sequences initiated 6% to 26% of VT/VF episodes. Programming to avoid S-L-S sequences can be difficult and should be personalized for each patient. Increasing the lower pacing rate in VVI mode does not significantly reduce the risk but can potentially lead to right ventricular apical pacing–mediated heart failure.[74] Ventricular pacing avoidance algorithms reduce the risk of the latter, and we routinely program these on except in special circumstances, such as patients with repolarization abnormalities. Capture-management algorithms can also lead to S-L-S sequences, and the potential proarrhythmic effects need further study. Although pause-prevention algorithms such as rate smoothing may reduce VT burden in selected patients, the randomized, controlled Ventricular Arrhythmia Suppression Trial (VAST) failed to show efficacy in the general ICD population.[75,76] S-L-S sequences can also result from Wenckebach upper-rate behavior, which can be suppressed by programming a dynamic postventricular atrial refractory period, which shortens the postventricular atrial refractory period at higher atrial rates.

ICD Programming During Electrical Storm

Recurrent ICD shocks in the course of electrical storm (defined as ≥3 VT/VF episodes in 24 hours) is deleterious and can lead to heart failure, sympathetic overdrive, and psychological trauma, which potentiates the risk for recurrent VT. Programming for electrical storm focuses on minimizing the number of shocks. In this situation, the duration to detection is increased to avoid therapy for self-terminating episodes. VT may reinitiate immediately after successful ATP, which may lead to misclassification of the ATP as unsuccessful. In some ICDs, the number of intervals to declare an end of episode can be decreased to allow for early classification of return to sinus rhythm. Additionally, redetection after therapy delivery

nominally requires fewer intervals than initial detection and is independently programmable in some ICDs; increasing the number of intervals lowers the risk of additional treatment for an episode with delayed termination after ATP or shock. Increasing the lower pacing rate may help suppress recurrent VT/VF. In hospitalized patients, ICD therapies are often turned off during electrical storm.

Remote Monitoring

Automated, wireless remote monitoring with clinician and patient alerts shortens the time to detection of changes in clinical status and device function and may reduce mortality risk.[70,77,78] It has been reviewed previously.[14]

Conclusions

Proper ICD programming minimizes unnecessary ICD therapy to reduce patient morbidity and mortality. Current evidence supports the use of various strategies to reduce shocks without compromising therapy effectiveness. The extension of detection duration to prevent treatment of self-terminating tachycardia has proven to be the most useful of these strategies, followed by the application of SVT-VT discrimination and the use of ATP to terminate VT.

Disclosures

Dr Friedman has received research support from Medtronic (grant administered by Mayo Clinic for investigator-initiated study), Biotronik, and Cameron Health, and is a speaker or consultant for Bard, Biotronik, Leadex, and Sorin. Dr Madhavan reports no conflicts.

References

1. The Antiarrhythmics Versus Implantable Defibrillators (AVID) Investigators. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. *N Engl J Med*. 1997;337:1576–1583.
2. Buxton AE, Lee KL, Fisher JD, Josephson ME, Prystowsky EN, Hafley G; Multicenter Unsustained Tachycardia Trial Investigators. A randomized study of the prevention of sudden death in patients with coronary artery disease. *N Engl J Med*. 1999;341:1882–1890.
3. Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, Daubert JP, Higgins SL, Brown MW, Andrews ML; Multicenter Automatic Defibrillator Implantation Trial II Investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med*. 2002;346:877–883.
4. Moss AJ, Schuger C, Beck CA, Brown MW, Cannom DS, Daubert JP, Estes NA 3rd, Greenberg H, Hall WJ, Huang DT, Kautzner J, Klein H, McNitt S, Olshansky B, Shoda M, Wilber D, Zareba W; MADIT-RIT Trial Investigators. Reduction in inappropriate therapy and mortality through ICD programming. *N Engl J Med*. 2012;367:2275–2283.
5. Sears SF Jr, Conti JB. Psychological aspects of cardiac devices and recalls in patients with implantable cardioverter defibrillators. *Am J Cardiol*. 2006;98:565–567.
6. Sears SF, Lewis TS, Kuhl EA, Conti JB. Predictors of quality of life in patients with implantable cardioverter defibrillators. *Psychosomatics*. 2005;46:451–457.
7. Pedersen SS, van den Broek KC, Sears SF Jr. Psychological intervention following implantation of an implantable defibrillator: a review and future recommendations. *Pacing Clin Electrophysiol*. 2007;30:1546–1554.
8. Sweeney MO, Sherfesee L, DeGroot PJ, Wathen MS, Wilkoff BL. Differences in effects of electrical therapy type for ventricular arrhythmias on mortality in implantable cardioverter-defibrillator patients. *Heart Rhythm*. 2010;7:353–360.
9. Wilkoff BL, Williamson BD, Stern RS, Moore SL, Lu F, Lee SW, Birgersdotter-Green UM, Wathen MS, Van Gelder IC, Heubner BM, Brown ML, Holloman KK; PREPARE Study Investigators. Strategic programming of detection and therapy parameters in implantable cardioverter-defibrillators reduces shocks in primary prevention patients: results from the PREPARE (Primary Prevention Parameters Evaluation) study. *J Am Coll Cardiol*. 2008;52:541–550.
10. Clementy N, Pierre B, Lallemand B, Marie O, Lemoine E, Cosnay P, Fauchier L, Babuty D. Long-term follow-up on high-rate cut-off programming for implantable cardioverter defibrillators in primary prevention patients with left ventricular systolic dysfunction. *Europace*. 2012;14:968–974.
11. Daubert JP, Zareba W, Cannom DS, McNitt S, Rosero SZ, Wang P, Schuger C, Steinberg JS, Higgins SL, Wilber DJ, Klein H, Andrews ML, Hall WJ, Moss AJ; MADIT II Investigators. Inappropriate implantable cardioverter-defibrillator shocks in MADIT II: frequency, mechanisms, predictors, and survival impact. *J Am Coll Cardiol*. 2008;51:1357–1365.
12. Poole JE, Johnson GW, Hellkamp AS, Anderson J, Callans DJ, Raitt MH, Reddy RK, Marchlinski FE, Yee R, Guarnieri T, Talajic M, Wilber DJ, Fishbein DP, Packer DL, Mark DB, Lee KL, Bardy GH. Prognostic importance of defibrillator shocks in patients with heart failure. *N Engl J Med*. 2008;359:1009–1017.
13. van Rees JB, Borleffs CJ, de Bie MK, Stijnen T, van Erven L, Bax JJ, Schalij MJ. Inappropriate implantable cardioverter-defibrillator shocks: incidence, predictors, and impact on mortality. *J Am Coll Cardiol*. 2011;57:556–562.
14. Koneru JN, Swerdlow CD, Wood MA, Ellenbogen KA. Minimizing inappropriate or "unnecessary" implantable cardioverter-defibrillator shocks: appropriate programming. *Circ Arrhythm Electrophysiol*. 2011;4:778–790.
15. Wilkoff BL, Hess M, Young J, Abraham WT. Differences in tachyarrhythmia detection and implantable cardioverter defibrillator therapy by primary or secondary prevention indication in cardiac resynchronization therapy patients. *J Cardiovasc Electrophysiol*. 2004;15:1002–1009.
16. Dorian P, Philippon F, Thibault B, Kimber S, Sterns L, Greene M, Newman D, Gelaznikas R, Barr A; ASTRID Investigators. Randomized controlled study of detection enhancements versus rate-only detection to prevent inappropriate therapy in a dual-chamber implantable cardioverter-defibrillator. *Heart Rhythm*. 2004;1:540–547.
17. Gasparini M, Menozzi C, Proclemer A, Landolina M, Iacopino S, Carboni A, Lombardo E, Regoli F, Biffi M, Burrone V, Denaro A, Boriani G. A simplified biventricular defibrillator with fixed long detection intervals reduces implantable cardioverter defibrillator (ICD) interventions and heart failure hospitalizations in patients with non-ischaemic cardiomyopathy implanted for primary prevention: the RELEVANT [Role of long dEtection window programming in patients with LEft VentriculAr dysfunction, Non-ischemic eTiology in primary prevention treated with a biventricular ICD] study. *Eur Heart J*. 2009;30:2758–2767.
18. Almendral J, Arribas F, Wolpert C, Ricci R, Adragao P, Cobo E, Navarro X, Quesada A; DATAS Steering Committee; DATAS Writing Committee; DATAS Investigators. Dual-chamber defibrillators reduce clinically significant adverse events compared with single-chamber devices: results from the DATAS (Dual chamber and Atrial Tachyarrhythmias Adverse events Study) trial. *Europace*. 2008;10:528–535.
19. Barold HS, Newby KH, Tomassoni G, Kearney M, Brandon J, Natale A. Prospective evaluation of new and old criteria to discriminate between supraventricular and ventricular tachycardia in implantable defibrillators. *Pacing Clin Electrophysiol*. 1998;21:1347–1355.
20. Brugada J, Mont L, Figueiredo M, Valentino M, Matas M, Navarro-López F. Enhanced detection criteria in implantable defibrillators. *J Cardiovasc Electrophysiol*. 1998;9:261–268.
21. García-Alberola A, Yli-Mäyry S, Block M, Haverkamp W, Martínez-Rubio A, Kottkamp H, Breithardt G, Borggrefe M. RR interval variability in irregular monomorphic ventricular tachycardia and atrial fibrillation. *Circulation*. 1996;93:295–300.
22. Le Franc P, Kuś T, Vinet A, Rocque P, Molin F, Costi P. Underdetection of ventricular tachycardia using a 40 ms stability criterion: effect of antiarrhythmic therapy. *Pacing Clin Electrophysiol*. 1997;20(pt 1):2882–2892.
23. Kettering K, Dörnberger V, Lang R, Vonthein R, Suchalla R, Bosch RF, Mewis C, Eigenberger B, Kühlkamp V. Enhanced detection criteria in implantable cardioverter defibrillators: sensitivity and specificity of the stability algorithm at different heart rates. *Pacing Clin Electrophysiol*. 2001;24(pt 1):1325–1333.
24. Swerdlow CD, Chen PS, Kass RM, Allard JR, Peter CT. Discrimination of ventricular tachycardia from sinus tachycardia and atrial fibrillation in a tiered-therapy cardioverter-defibrillator. *J Am Coll Cardiol*. 1994;23:1342–1355.
25. Glikson M, Swerdlow CD, Gurevitz OT, Daoud E, Shivkumar K, Wilkoff B, Shipman T, Friedman PA. Optimal combination of discriminators for

differentiating ventricular from supraventricular tachycardia by dual-chamber defibrillators. *J Cardiovasc Electrophysiol*. 2005;16:732–739.
25a. Hayes DL, Asirvatham SJ, Friedman PA. Cardiac pacing, defibrillation and resynchronization: a clinical approach. 3rd ed.: Wiley- Blackwell; 2012.
26. Swerdlow CD, Brown ML, Lurie K, Zhang J, Wood NM, Olson WH, Gillberg JM. Discrimination of ventricular tachycardia from supraventricular tachycardia by a downloaded wavelet-transform morphology algorithm: a paradigm for development of implantable cardioverter defibrillator detection algorithms. *J Cardiovasc Electrophysiol*. 2002;13:432–441.
27. Gold MR, Shorofsky SR, Thompson JA, Kim J, Schwartz M, Bocek J, Lovett EG, Hsu W, Morris MM, Lang DJ. Advanced rhythm discrimination for implantable cardioverter defibrillators using electrogram vector timing and correlation. *J Cardiovasc Electrophysiol*. 2002;13:1092–1097.
28. Bardy GH, Lee KL, Mark DB, Poole JE, Packer DL, Boineau R, Domanski M, Troutman C, Anderson J, Johnson G, McNulty SE, Clapp-Channing N, Davidson-Ray LD, Fraulo ES, Fishbein DP, Luceri RM, Ip JH; Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *N Engl J Med*. 2005;352:225–237.
29. Bailin SJ, Niebauer M, Tomassoni G, Leman R; Photon Investigators. Clinical investigation of a new dual-chamber implantable cardioverter defibrillator with improved rhythm discrimination capabilities. *J Cardiovasc Electrophysiol*. 2003;14:144–149.
30. Wilkoff BL, Kühlkamp V, Volosin K, Ellenbogen K, Waldecker B, Kacet S, Gillberg JM, DeSouza CM. Critical analysis of dual-chamber implantable cardioverter-defibrillator arrhythmia detection: results and technical considerations. *Circulation*. 2001;103:381–386.
31. De Groot JR, Schroeder-Tanka JM, Visser J, Willems AR, De Voogt WG. Clinical results of far-field R-wave reduction with a short tip-ring electrode. *Pacing Clin Electrophysiol*. 2008;31:1554–1559.
32. de Voogt W, van Hemel N, Willems A, Visser J, Chitre Y, Bornzin G, Helland J. Far-field R-wave reduction with a novel lead design: experimental and human results. *Pacing Clin Electrophysiol*. 2005;28:782–788.
33. Asirvatham SJ, Bruce CJ, Danielsen A, Johnson SB, Okumura Y, Kathmann E, Packer DL, Friedman PA. Intramyocardial pacing and sensing for the enhancement of cardiac stimulation and sensing specificity. *Pacing Clin Electrophysiol*. 2007;30:748–754.
34. Henz BD, Friedman PA, Bruce CJ, Okumura Y, Johnson SB, Danielsen A, Packer DL, Asirvatham SJ. Synchronous ventricular pacing without crossing the tricuspid valve or entering the coronary sinus–preliminary results. *J Cardiovasc Electrophysiol*. 2009;20:1391–1397.
35. Deisenhofer I, Kolb C, Ndrepepa G, Schreieck J, Karch M, Schmieder S, Zrenner B, Schmitt C. Do current dual chamber cardioverter defibrillators have advantages over conventional single chamber cardioverter defibrillators in reducing inappropriate therapies? A randomized, prospective study. *J Cardiovasc Electrophysiol*. 2001;12:134–142.
36. Kulhlkamp V, Dörnberger V, Mewis C, Suchalla R, Bosch RF, Seipel L. Clinical experience with the new detection algorithms for atrial fibrillation of a defibrillator with dual chamber sensing and pacing. *J Cardiovasc Electrophysiol*. 1999;10:905–915.
37. Theuns DA, Klootwijk AP, Goedhart DM, Jordaens LJ. Prevention of inappropriate therapy in implantable cardioverter-defibrillators: results of a prospective, randomized study of tachyarrhythmia detection algorithms. *J Am Coll Cardiol*. 2004;44:2362–2367.
38. Friedman PA, McClelland RL, Bamlet WR, Acosta H, Kessler D, Munger TM, Kavesh NG, Wood M, Daoud E, Massumi A, Schuger C, Shorofsky S, Wilkoff B, Glikson M. Dual-chamber versus single-chamber detection enhancements for implantable defibrillator rhythm diagnosis: the Detect Supraventricular Tachycardia Study. *Circulation*. 2006;113:2871–2879.
39. Theuns DA, Rivero-Ayerza M, Boersma E, Jordaens L. Prevention of inappropriate therapy in implantable defibrillators: a meta-analysis of clinical trials comparing single-chamber and dual-chamber arrhythmia discrimination algorithms. *Int J Cardiol*. 2008;125:352–357.
40. Friedman PA, Slusser J, Hodge DO, Bradley DJ, Koestler C, Kusumoto F, Munger TM, Bailey K, Militanu A, Glikson M. Prospective randomized trial of dual chamber vs. single chamber ICDs to minimize shocks in optimally programmed devices *Heart Rhythm*. 2012;9:1583.
41. Gold MR, Theuns DA, Knight BP, Sturdivant JL, Sanghera R, Ellenbogen KA, Wood MA, Burke MC. Head-to-head comparison of arrhythmia discrimination performance of subcutaneous and transvenous ICD arrhythmia detection algorithms: the START study. *J Cardiovasc Electrophysiol*. 2012;23:359–366.
42. Sweeney MO. Antitachycardia pacing for ventricular tachycardia using implantable cardioverter defibrillators:. *Pacing Clin Electrophysiol*. 2004;27:1292–1305.
43. Wathen MS, DeGroot PJ, Sweeney MO, Stark AJ, Otterness MF, Adkisson WO, Canby RC, Khalighi K, Machado C, Rubenstein DS, Volosin KJ. Prospective randomized multicenter trial of empirical antitachycardia pacing versus shocks for spontaneous rapid ventricular tachycardia in patients with implantable cardioverter-defibrillators: Pacing fast Ventricular Tachycardia Reduces Shock Therapies (PainFREE Rx II) Trial results. *Circulation*. 2004;110:2591–2596.
44. Grimm W, Plachta E, Maisch B. Antitachycardia pacing for spontaneous rapid ventricular tachycardia in patients with prophylactic cardioverter-defibrillator therapy. *Pacing Clin Electrophysiol*. 2006;29:759–764.
45. Wilkoff BL, Ousdigian KT, Sterns LD, Wang ZJ, Wilson RD, Morgan JM; EMPIRIC Trial Investigators. A comparison of empiric to physician-tailored programming of implantable cardioverter-defibrillators: results from the prospective randomized multicenter EMPIRIC trial. *J Am Coll Cardiol*. 2006;48:330–339.
46. Gillis AM, Leitch JW, Sheldon RS, Morillo CA, Wyse DG, Yee R, Klein GJ, Mitchell LB. A prospective randomized comparison of autodecremental pacing to burst pacing in device therapy for chronic ventricular tachycardia secondary to coronary artery disease. *Am J Cardiol*. 1993;72:1146–1151.
47. Peters RW, Zhang X, Gold MR. Clinical predictors and efficacy of antitachycardia pacing in patients with implantable cardioverter defibrillators: the importance of the patient's sex. *Pacing Clin Electrophysiol*. 2001;24:70–74.
48. Gulizia MM, Piraino L, Scherillo M, Puntrello C, Vasco C, Scianaro MC, Mascia F, Pensabene O, Giglia S, Chiarandà G, Vaccaro I, Mangiameli S, Corrao D, Santi E, Grammatico A; PITAGORA ICD Study Investigators. A randomized study to compare ramp versus burst antitachycardia pacing therapies to treat fast ventricular tachyarrhythmias in patients with implantable cardioverter defibrillators: the PITAGORA ICD trial. *Circ Arrhythm Electrophysiol*. 2009;2:146–153.
49. Charos GS, Haffajee CI, Gold RL, Bishop RL, Berkovits BV, Alpert JS. A theoretically and practically more effective method for interruption of ventricular tachycardia: self-adapting autodecremental overdrive pacing. *Circulation*. 1986;73:309–315.
50. Gasparini M, Anselme F, Clementy J, Santini M, Martínez-Ferrer J, De Santo T, Santi E, Schwab JO; ADVANCE CRT-D Investigators. BIVentricular versus right ventricular antitachycardia pacing to terminate ventricular tachyarrhythmias in patients receiving cardiac resynchronization therapy: the ADVANCE CRT-D Trial. *Am Heart J*. 2010;159:1116–1123.e2.
51. Yee R, Birgersdotter-Green U, Belk P, Jackson T, Christensen J, Wathen MS. The relationship between pacing site and induction or termination of sustained monomorphic ventricular tachycardia by antitachycardia pacing. *Pacing Clin Electrophysiol*. 2010;33:27–32.
52. Haghjoo M, Hajahmadi M, Fazelifar AF, Sadr-Ameli MA. Efficacy and safety of different antitachycardia pacing sites in the termination of ventricular tachycardia in patients with biventricular implantable cardioverter-defibrillator. *Europace*. 2011;13:509–513.
53. Schoels W, Steinhaus D, Johnson WB, O'Hara G, Schwab JO, Jenniskens I, Degroot PJ, Tang F, Helmling E; EnTrust Clinical Study Investigators. Optimizing implantable cardioverter-defibrillator treatment of rapid ventricular tachycardia: antitachycardia pacing therapy during charging. *Heart Rhythm*. 2007;4:879–885.
54. Swerdlow CD, Friedman PA. Advanced ICD troubleshooting: part I. *Pacing Clin Electrophysiol*. 2005;28:1322–1346.
55. Bernier M, Essebag V. Inappropriate shock despite successful termination of supraventricular tachycardia by antitachycardia pacing during charging. *Pacing Clin Electrophysiol*. 2010;33:e81–e83.
56. Mansour F, Khairy P. Programming ICDs in the modern era beyond out-of-the box settings. *Pacing Clin Electrophysiol*. 2011;34:506–520.
57. Strickberger SA, Klein GJ. Is defibrillation testing required for defibrillator implantation? *J Am Coll Cardiol*. 2004;44:88–91.
58. Swerdlow CD, Russo AM, Degroot PJ. The dilemma of ICD implant testing. *Pacing Clin Electrophysiol*. 2007;30:675–700.
59. Brignole M, Occhetta E, Bongiorni MG, Proclemer A, Favale S, Iacopino S, Calò L, Vado A, Buja G, Mascioli G, Quartieri F, Tritto M, Parravicini U, Castro A, Tomasi C, Villani GQ, D'Acri MG, Klersy C, Gasparini M; SAFE-ICD Study Investigators. Clinical evaluation of defibrillation testing in an unselected population of 2,120 consecutive patients undergoing first implantable cardioverter-defibrillator implant. *J Am Coll Cardiol*. 2012;60:981–987.
60. Steinhaus DM, Cardinal DS, Mongeon L, Musley SK, Foley L, Corrigan S. Internal defibrillation: pain perception of low energy shocks. *Pacing Clin Electrophysiol*. 2002;25:1090–1093.
61. Kroll MW, Efimov IR, Tchou PJ. Present understanding of shock polarity for internal defibrillation: the obvious and non-obvious clinical implications. *Pacing Clin Electrophysiol*. 2006;29:885–891.

62. Moss AJ, Greenberg H, Case RB, Zareba W, Hall WJ, Brown MW, Daubert JP, McNitt S, Andrews ML, Elkin AD; Multicenter Automatic Defibrillator Implantation Trial-II (MADIT-II) Research Group. Long-term clinical course of patients after termination of ventricular tachyarrhythmia by an implanted defibrillator. *Circulation*. 2004;110:3760–3765.
63. Bhavnani SP, Kluger J, Coleman CI, White CM, Guertin D, Shafi NA, Yarlagadda RK, Clyne CA. The prognostic impact of shocks for clinical and induced arrhythmias on morbidity and mortality among patients with implantable cardioverter-defibrillators. *Heart Rhythm*. 2010;7:755–760.
64. Elayi CS, Whitbeck MG, Charnigo R, Shah J, Macaulay TE, Morales G, Gurley JC, Kakavand B, Thal S, Ching CK, Khaykin Y, Verma A, Barrett C, Bai R, Di Biase L, Patwardhan A, Moliterno DJ, Natale A; AFFIRM Study Investigators. Is there an association between external cardioversions and long-term mortality and morbidity? Insights from the Atrial Fibrillation Follow-up Investigation of Rhythm Management study. *Circ Arrhythm Electrophysiol*. 2011;4:465–469.
65. Arya A, Haghjoo M, Dehghani MR, Fazelifar AF, Nikoo MH, Bagherzadeh A, Sadr-Ameli MA. Prevalence and predictors of electrical storm in patients with implantable cardioverter-defibrillator. *Am J Cardiol*. 2006;97:389–392.
66. Gatzoulis KA, Andrikopoulos GK, Apostolopoulos T, Sotiropoulos E, Zervopoulos G, Antoniou J, Brili S, Stefanadis CI. Electrical storm is an independent predictor of adverse long-term outcome in the era of implantable defibrillator therapy. *Europace*. 2005;7:184–192.
67. Mitchell LB, Pineda EA, Titus JL, Bartosch PM, Benditt DG. Sudden death in patients with implantable cardioverter defibrillators: the importance of post-shock electromechanical dissociation. *J Am Coll Cardiol*. 2002;39:1323–1328.
68. Bourke JP, Turkington D, Thomas G, McComb JM, Tynan M. Florid psychopathology in patients receiving shocks from implanted cardioverter-defibrillators. *Heart*. 1997;78:581–583.
69. Schron EB, Exner DV, Yao Q, Jenkins LS, Steinberg JS, Cook JR, Kutalek SP, Friedman PL, Bubien RS, Page RL, Powell J. Quality of life in the antiarrhythmics versus implantable defibrillators trial: impact of therapy and influence of adverse symptoms and defibrillator shocks. *Circulation*. 2002;105:589–594.
70. Saxon LA, Hayes DL, Gilliam FR, Heidenreich PA, Day J, Seth M, Meyer TE, Jones PW, Boehmer JP. Long-term outcome after ICD and CRT implantation and influence of remote device follow-up: the ALTITUDE survival study. *Circulation*. 2010;122:2359–2367.
71. Swerdlow CD, Gunderson BD, Ousdigian KT, Abeyratne A, Stadler RW, Gillberg JM, Patel AS, Ellenbogen KA. Downloadable algorithm to reduce inappropriate shocks caused by fractures of implantable cardioverter-defibrillator leads. *Circulation*. 2008;118:2122–2129.
72. Kallinen LM, Hauser RG, Tang C, Melby DP, Almquist AK, Katsiyiannis WT, Gornick CC. Lead integrity alert algorithm decreases inappropriate shocks in patients who have Sprint Fidelis pace-sense conductor fractures. *Heart Rhythm*. 2010;7:1048–1055.
73. Sweeney MO, Ruetz LL, Belk P, Mullen TJ, Johnson JW, Sheldon T. Bradycardia pacing-induced short-long-short sequences at the onset of ventricular tachyarrhythmias: a possible mechanism of proarrhythmia? *J Am Coll Cardiol*. 2007;50:614–622.
74. Himmrich E, Przibille O, Zellerhoff C, Liebrich A, Rosocha S, Andreas K, Nebeling D, Omogbehin B, Meyer J. Proarrhythmic effect of pacemaker stimulation in patients with implanted cardioverter-defibrillators. *Circulation*. 2003;108:192–197.
75. Friedman PA, Jalal S, Kaufman S, Villareal R, Brown S, Hahn SJ, Lerew DR. Effects of a rate smoothing algorithm for prevention of ventricular arrhythmias: results of the Ventricular Arrhythmia Suppression Trial (VAST). *Heart Rhythm*. 2006;3:573–580.
76. Viskin S, Fish R, Roth A, Copperman Y. Prevention of torsade de pointes in the congenital long QT syndrome: use of a pause prevention pacing algorithm. *Heart*. 1998;79:417–419.
77. Crossley GH, Boyle A, Vitense H, Chang Y, Mead RH; CONNECT Investigators. The CONNECT (Clinical Evaluation of Remote Notification to Reduce Time to Clinical Decision) trial: the value of wireless remote monitoring with automatic clinician alerts. *J Am Coll Cardiol*. 2011;57:1181–1189.
78. Varma N, Epstein AE, Irimpen A, Schweikert R, Love C; TRUST Investigators. Efficacy and safety of automatic remote monitoring for implantable cardioverter-defibrillator follow-up: the Lumos-T Safely Reduces Routine Office Device Follow-up (TRUST) trial. *Circulation*. 2010;122:325–332.

KEY WORDS: defibrillators, implantable ■ heart arrest ■ shock