2015 HRS/EHRA/APHRS/SOLAECE expert consensus statement on optimal implantable cardioverter-defibrillator programming and testing

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KEYWORDS Implantable cardioverter-defibrillator; Bradycardia mode and rate; Tachycardia detection; Tachycardia therapy; Defibrillation testing; Programming

ABBREVIATIONS **aCRT** = adaptive cardiac resynchronization therapy; **AF** = atrial fibrillation; **ATP** = antitachycardia pacing; **CI** = confidence interval; **CL** = cycle length; **CRT** = cardiac resynchronization therapy; **CRT-D** = cardiac resynchronization therapy–defibrillator; **DT** = defibrillation testing; **EEG** = electroencephalography; **EGM** = electrogram; **HF** = heart failure; **HR** = hazard ratio; **ICD** = implantable cardioverter-defibrillator; **LV** = left ventricle; **LVEF** = left ventricular ejection fraction; **MI** = myocardial infarction; **MVP** = managed ventricular pacing; **NCDR** = National Cardiovascular Data Registry; **NYHA** = New York Heart Association; **OR** = odds ratio; **PEA** = peak endocardial acceleration; **PVC** = premature ventricular contraction; **RCT** = randomized clinical trial; **RV** = right ventricle; **SCD** = sudden cardiac death; **S-ICD** = subcutaneous implantable cardioverter-defibrillator; **SVT** = supraventricular tachycardia; **TIA** = transient ischemic attack; **VF** = ventricular fibrillation; **VT** = ventricular tachycardia (Heart Rhythm 2016;13:e50–e86)

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Introduction

Implantable cardioverter-defibrillator (ICD) therapy is clearly an effective therapy for selected patients in definable populations. The benefits and risks of ICD therapy are directly impacted by programming and surgical decisions. This flexibility is both a great strength and a weakness, for which there has been no prior official discussion or guidance. It is the consensus of the 4 continental electrophysiology societies that there are 4 important clinical issues for which there are sufficient ICD clinical and trial data to provide evidence-based expert guidance. This document systematically describes the greater than 80% (83%–100%, mean 96%) required consensus achieved for each recommendation by official balloting in regard to the programming of (1) bradycardia mode and rate, (2) tachycardia detection, (3) tachycardia therapy, and (4) the intraprocedural testing of defibrillation efficacy. Representatives nominated by the Heart Rhythm Society (HRS), European Heart Rhythm Association (EHRA), Asian Pacific Heart Rhythm Society (APHRS), and the *Sociedad Latinoamericana de Estimulacion Cardiaca y Electrofisiologia* (SOLAECE-Latin American Society of Cardiac Pacing and Electrophysiology) participated in the project definition, the literature review, the recommendation development, the writing of the document, and its approval. The 32 recommendations were balloted by the 35 writing committee members and were approved by an average of 96%.

The classification of the recommendations and the level of evidence follow the recently updated ACC/AHA standard.[1,2] Class I is a strong recommendation, denoting a benefit greatly exceeding risk. Class IIa is a somewhat weaker recommendation, with a benefit probably exceeding risk, and Class IIb denotes a benefit equivalent to or possibly exceeding risk. Class III is a recommendation against a specific treatment because either there is no net benefit or there is net harm. Level of Evidence A denotes the highest level of evidence from more than 1 high-quality randomized clinical trial (RCT), a meta-analysis of high-quality RCTs, or RCTs corroborated by high-quality registry studies. Level of evidence B indicates moderate-quality evidence from either RCTs with a meta-analysis (B-R) or well-executed nonrandomized trials with a meta-analysis (B-NR). Level of evidence C indicates randomized or nonrandomized observational or registry studies with limited data (C-LD) or from expert opinions (C-EO) based on clinical experience in the absence of credible published evidence. These recommendations were also subject to a 1-month public comment period. Each society then officially reviewed, commented, edited, and endorsed the final document and recommendations. All author and peer reviewer disclosure information is provided in Appendix A.

The care of individual patients must be provided in context of their specific clinical condition and the data available on that patient. Although the recommendations in this document provide guidance for a strategic approach to ICD programming, as an individual patient's condition changes or progresses and additional clinical considerations become apparent, the programming of their ICDs must reflect those changes. Remote and in-person interrogations of the ICD and clinical monitoring must continue to inform the programming choices made for each patient. The recommendations in this document specifically target adult patients and might not be applicable to pediatric patients, particularly when programming rate criteria.

Please consider that each ICD has specific programmable options that might not be specifically addressed by the 32 distinctive recommendations in this document. Appendix B, published online (http://www.hrsonline.org/appendix-b), contains the writing committee's translations specific to each manufacturer and is intended to best approximate the recommended behaviors for each available ICD model.

Bradycardia Mode and Rate Programming

Single- or Dual-Chamber Pacing Mode

Evidence. Because the ICD is primarily indicated for tachycardia therapy, there might be some uncertainty regarding optimal bradycardia management for ICD patients. Data from clinical studies adequately address only the programmed mode rather than the number of leads implanted, the number of chambers stimulated, or how frequently the patients required bradycardia support. It is of note that most information on pacing modes has been collected from pacemaker patients, and these patients are clinically distinct from ICD recipients. Dual-chamber pacing (atrial and ventricular) has been compared with single-chamber pacing (atrial or ventricular) in patients with bradycardia in 5 multicenter, parallel, randomized trials, in 1 meta-analysis of randomized trials, and in 1 systematic review that also included 30 randomized crossover comparisons and 4 economic analyses.[3–9] Meta analyses comparing dual-chamber to single-chamber ICDs did not evaluate pacing modes.[10,11] Compared with single-chamber pacing,

dual-chamber pacing results in small but potentially significant benefits in patients with sinus node disease and/or atrioventricular block. No difference in mortality has been observed between ventricular pacing modes and dual-chamber pacing modes. Dual-chamber pacing was associated with a lower rate of atrial fibrillation (AF) and stroke.[12] The benefit in terms of AF prevention was more marked in trials comprised of patients with sinus node disease. Although trends in favor of dual-chamber pacing have been observed in some trials, there was no benefit in terms of heart failure (HF). In patients without symptomatic bradycardia, however, the Dual Chamber and VVI Implantable Defibrillator (DAVID) trial in ICD recipients showed that one specific choice of dual-chamber rate-responsive (DDDR) programming parameters led to poorer outcomes than VVI backup pacing, most likely secondary to unnecessary right ventricular (RV) pacing. The fact that RV stimulation was responsible was reinforced in the DAVID II trial, in which AAI pacing was demonstrated to be noninferior to VVI backup pacing.[13]

Approximately a quarter of patients with either sinus node disease or atrioventricular block develop "pacemaker syndrome" with VVI pacing usually associated with retrograde (ventricular to atrial) conduction, which in turn is associated with a reduction in the quality of life.[14] In crossover trials, symptoms of pacemaker syndrome (dyspnea, dizziness, palpitations, pulsations, and chest pain) were reduced by reprogramming to a dual-chamber mode.[14] Dual-chamber pacing is associated with better exercise performance compared with single-chamber VVI pacing without rate adaptation, but produces similar exercise performance when compared with rate-responsive VVIR pacing. Because of the additional lead, dual-chamber devices involve longer implantation times, have a higher risk of complications, and are more expensive. However, because of the additional clinical consequences of pacemaker syndrome and AF (and its sequelae), the overall cost difference between single- and dual-pacing systems is moderated.

In patients with persistent sinus bradycardia, atrial rather than ventricular dual-chamber pacing is the pacing mode of choice. There is evidence for superiority of atrial-based pacing over ventricular pacing for patients who require pacing for a significant proportion of the day. The evidence is stronger for patients with sinus node disease, in whom dual-chamber pacing confers a modest reduction in AF and stroke, but not in hospitalization for HF or death compared with ventricular pacing. In patients with acquired atrioventricular block, large randomized parallel trials were unable to demonstrate the superiority of dual-chamber pacing over ventricular pacing with regard to hard clinical endpoints of mortality and morbidity.[4,6–8] The benefit of dual-chamber over ventricular pacing is primarily due to the avoidance of pacemaker syndrome and to improved exercise capacity.[14] Even if it is a softer endpoint, pacemaker syndrome is associated with a reduction in quality of life that justifies the preference for dual-chamber pacing when reasonable; thus, there is strong evidence for the superiority of dual-chamber pacing over ventricular pacing that is limited to symptom improvement. Conversely, there is strong evidence of non-superiority with regard to survival and morbidity. The net result is that the indications for programming the dual-chamber modes are weaker and the choice regarding the pacing mode should be individualized, taking into consideration the increased complication risk and costs of dual-chamber devices. Because ICD patients usually do not require bradycardia support, with the exception of patients who require cardiac resynchronization, programming choices should avoid pacing and in particular avoid single ventricular pacing, if possible.[15,16]

Programming of Rate Modulation

The benefit of rate response programming has been evaluated in patients with bradycardia in 5 multicenter, randomized trials and in 1 systematic review that also included 7 single-center studies.[17–22] Most of these data were obtained from pacemaker studies and must be interpreted in that light.

Although there is evidence of the superiority of VVIR pacing compared with VVI pacing in improving quality of life and exercise capacity, improvements in exercise capacity with DDDR compared with DDD have been inconsistent. In 2 small studies on patients with chronotropic incompetence comparing DDD and DDDR pacing, the latter had improved quality of life and exercise capacity; however, a larger, multicenter randomized trial (Advanced Elements of Pacing Randomized Controlled Trial [ADEPT]) failed to show a difference in patients with a modest blunted heart rate response to exercise.[17–19] In addition, DDDR programming in cardiac resynchronization therapy (CRT) patients has the potential to impair AV synchrony and timing. It should be noted that trials evaluating CRT generally did not use rate-responsive pacing, and many in fact avoided atrial stimulation using atrial sensed and ventricular paced pacing modes with a lower base rate. However, the Pacing Evaluation-Atrial Support Study in Cardiac Resynchronization Therapy (PEGASUS CRT) trial is the exception and did not demonstrate adverse impact on mortality and HF events.[23]

Sinus Node Disease

In patients with persistent or intermittent sinus node dysfunction or chronotropic incompetence, the first choice is DDDR with algorithms responding to intermittent atrioventricular conduction. There is sufficient evidence for the superiority of VVIR compared with VVI in improving quality of life and exercise capacity. The evidence is much weaker in dual-chamber pacing (DDDR vs DDD).

Although only an issue when there is some concomitant AV block, the upper rate limit should be programmed higher than the fastest spontaneous sinus rhythm to avoid upper rate limit behavior. To avoid symptomatic bradycardia, the lower rate should be programmed on an individual basis, according to the clinical characteristics and the underlying cardiac substrate of the patient.

Atrial Fibrillation and Atrioventricular Block

Patients with permanent AF and either spontaneous or AV junctional ablation-induced high-degree atrioventricular block have little to no chronotropic response to exercise; thus, VVIR pacing is associated with better exercise performance, improved daily activities, improved quality of life, and decreased symptoms of shortness of breath, chest pain, and heart palpitations, compared with VVI.[20–22,24–26] Therefore, rate-adaptive pacing is the first choice of pacing mode; fixed-rate VVI pacing should be abandoned in patients with permanent AF and atrioventricular block. It is the experts' opinion that the minimum rate can be programmed higher (e.g., 70 bpm) than for sinus rhythm patients, in an attempt to compensate for the loss of active atrial filling. In addition, the maximum sensor rate should be programmed restrictively (e.g., 110–120 bpm) to avoid "overpacing" (i.e., pacing with a heart rate faster than necessary), which can be symptomatic, particularly in patients with coronary artery disease. In a small study, however, it was found that rate-responsive pacing could be safe and effective in patients with angina pectoris, without an increase in subjective or objective signs of ischemia.[25] The lower rate should be programmed on an individual basis, according to the clinical characteristics and the underlying cardiac substrate of the patient. The clinical benefit of programming a lower resting rate at night based on internal clocks has not been evaluated in ICD patients. There is some concern that atrioventricular junction ablation and permanent ventricular pacing might predispose the patient to an increased risk of sudden cardiac death (SCD) related to a bradycardia-dependent prolongation of the QT interval. This risk might be overcome by setting the ventricular pacing rate to a minimum of 80 or 90 bpm for the first 1–2 months following the atrioventricular junction ablation, then reducing it to a conventional 60–70 bpm.[27,28] Not all patients with AF and milder forms of atrioventricular block will require a high percentage of ventricular pacing or have a wide QRS. Physicians should consider the risk of increasing preexisting left ventricular (LV) dysfunction with RV pacing vs improved chronotropic responsiveness and the potential value of CRT.

Intact Atrioventricular Conduction

Right Ventricular Pacing

The results of a number of large-scale, prospective randomized trials demonstrated a significant reduction in AF in pacemaker patients with atrial-based pacing (AAI or DDD) compared with patients with ventricular-based pacing.[4,8,29] In the Mode Selection Trial, which enrolled 2010 patients with sick sinus syndrome, the risk of AF increased linearly with the increasing percentage of RV pacing.[30] At the same time, deleterious effects of RV pacing in patients with LV dysfunction (left ventricular ejection fraction [LVEF] $\leq$40%) implanted with dual-chamber ICD systems were observed in the Dual Chamber and VVI Implantable Defibrillator (DAVID) trial, which included 506 ICD patients without indications for bradycardia pacing. Patients within the DDDR-70 group (with paced and sensed atrioventricular delays of 170 and 150 ms, respectively, in most of the DDDR group patients) showed a trend toward higher mortality and an increased incidence of HF compared with the patients programmed to ventricular backup pacing—the VVI-40 group. Within the DDDR-70 group, there were more cardiac events when the percentage of ventricular pacing exceeded 40% ($P = .09$) compared with patients with $<$40% of RV pacing, although almost all the patients had $>$95% RV stimulation (DDDR-70) or $<$5% RV stimulation (VVI-40).[31,32] However, a more detailed *post hoc* analysis of the Inhibition of Unnecessary RV Pacing With AtrialVentricular Search Hysteresis in ICDs (INTRINSIC RV) trial revealed that the most favorable clinical results were not in the VVI groups with the least percentage of RV pacing but in the subgroup that had DDD pacing with longer atrioventricular delays and 11%–19% of ventricular pacing. This parameter selection probably helped patients to avoid exceedingly low heart rates while preserving intrinsic atrioventricular conduction most of the time.[31,33] In the Second Multicenter Automated Defibrillator Implantation Trial (MADIT II), a higher risk of HF was observed in patients who had a greater than 50% burden of RV pacing.[34] In another large observational study of 456 ICD patients without HF at baseline, a high RV pacing burden (RV pacing more than 50% of the time) was associated with an increased risk of HF events and appropriate ICD shocks.[35] Optimally, RV stimulation should be avoided, but the precise tradeoff between the percentage of ventricular pacing and atrioventricular timing is unclear in non-CRT patients.

Non-CRT Devices: Algorithms to Reduce Right Ventricular Stimulation

The importance of reducing or avoiding RV pacing in ICD patients with LV dysfunction was illustrated in the DAVID trial.[31] The feasibility of algorithms designed to decrease the burden of unnecessary ventricular pacing has been demonstrated in patients with dual-chamber pacemakers.[36–38] These algorithms usually provide functional AAI pacing with monitoring of atrioventricular conduction and an automatic mode switch from AAI to DDD during episodes of atrioventricular block. Some studies directly compared various algorithms to decrease ventricular pacing, showing that a "managed ventricular pacing" (MVP) algorithm resulted in greater ventricular pacing reduction than an "atrioventricular search" algorithm[39,40]: however, no randomized studies comparing these two algorithms with respect to important cardiovascular endpoints (e.g., HF, cardiac death) have been performed. The results of the studies on these pacing algorithms are summarized in Table 1.

Unnecessary RV pacing should be minimized by using specific algorithms or programming longer atrioventricular delays, and this process is more important for patients with a higher risk of AF or who already have poorer LV function.[49] Patients with longer baseline PR intervals have a higher risk of AF regardless of the percentage of ventricular pacing or

Table 1 Influence of pacing modes and algorithms on clinical endpoints

Study	Patients (PM/ICD)	Results and remarks
SAVE PACe, randomized multicenter (2007)[41]	1065 (PM)	40% relative risk reduction of AF in the MVP group compared with DDD pacing (4.8% absolute risk reduction).
MVP, randomized multicenter (2011)[42]	1030 (ICD)	No superiority of MVP over VVI-40 in terms of AF, VT/VF, quality of life, HF.
Steinbach et al, retrospective single-center (2011)[43]	102 (PM)	In patients over 75 years of age, MVP showed lower rates of HF episodes and all-cause mortality than conventional DDD pacing
long-MinVPACE, randomized single-center (2011)[44]	66 (PM)	Less RV pacing, less AF burden in MinVP group patients compared with DDDR (mean 12.8 vs 47.6%). Chosen AV/PV delay (150/130 ms) was probably too short in the DDDR (control) group.
Generation MVP, observational multicenter (2012)[45]	220 (PM)	Significantly fewer atrial arrhythmias when programmed to MVP compared with DDD.
PreFER MVP, randomized multicenter (2014)[46]	605 (556 PM, 49 ICD)	No difference between cardiovascular hospitalization, AF, and the composite of death and hospitalization between the MVP and DDD groups. The authors stated that "patients were enrolled upon elective replacement of the device, and were healthy enough to survive the first device without experiencing a significant decrease in LV function."
MINERVA, randomized multicenter (2014)[47]	1300 (PM)	AF burden: no superiority of MVP pacing compared with the DDDR mode (AV/PV delay $>$180/210 ms in greater than 60% of patients, 53% of RV pacing). MVP in combination with atrial antitachycardia pacing was superior to both DDDR and MVP-only.
COMPARE, randomized multicenter (2014)[48]	385 (PM)	Lower percentage of ventricular pacing (%VP) in the MVP group compared with the SearchAV+ group. A trend in the correlation between %VP and AT/AF burden.

AT = atrial tachycardia; HF = heart failure; MVP = Managed Ventricular Pacing; PM = pacemaker.

the length of the programmed atrioventricular interval.[50] Use of the AAIR pacing mode with exceedingly long atrioventricular conduction times can lead to "AAIR pacemaker syndrome" and actually increases the risk of AF compared with the DDDR mode, as was shown in the Danish Multicenter Randomized Trial on Single Lead Atrial versus Dual-Chamber Pacing in Sick Sinus Syndrome (DANPACE).[3,51] Therefore, excessively long atrioventricular delays resulting in nonphysiologic atrioventricular contraction patterns should be avoided. The potential harm of atrial pacing with a prolonged atrioventricular delay was also demonstrated in the MVP trial. In the MVP trial, dual-chamber pacing with the MVP algorithm was not superior to ventricular backup pacing (VVI 40 bpm) with respect to HF events. After a follow-up of 2.4 years, there was an apparent increase in HF events that was limited primarily to patients with a baseline PR interval of $>$230 ms (mean PR of 255–260 ms).[42] Long atrioventricular intervals also predispose the patient to repetitive atrioventricular reentrant rhythms, "repetitive nonreentrant VA synchrony," or "atrioventricular desynchronization arrhythmia," which manifest as mode switching but which also cause sustained episodes with poor hemodynamics.[52] Thus, based on the available data, it appears that atrial pacing with excessively long atrioventricular delays should be avoided.

Algorithms that minimize ventricular pacing sometimes lead to inadvertent bradycardia or spontaneous premature, beat-related short-long-short RR interval sequences with proarrhythmic potential.[53–55] However, in a study retrospectively analyzing the onset of ventricular tachycardia (VT) in ICD patients, the MVP mode was less frequently associated with the onset of VT compared with the DDD and VVI modes.[54] Atrioventricular decoupling (greater than 40% of atrioventricular intervals exceeding 300 ms) was observed in 14% of the ICD patients in the Marquis ICD MVP study, which might have a negative effect on ventricular filling.[56]

In ICD patients with structural heart disease, spontaneous atrioventricular conduction can become prolonged instead of shortening, with increased atrial paced heart rates.[33] This outcome frequently leads to a higher percentage of ventricular paced complexes. In view of the results of the ADEPT trial, which failed to demonstrate the clinical superiority of combined rate modulation and DDD pacing, the need for and aggressiveness of sensor-driven rate responses should be individualized or eliminated.[19] Rate-dependent shortening of atrioventricular delay could have the same effect and should usually be avoided.

Patients with hypertrophic cardiomyopathy represent a small but intricate subset of the ICD population for whom pacing has not been demonstrated to be a consistently effective treatment for outflow tract obstruction. However, according to the 2011 ACCF/AHA Hypertrophic Cardiomyopathy Guideline, dual-chamber ICDs are reasonable for patients with resting LV outflow tract gradients more than 50 mm Hg, and who have indications for ICD implantation to reduce mortality.[57] In these patients, atrioventricular delays should be individually programmed to be short enough to achieve RV preexcitation and decrease LV outflow tract gradient, but not too short, which would impair LV filling; usually in the ranges of 60–150 ms.[58,59] There are few studies of pacing modes in these patients, and they are

limited by small numbers and the failure to quantify important cardiac outcomes.

In conclusion, atrioventricular interval programming and choosing between DDDR and MVP or other atrioventricular interval management modes should be performed on an individual basis. The goal is to minimize the percentage of RV pacing and to avoid atrial-based pacing with atrioventricular intervals exceeding 250–300 ms leading to atrioventricular uncoupling. In patients with prolonged PR intervals and impaired LV function, biventricular pacing can be considered.

Cardiac Resynchronization Therapy: Consistent Delivery of Ventricular Pacing

CRT in combination with a defibrillator device (CRT-D) improves survival and cardiac function in patients with LV systolic dysfunction, prolonged QRS duration, and mild-to-severe HF.[60–62] The beneficial effect of CRT-D compared with ICD is likely to be derived from biventricular pacing, with a decrease in dyssynchrony and an improvement in cardiac function. The percentage of biventricular pacing capture in the ventricles can be negatively influenced by a number of factors, including atrial tachyarrhythmias, premature ventricular complexes, and programming of the atrioventricular delay, giving way to the intrinsic conduction of the patient and a reduced percentage of biventricular pacing. Some large observational studies have investigated the optimal level of biventricular pacing percentage and found a higher percentage to be associated with more pronounced CRT benefits. An optimal CRT benefit was observed with a biventricular pacing percentage as close to 100% as possible.[63–66]

In the analysis of the left bundle branch block population in the MADIT-CRT trial, those patients with less than 90% biventricular pacing had similar rates of HF and death compared with the patients randomized to no CRT. By contrast, biventricular pacing exceeding 90% was associated with a benefit of CRT-D in terms of HF or death when compared with ICD patients and no CRT. Biventricular pacing 97% and greater was associated with a further reduction in HF or death and a significant reduction in death alone. Consistently, every 1% increase in biventricular pacing percentage was associated with a 6% risk reduction in HF or death, a 10% risk reduction in death alone, and an increase in LV reverse remodeling.[67] Therefore, in ICD patients with biventricular pacing, it can be beneficial to adjust the therapy to produce the highest achievable percentage of ventricular pacing, preferably above 98%, to improve survival and reduce HF hospitalization. Approaches to increasing the percentage of biventricular pacing include programming shorter but hemodynamically appropriate atrioventricular delays and minimizing atrial and ventricular ectopic activity and tachyarrhythmias.

Optimizing the location of ventricular pacing sites and the timing of the pacing pulses can significantly improve cardiac hemodynamics in CRT patients. Echocardiographic optimization of atrioventricular delays in CRT patients can alleviate HF symptoms and increase exercise capacity compared with nominal programming, particularly when approaching nonresponding populations.[68] However, echocardiographic optimization in the PROSPECT study did not support this approach in a randomized trial, and the Frequent Optimization Study Using the QuickOpt Method (FREEDOM) trials failed to provide evidence supporting the benefit of CRT optimization and did not demonstrate superiority of the respective algorithms over nominal or empiric programming.[69–71] There are limited data supporting the use of LV-only stimulation in a small subset of patients who fail to respond to biventricular stimulation.[72] Adaptive CRT (aCRT) is an algorithm that periodically measures intrinsic conduction and dynamically adjusts CRT pacing parameters. The algorithm withholds RV pacing when intrinsic electrical conduction to the RV is normal and provides adjustment of CRT pacing parameters based on electrical conduction. A prospective, multicenter, randomized, double-blind clinical trial demonstrated the safety and efficacy of the aCRT algorithm.[73] This algorithm can increase the longevity of the implantable device and replace a manual device optimization process with an automatic ambulatory algorithm, although echo optimization might still be needed, at least in nonresponders. The Clinical Evaluation on Advanced Resynchronization (CLEAR) study assessed the effects of CRT with automatically optimized atrioventricular and interventricular delays, based on a peak endocardial acceleration (PEA) signal system. PEA-based optimization of CRT in patients with HF significantly increased the proportion of patients who improved with therapy during follow-up, mainly through an improved New York Heart Association (NYHA) class.[74]

Bradycardia Mode and Rate Programming Recommendations	Class of Recommendation	Level of Evidence
In ICD patients who also have sinus node disease and guideline-supported indications for a bradycardia pacemaker, it is beneficial to provide dual-chamber pacing to reduce the risk of AF and stroke, to avoid pacemaker syndrome, and to improve quality of life.	I	B-R
In single- or dual-chamber ICD patients without guideline-supported indications for bradycardia pacing, adjusting the pacing parameters is recommended so that ventricular stimulation is minimized to improve survival and reduce HF hospitalization.	I	B-R
In ICD patients who have sinus rhythm, no or only mild LV dysfunction, and atrioventricular block where ventricular pacing is expected, it is reasonable to provide dual-chamber pacing in preference to single-chamber ventricular pacing to avoid pacemaker syndrome and to improve quality of life.	IIa	B-R
In ICD patients who have sinus rhythm, mild-to-moderate LV dysfunction, and atrioventricular block where ventricular pacing is expected, it is reasonable to provide CRT in preference to dual-chamber ventricular pacing to improve the combination of HF hospitalization, LV enlargement, and death.	IIa	B-R
In ICD patients who have chronotropic incompetence, it can be beneficial to program the ICD to provide sensor-augmented rate response, especially if the patient is young and physically active.	IIa	B-NR
In dual-chamber ICD patients with native PR intervals of 230 ms or less, it can be beneficial to program the mode, automatic mode change, and rate response so that the patient's native atrioventricular conduction minimizes ventricular pacing.	IIa	B-R
In biventricular pacing ICD patients, it can be beneficial to adjust the therapy to produce the highest achievable percentage of ventricular pacing, preferably above 98%, to improve survival and reduce HF hospitalization.	IIa	B-NR
In biventricular pacing ICD patients, it can be reasonable to activate the algorithms providing automatic adjustment of atrioventricular delay and/or LV-RV offset to obtain a high percentage of synchronized pacing and reduce the incidence of clinical events.	IIb	B-R

Tachycardia Detection Programming

Following significant technological changes in ICDs in recent years, the concept of optimal ICD programming has changed dramatically. From the dawn of this therapy in the early 1980s to the first decade of the 21st century, the rapid detection and treatment of VT and ventricular fibrillation (VF) have been stressed. The argument for rapid detection of VT and VF derived from a number of factors. Initial skepticism regarding the feasibility of sudden death prevention with ICDs, the fact that early ICD patients had all survived one or more cardiac arrests, concern for undersensing and underdetection (of VF in particular), demonstration of an increasing defibrillation threshold with prolonged VF duration, and the increased energy requirement of monophasic defibrillation all created a culture of programming for rapid tachycardia detection and the shortest possible time to initial therapy.[75–77] The initial generations of ICDs did not record and save electrograms (EGMs), leading to a reduced appreciation for the frequency and impact of inappropriate shocks. With the advent and then dominance of primary prevention indications, avoidable shocks assumed a relatively larger proportion of total therapy.[78–83] Gradually, publications have increased awareness of the frequency and the diverse range of adverse outcomes associated with avoidable ICD therapy, and have demonstrated that avoidable ICD shocks can be reduced by evidence-based programming of the detection rate, detection duration, antitachycardia pacing (ATP), algorithms that discriminate supraventricular tachycardia (SVT) from VT, and specific programming to minimize the sensing of noise.[81–92]

Duration Criteria for the Detection of Ventricular Arrhythmia

Until recently, default device programming used short-duration "detection" criteria that varied by manufacturer and a tachycardia rate of approximately 2.8 to 5 seconds before either ATP or charging (including detection time plus duration or number of intervals).[82,93] With increased awareness of the potential harm from inappropriate shocks and the realization from stored pacemaker EGMs that even long episodes of VT can self-terminate, a strategy of prolonged

detection settings has been explored. This strategy allows episodes to self-terminate without requiring device intervention and reduces inappropriate therapy for nonmalignant arrhythmias. The benefit of programming a prolonged detection duration (30 of 40 beats) was first reported in the Prevention Parameters Evaluation (PREPARE) study on exclusively primary prevention subjects (n = 700), and compared outcomes to a historical ICD cohort programmed at "conventional detection delays" with about half programmed to 12 of 16 intervals within the programmed detection zone and half to 18 of 24 intervals.[94] The programming in PREPARE demonstrated a significant reduction in inappropriate shocks for supraventricular arrhythmia and in avoidable shocks for VT. In addition, a composite endpoint was reduced as well: the morbidity index, which consists of shocks, syncope, and untreated sustained VT. Within the limitations of a nonrandomized study, it was concluded that extending detection times reduces shocks without increasing serious adverse sequelae.

In 2009, the Role of Long-Detection Window Programming in Patients with Left Ventricular Dysfunction, Non-Ischemic Etiology in Primary Prevention Treated with a Biventricular ICD (RELEVANT) study confirmed and expanded the results of the PREPARE trial in a cohort of 324 primary prevention CRT-D patients with nonischemic cardiomyopathy.[95] The subjects were treated with simplified VT management, which implies much longer detection for VF episodes (30 of 40) compared with the control group (12 of 16) and a monitor-only window for VT. As in PREPARE, the RELEVANT study group experienced a significantly reduced burden of ICD interventions (81% reduction) without increasing the incidence of syncope. Fewer inappropriate shocks and HF hospitalizations were reported in the RELEVANT study group compared with the control group.

The Multicenter Automatic Defibrillator Implantation Trial: Reduce Inappropriate Therapy (MADIT-RIT), a 3-arm study, compared a conventional programming strategy (a 1-second delay for VF [equivalent to approximately 12 intervals including detection plus delay] and a 2.5-second delay for VT detection [equivalent to approximately 16 intervals including detection plus delay]) (Arm A) to both a high-rate cutoff with a VF zone starting at 200 bpm (Arm B) (discussed in section Rate Criteria for the Detection of Ventricular Arrhythmia and discussed as referenced in reference[96].) and to a delayed therapy strategy with a 60-second delay for rates between 170 and 199 bpm, a 12-second delay at 200 to 249 bpm, and a 2.5-second delay at 250 bpm (Arm C).[96] The MADIT-RIT population was exclusively primary prevention and included approximately an equal proportion of nonischemic and ischemic cardiomyopathy patients. All the patients were implanted with either a dual-chamber ICD or a CRT-D programmed to deliver ATP before charging. After a mean 1.4-year follow-up, the prolonged detection group (Arm C) was associated with a reduction in treated VT/VF leading to a 76% reduction in the primary endpoint of the first inappropriate therapy ($P < .001$), as well as a significant reduction in the first appropriate therapy, appropriate ATP, and inappropriate ATP, but not in appropriate or inappropriate shock.

The Avoid Delivering Therapies for Non-Sustained Arrhythmias in ICD Patients III (ADVANCE III) trial reported that a long detection was associated with a highly significant reduction of overall therapies (appropriate and inappropriate ATP and/or shocks), inappropriate shocks, and all-cause hospitalizations.[97] Importantly, like PREPARE, RELEVANT, and MADIT-RIT, the extended detection duration used in the ADVANCE III trial (30 of 40) did not negatively impact the rate of syncopal events. There was no significant difference in mortality between the optimal and the conventional programming groups. Compared with the MADIT-RIT trial, the ADVANCE III control group had a longer detection duration (primarily in the VF zone), and enrolled a larger cohort of subjects covering all ICD types (single, dual, and CRT with ATP delivered during charging) for both primary and secondary prevention indications. Finally, the Programming Implantable Cardioverter-Defibrillators in Patients With Primary Prevention Indication (PROVIDE) trial randomized 1670 patients to conventional programming (12-beat detection in each of 2 zones) or experimental programming (2 VT and 1 VF zone requiring 25-, 18-, and 12-beat detection, respectively).[98] PROVIDE observed a significant 36% reduction in the 2-year all-cause shock rate and an improved survival (hazard ratio [HR] 0.7; 95% confidence interval [CI] 0.50–0.98; $P = .036$).

Whereas PREPARE, RELEVANT, MADIT-RIT, and PROVIDE only enrolled primary prevention patients, a subset of the ADVANCE III study evaluated the efficacy and safety of a long-detection approach in secondary prevention patients who have a known higher burden of arrhythmic episodes. In this particular subset of 25% of the enrolled patients, ADVANCE III reported that a long detection duration reduced the overall therapies delivered, primarily due to a significant 36% reduction in appropriate shocks.[99] Syncopal episodes related to arrhythmic events and deaths were similar between the 2 groups.

Following shortly on the heels of these trials, 2 meta-analyses including the above studies were published in 2014. Tan et al presented the data from the RELEVANT, PREPARE, MADIT-RIT, ADVANCE III, PROVIDE, and EMPIRIC trials.[100,101] A 30% reduction in the risk of death was found in the therapy reduction group when including all 6 studies; however, similar results were observed when separately considering the 4 randomized trials and the 2 observational studies. Data on the appropriateness of shocks were available only for RELEVANT, MADIT-RIT, ADVANCE III, and PROVIDE, and a 50% reduction in inappropriate shock was observed without an increased risk of syncope and appropriate shock.

A meta-analysis evaluated the impact of a prolonged arrhythmia detection duration on outcome[102]—thus excluding the EMPIRIC trial (which used 18 of 24 intervals for VF detection), the PREPARE trial (which used a historical control group), and the high-rate therapy arm of the MADIT-RIT. Analyzing the cohort of patients enrolled in

RELEVANT, Arm C of MADIT-RIT, ADVANCE III, and PROVIDE, the meta-analysis reported a reduction of overall burden of therapies, driven by the greater than 50% reduction in appropriate and inappropriate ATP and the 50% reduction in inappropriate shocks. A reduction in all-cause mortality was observed without an increase in the risk of syncope.

All the reports above clearly stress the necessity to consider a long detection window setting as a "default" strategy for ICD programming. Moreover, they underline the importance of choosing to reprogram the ICD rather than using the manufacturers' out-of-the-box settings. A summary of the large comparative datasets of tachycardia detection is presented in Table 2.

Limitations of Data on the Duration of Tachycardia Required for Detection

Although the findings on the effect of tachycardia detection duration are based on roughly 7000 patients, there are limitations. Data on secondary prevention patients are limited to 25% of the 1902 patients enrolled in the ADVANCE III trial (n = 477). Although this proportion is a fair representation of the real-world population receiving an ICD, more data are needed to fully understand the impact of a long-detection strategy in this subgroup of patients. MADIT-RIT and RELEVANT did not include single-chamber ICDs, and MADIT-RIT excluded patients with permanent AF. The PROVIDE and MADIT-RIT trials were designed to assess the time to first therapy and not the overall rate of therapies. MADIT-RIT, ADVANCE III, RELEVANT, and PROVIDE used devices from 3 different manufacturers with detection strategies leading to different detection times, intervals, and definitions. Some manufacturers of ICDs are not represented at all in these trials. Programming in the trial control groups was highly heterogeneous, with time until ATP or charging for VF as varied as about 11–12 intervals (approximately 3.4 seconds at 200 bpm) in MADIT-RIT and PROVIDE and 18 intervals (approximately 5.4 seconds) in ADVANCE III. An approximate translation of the impact of the number of intervals to detection and tachycardia cycle length (CL) are listed in Table 3. A further limitation is the relatively short duration and lack of inclusion of the patients with the most severe illness receiving an ICD. This limitation minimizes the exposure to relatively rare events that might occur in nonclinical trial, "real-world" patients. Lastly, as ICD batteries deplete, the charge time lengthens. The effect of such a delay to shock therapy in addition to prolonged detection times has not been studied.

Rate Criteria for the Detection of Ventricular Arrhythmia

Ventricular tachyarrhythmia detection by implantable devices is primarily based on heart rate. Heart rates can be extremely rapid during ventricular tachyarrhythmias, and it is less likely that such rates are achieved during supraventricular tachyarrhythmias—thus making rate a powerful component of arrhythmia discrimination. However, VT can also present slower rates in the range of those of supraventricular tachyarrhythmias or even of sinus tachycardia. Therefore, any rate cutoff will always imply a tradeoff between maximizing sensitivity for ventricular tachyarrhythmia detection at the expense of inappropriate detection of fast supraventricular tachyarrhythmias and maximizing specificity at the expense of some slow VTs going undetected.[103]

Because ICD therapy was initially employed in secondary prevention patients, the cutoff rate was usually tailored to a rate slightly below that of the observed VT. With the development of ICD use in primary prevention, the detection

Table 2 Tachycardia detection evidence

Study	Participants (N)	Short detection controls	Prolonged detection intervention	Findings
PREPARE	1391 Nonrandomized Primary prevention	12 of 16 (58%) 18 of 24 (42%)	30 of 40	Reduction in inappropriate shocks (SVT), avoidable shocks (VT), and "morbidity index"
RELEVANT	324 Nonrandomized Primary prevention	12 of 16	30 of 40	Reduction in inappropriate shocks (SVT), avoidable shocks (VT), and HF hospitalizations
MADIT-RIT	1500 Randomized Primary prevention	2.5 s (170–199 bpm) 1 s (≥200 bpm)	60 s (170–199 bpm) 12 s (200–249 bpm) 2.5 s (≥250 bpm)	Reduction in first inappropriate therapy, first appropriate therapy, appropriate ATP, and inappropriate ATP; improved survival
ADVANCE-III	1902 Randomized Primary & secondary prevention	18 of 24	30 of 40	Reduction in overall therapies, inappropriate shocks, and all-cause hospitalizations
PROVIDE	1670 Randomized Primary prevention	12 beats	25 beats (180–214 bpm) 18 beats (214–250 bpm) 12 beats (>250 bpm)	Reduction in all-cause shock rate; improved survival

Table 3 Approximating the time taken to detect 30 intervals using fixed 8 of 10 interval detection plus adding a time delay, for a range of heart rates

Arrhythmia characteristic		Interval-based detection	8 of 10 interval detection, then delay	
Beats per minute	Cycle length (ms)	Time to detect 30 intervals (s)	Time to detect 8 intervals (s)	Subsequent delay to approximate a 30-interval detection time
180	333	10.0	2.7	7.0
200	300	9.0	2.4	6.5
220	273	8.2	2.2	6.0
240	250	7.5	2.0	5.5
260	231	6.9	1.8	5.0
280	214	6.4	1.7	4.5
300	200	6.0	1.6	4.5

rate came into question because there is no history of sustained tachycardia in these patients. The recognition of a significant rate of inappropriate therapies in primary prevention studies, and their potentially deleterious consequences, prompted the development of studies that tested whether programming faster rate criteria reduced avoidable ICD therapies, particularly shocks. In many of these studies, however, testing also involved programming parameters other than rate, and those have been discussed as described below.

In the MADIT-RIT trial of primary prevention patients, conventional therapy (rate cutoff 170 bpm, n = 514) was compared with a "high-rate group" in which rate cutoff was 200 bpm (n = 500).[96] The primary endpoint of first occurrence of inappropriate therapy was observed in 20% of the conventional group and in 4% of the high-rate group ($P < .001$) over a mean follow-up of 1.4 years. ICD shocks occurred in 4% and 2% of patients in the conventional and high-rate groups, respectively. The proportion of patients with appropriate therapies was also significantly different (22% vs 9% in the conventional and high-rate groups, respectively). It is important to note that all-cause mortality in the conventional group (6.6%) was approximately double that of the high-rate group (3.2%, $P = .01$).

In a single-center observational study, 365 primary prevention patients were prospectively studied, with programming including a single shock-only zone over 220 bpm.[104] During a mean follow-up of 42 months, 11% of the patients (7% in the first 2 years) experienced appropriate shocks, and only 6.6% experienced inappropriate shocks. It was notable that in the monitoring zone over 170 bpm, self-limiting VT episodes were detected in 12% of the patients but were symptomatic in only 1.9%. The mortality rate was 17%, with one case of unexplained sudden death.

A recent primary prevention study revealed that there was considerable overlap between the ventricular rates of supraventricular and ventricular arrhythmias, and the majority of inappropriate shocks occurred at rates between 181 and 213 bpm.[98] These data also support the notion that for primary prevention patients it is safe to increase the rate cutoff up to 200 bpm to reduce these potentially avoidable therapies, a practice that was also supported by the results of the MADIT-RIT trial.

In secondary prevention patients, no trial has randomized the detection rate and compared outcomes. However, the ADVANCE III Secondary Prevention substudy confirmed the safety of not programming therapy for rates <188 bpm; syncope was rare at 2 to 3 episodes per 100 patient-years.[105] Previously published recommendations suggest a VT zone starting at 10 to 20 bpm slower than the observed tachycardia rate, usually including a 2- or 3-zone arrhythmia detection scheme (as discussed elsewhere).[106] Clinicians should allow a larger rate differential when starting a patient on an antiarrhythmic drug that might slow the clinical tachycardia rate (e.g., amiodarone).

Single- or Multi-Zone Detection

Modern ICDs allow the rate to be classified into single or multiple zones. This classification permits different criteria to be applied for detection (e.g., number of intervals) and for tiered therapy (e.g., different adaptive CLs for slower vs faster VTs and more sequences of ATP for slower and presumably hemodynamically more stable VTs). Additionally, because some manufacturers tie SVT discrimination algorithms to specific VT zones, programming more than one tachycardia zone allows for greater specificity in discriminating VT from SVT (see online Appendix B). Although there are trials in which arms differ in whether a single zone or multiple zones are used, this is typically performed to allow programming of various detection, discrimination, or therapies for comparison. Thus, the number of zones was not the randomization variable being directly compared. Therefore, the concept of single- vs multizone programming as a head-to-head comparison is not well tested. The MADIT-RIT study randomized primary prevention ICD patients into 1 of 3 arms with single-, dual-, or triple-zone programming (the single-zone arm also had a monitoring zone). Although the trial's aim was to compare conventional therapy with high-rate and delayed therapy, the outcome for the single-zone arm (high-rate) was comparable to the triple-zone (delayed) arm and superior to the dual-zone (conventional) arm, with regard to inappropriate shock.[96] This study is consistent with multiple studies in ICD programming in which the use of multiple-zone programming has allowed for flexibility in programming strategies

with regard to detection, discrimination, and therapy. Additionally, there are observational data from the ALTITUDE Real World Evaluation of Dual-Zone ICD and CRT-D Programming Compared to Single-Zone Programming (ALTITUDE REDUCES) study that show that dual-zone programming is associated with fewer shocks than single-zone programming, at least for rates <200 bpm.[64] Therefore, the authors conclude that using more than 1 detection zone can be useful for modern ICD programming. It should be noted that ATP before or during charging was used in the majority of studies described in both the tachycardia detection and therapy sections and thus is recommended for longer detection.

Discrimination Between Supraventricular and Ventricular Arrhythmia

The SVT-VT discrimination process classifies a sequence of sensed EGMs that satisfies rate and duration criteria as either SVT (therapy withheld) or VT/VF (therapy given). *Discriminators* are individual algorithm components that provide a partial rhythm classification or a definitive classification for a subset of rhythms. *Discrimination algorithms* combine individual component discriminators to produce a final rhythm classification. Discrimination algorithms vary among manufacturers and between individual ICD models (see online Appendix B). The final rhythm classification can differ depending on the technical details of how each individual discriminator is calculated, the nominal or programmed threshold for each discriminator, the order in which discriminator components are applied, and the logical connections between them (e.g., "and" vs "or"). In some ICDs, rhythms classified as VT/VF undergo a subsequent sensing-verification step to confirm that EGMs represent true cardiac activation.

SVT-VT Discriminator Components

Individual discriminators can be considered in relation to the EGMs analyzed as ventricular-only or both atrial and ventricular, by the rhythm that they identify (e.g., AF, sinus tachycardia, VT), or by the type of EGM information analyzed (intervals vs morphology). Note that ventricular rate alone is a mandatory discriminator, as discussed in the section above. We summarize the most commonly used discriminators. More comprehensive discussions are available in the literature.[107–111]

Rejection of Sinus Tachycardia by Onset

Several interval-based discriminators focus on differences in the onset of sinus tachycardia (gradual and parallel acceleration of atrial and conducted ventricular intervals) compared with VT (typically abrupt, with at least transient atrioventricular dissociation). *Sudden (abrupt) onset* was one of the first single-chamber, interval-based discriminators. It withholds therapy if acceleration across the sinus-VT rate boundary is gradual. Because onset discriminators classify the rhythm only once, and thus cannot correct misclassifications, they are now used infrequently and only with an override feature and/or other discriminators.[112–115] *Chamber of onset* is a related, interval-based, dual-chamber discriminator that classifies a 1:1 tachycardia as SVT if the atrial rhythm accelerates at the device-defined onset. A related "Sinus Tachycardia®" discriminator classifies a tachycardia as VT if either the RR or the PR intervals deviate sufficiently from the range of the immediately preceding sinus intervals.[116]

Rejection of AF by Ventricular Interval Regularity

Ventricular interval regularity (*interval stability*) is an explicit single-chamber, interval-based discriminator that classifies the rhythm as AF if the ventricular intervals are sufficiently irregular. Because interval variability in conducted AF decreases at faster rates, stability becomes unreliable in discriminating VT from conducted AF at ventricular rates greater than 170 bpm.[112,115] Interval stability can also fail if drugs (e.g., amiodarone) cause monomorphic VT to become irregular or induce polymorphic VT to slow into the SVT-VT discrimination zone.[114,117]

Diagnosis of VT by Dual-Chamber Components: Atrial vs Ventricular Rate and Atrioventricular Association

In contrast to the single-chamber discrimination algorithms above that diagnose SVT when their criteria are fulfilled, 2 separate, interval-based, dual-chamber discrimination algorithms diagnose VT. First, ***atrial rate vs ventricular rate*** diagnoses VT if the ventricular rate exceeds the atrial rate.[118] Second, ***atrioventricular dissociation*** identifies isorhythmic VT during sinus tachycardia. Inversely, the atrioventricular ***association*** discriminator diagnoses SVT in the presence of N:1 (e.g., 2:1, 4:1) atrioventricular association consistent with atrial flutter at a fixed conduction ratio.

The Ventricular Electrogram Morphology Discriminator

This versatile, single-chamber discriminator is the only algorithm component that does not rely on inter-EGM intervals. It classifies tachycardias as SVT if the morphology (shape) of the ventricular EGM is sufficiently similar to the morphology during a conducted baseline rhythm. It can potentially discriminate any SVT from VT, including SVTs that challenge other discriminators, such as abrupt-onset 1:1 SVTs and irregular VT during AF. Contemporary ICDs (including subcutaneous ICD [S-ICD]) analyze EGMs from the shock electrodes, which record a larger field of view than EGMs from pace-sense electrodes.[119] They operate using a common series of steps and are susceptible to common failure modes.[111,120–123] The first common step is acquisition of a baseline rhythm template by mathematically extracting EGM features and storing them. Both the acquisition of the initial template and the subsequent template updating are automated in most ICDs. Nevertheless, physicians should confirm that the conducted baseline beats match the template both at implant and during follow-up. For CRT patients, the template must be manually collected. If the wavelet signal

during template acquisition appears clipped, adjustments specific to the manufacturer might be necessary.

SVT-VT Discrimination Algorithms

Discrimination algorithms combine component discriminators to provide a final rhythm classification of VT/VF or SVT. The morphology discriminator frequently forms the primary component of single-chamber algorithms with stability playing a secondary role and sudden onset used sparingly. By contrast, the cornerstone of most dual-chamber algorithms is explicit or implicit comparison of atrial vs ventricular rates. Because the ventricular rate is greater than the atrial rate in more than 80% of VTs, algorithms that compare atrial and ventricular rates as their first step apply additional SVT discriminators to fewer than 20% of VTs, reducing the risk that they will misclassify VT as SVT.[124,125] Most dual-chamber algorithms further restrict single-chamber discriminators to tachycardias for which they offer the greatest benefit; thus, stability is applied only if AF is confirmed by direct calculation of the atrial rate or the atrial rate is greater than the ventricular rate. Similarly, sudden onset, chamber of onset, or 1:1 atrioventricular association are applied only if the atrial rate equals the ventricular rate. The use of discriminators in redetection varies among manufacturers and has not been systematically studied.

Assessing Clinical Benefits and Risks

What Evidence Supports a Benefit?

1. The annual rate of inappropriate shocks has fallen dramatically from 37%–50% for SVT alone in early studies to 1%–5% for all causes in modern clinical trials.[97,118,126–128] This decrease is likely due to differences in both clinical populations and the programming of multiple ICD parameters, including longer detection time and higher rate cutoffs. Thus, it is difficult to isolate the differential effect of SVT-VT discrimination algorithms using clinical data. These studies have programmed discrimination algorithms to ON, however, so it seems reasonable to use them.
2. Although clinical trials that reported dramatic reductions in shocks for SVT programmed discrimination algorithms consistently, they have been programmed inconsistently in clinical practice, and the rate of inappropriate shocks for SVT has been higher in observational studies of remote-monitoring ICD databases. In the ALTITUDE REDUCES study on 15,991 patients in the Latitude® database, SVT was the most common cause of shocks when the detection rate was $\leq$180 bpm.[129] For detection rates $\leq$170 bpm, the rate of inappropriate shocks at 1 year was significantly lower with dual-zone programming, which permits SVT-VT discrimination, than single-zone programming, which does not (9.6% vs 4.3%). Similarly, Fischer et al[130] analyzed shocks in 106,513 patients in the CareLink® database; programming SVT-VT discrimination ON was associated with a 17% reduction in all-cause shocks.
3. Sophisticated simulations indicate that SVT-VT discrimination algorithms have substantial benefit. For example, the SCD-HeFT study on primary prevention patients did not use discriminators. A validated Monte Carlo simulation predicted that use of single- or dual-chamber SVT-VT discriminators alone would have reduced inappropriate shocks for SVT by 75.5% and 78.8%, respectively.[131]

Which Patients are Most Likely to Benefit, and Which are Least Likely to Benefit?

Despite limited direct evidence, it seems clear that patients will benefit most if the rates of their VTs and SVTs overlap. This includes patients with slower monomorphic VT, those at risk for AF with rapid ventricular rates, or those capable of exercising to sinus rates in the VT zone.[103,132] In secondary prevention patients with slower VT, older discrimination algorithms reduced shocks for SVT compared with rate-only detection. The benefit is less for primary prevention patients, secondary prevention patients at risk only for VF, and those who cannot sustain rapid atrioventricular conduction. Patients with permanent complete atrioventricular block do not benefit.

What are the Risks?

The risk of the misclassification of either VT or VF as SVT by the discrimination algorithms can either prevent VT detection or delay the time to therapy (underdetection), as documented in clinically significant situations.[112,113,115,125] When modern algorithms are programmed to recommended parameters, clinically significant underdetection is rare. Large clinical trials on multiple shock-reduction strategies (including SVT-VT discrimination) report no or minimal and statistically insignificant increases in syncope.[95,97,126,133] Most reports do not include the causes of syncope and thus do not permit identification of whether discrimination algorithms contributed to any of the syncopal episodes by prolonging detection. However, in the PREPARE study, no syncopal episode was caused by untreated tachycardia.[133] In general, discriminators that re-evaluate the rhythm classification during ongoing tachycardia reduce the risk of underdetection compared with those that withhold therapy if the rhythm is misclassified by the initial evaluation (e.g., onset, chamber of origin algorithms).

Additional Considerations

SVT Limit

SVT-VT discrimination applies from the VT detection rate to the SVT limit rate, which is programmable independently of the VT/VF therapy zones with some manufacturers (preferable), but which might be linked to one of the zone boundaries in others. The minimum CL for SVT-VT discrimination should be set to prevent clinically significant delays in the detection of hemodynamically unstable VT. PREPARE, EMPIRIC, and MADIT-RIT all support the safety of empirical programming at 200 bpm.[96,101,134] In

MADIT-II, approximately 50% of SVT episodes were faster than 170 bpm, and a few were as fast as 250 bpm.[82] In INTRINSIC RV, SVT comprised 19% of episodes, with rates between 200 and 250 bpm.[135] More limited and preliminary data from PainFree SST support programming up to 222–230 bpm.[116,136] We suggest the SVT limit not exceed 230 bpm in adults without a patient-specific indication, based on the low incidence of SVTs in this rate range among ICD patients and the potential—however small—for misclassifying hemodynamically unstable VT.

Duration-Based "Safety-Net" Features to Override Discriminators

These features deliver VT/VF therapy if a tachycardia satisfies the ventricular rate criterion for a sufficient duration, even if the discrimination algorithm indicates SVT. The premise is that the ventricular rate during transient sinus tachycardia or AF will decrease to below the VT rate boundary before the override duration is exceeded. In one study, an override duration of 3 minutes delivered inappropriate therapy to 10% of SVTs.[112] Because SVT is much more common than VT, programming an override duration of less than 5–10 minutes results primarily or solely in inappropriate SVT therapy.[122] Although more data would be useful, in the absence of a documented benefit, we recommend programming this feature OFF or long (minutes) without a patient-specific or device-specific indication.

Dual-Chamber vs Single-Chamber Algorithms

Clinical trials and simulated testing of induced arrhythmias that compared single- vs dual-chamber discriminators have reported inconsistent results.[10,33,137–139] Two meta-analyses found no superiority of dual-chamber ICDs in terms of mortality or inappropriate therapies.[11,140] Any benefit of dual-chamber discrimination is likely restricted to specific patient groups.[103,138] For example, the Dual Chamber and Atrial Tachyarrhythmias Adverse Events (DATAS) trial of predominantly secondary prevention patients with slower VTs reported modest benefit from dual-chamber discrimination, while the recent Reduction and Prevention of Tachyarrhythmias and Shocks Using Reduced Ventricular Pacing with Atrial Algorithms (RAPTURE) trial of primary prevention patients programmed to a fast detection rate ($>$182 bpm) and long detection duration (30/40 intervals) did not.[103,138,139] Inappropriate therapy for SVT occurred in only 2% of the patients in each group. Recent data from PainFree SST notes very low rates of inappropriate shocks (3.7% for single chamber; 2.8% for dual and triple chamber after 2 years). The choice of device was not randomized, suggesting that when physicians chose a dual- or triple-chamber device (perhaps due to known atrial arrhythmia or bradycardia), inappropriate shock rates were minimized.[136] The Optimal Anti-Tachycardia Therapy in Implantable Cardioverter-Defibrillator Patients Without Pacing Indications (OPTION) trial randomized 462 patients to single- or dual-chamber programming and noted inappropriate shock rates of 10.3% for single chamber vs 4.3% for dual chamber after 27 months ($P = .015$). Atrial lead-related complications were 1.3%, therapy was delivered from 170 bpm (VT) and 200 bpm (VF), and no difference in ventricular pacing percentage was noted.[141] Dual-chamber algorithms probably reduce the risk of underdetection compared with single-chamber algorithms because more than 80% of VTs with a ventricular rate greater than the atrial rate undergo no further analysis.[103,124,125] However, the rate of clinically significant underdetection with modern programming is so low that this difference is rarely of clinical significance. In most patients, improved SVT-VT discrimination should not be considered an indication for a dual- vs single-chamber ICD. Even if a dual-chamber ICD is implanted, dual-chamber discrimination should be programmed only if the atrial lead becomes chronic or if atrial sensing is unreliable. Accurate sensing of atrial EGMs is essential for dual-chamber SVT-VT discrimination. Atrial lead dislodgments, oversensing of far-field R waves, or undersensing due to low-amplitude atrial signals can cause misclassification of VT/SVT. On implant, it is important to position the atrial lead to minimize far-field R waves.

Ventricular Oversensing

Excluding recalled leads, ventricular oversensing accounts for less than 10% of inappropriate shocks, but it often results in repetitive shocks and severe symptoms.[82,142–144] Recently introduced features reduce inappropriate therapies from oversensing of physiologic T waves and nonphysiologic signals related to pace-sense lead failures as discussed below.

Programming to Reduce T-Wave Oversensing

The problem of T-wave oversensing relates to the basic requirement that ICDs reliably sense VF, which is characterized by RR intervals shorter than the normal QT interval and some EGMs with low amplitudes and slew rates. Approaches to minimizing T-wave oversensing include reprogramming ventricular sensitivity, altering sensing bandwidth, and changing the sensing bipole.[109,123,145] One manufacturer provides an algorithm that withholds therapy after rate and duration criteria for VT/VF are fulfilled if a specific pattern of T-wave oversensing is identified.[146] T-wave oversensing rates vary based on device design; using an appropriate high band-pass filter results in very low rates of T-wave oversensing.[142] Because T-wave oversensing is unpredictable, features that minimize T-wave oversensing should be enabled proactively at implant, providing they do not cause undersensing in VF.[146]

Lead-Related Oversensing

Oversensed signals caused by pace-sense lead failure have specific interval patterns and EGM characteristics.[145,147,148] Present algorithms identify three features: (1) intervals can be too short to represent successive ventricular activations; (2) such short intervals are often transient and can be repetitive; and (3) in true bipolar leads, oversensed signals

are absent on the shock EGM. Algorithms can provide warning alerts, withhold shocks after spurious detection of VT/VF, or both. All 3 criteria can provide alerts, but only the third is applied to withhold shocks. The present algorithms were developed to identify impending lead failures on recalled leads, notably the Sprint Fidelis. These algorithms might not be appropriate for detecting failures in other leads.[144] There is a high false-positive rate when using these algorithms, and caregivers must carefully review the device data that caused the alert to ensure the lead experienced a true failure.[145]

Alerts that combine both oversensing and abrupt changes in impedance trends provide earlier warning of lead failure than a fixed impedance threshold.[144,145,149] Such alerts can be delivered via wireless remote monitoring and/or by notifying the patient via vibration or an audible tone. Caregivers must respond rapidly to alerts to minimize inappropriate shocks.[144,149] Wireless remote monitoring has been reported to reduce response time.[150] The principal disadvantage of lead alerts is false-positive triggers. The principal risk of shock-withholding algorithms is a failure to shock VF, which is extremely rare.[151] In addition to algorithmic approaches, oversensing due to failure of the cable leading to the ring electrode can be prevented by changing the programming of the sensing configuration from true bipolar to integrated bipolar. This approach is appropriate prophylactically or as temporary programming after a ring electrode cable failure; it is not a permanent solution, however, because increased rates of high-voltage cable fractures have been documented after sensing cable fractures.[152]

The Subcutaneous Defibrillator (S-ICD)

The novel S-ICD follows many of the same principles as intravascular ICDs but is considered here separately for duration criteria, rate criteria, and discrimination algorithms. Candidates for the S-ICD must initially be screened with a modified tri-channel surface electrocardiogram that mimics the sensing vectors of the S-ICD system. This test is designed to assess the R-wave to T-wave ratio for appropriate signal characteristics and relationships. If the screening is not satisfactory for at least 1 of the 3 vectors supine and standing, an S-ICD should not be implanted. On implant, the S-ICD automatically analyzes and selects the optimal sensing vector.

Detection of VT or VF by the S-ICD is programmable using a single or dual zone. In the single-zone configuration, shocks are delivered for detected heart rates above the programmed rate threshold: the "shock zone."[134] In the dual-zone configuration, arrhythmia discrimination algorithms are active from the lower rate: the "conditional shock zone." In this latter zone, a unique discrimination algorithm is used to classify rhythms as either shockable or nonshockable. If they are classified as supraventricular arrhythmias or nonarrhythmic oversensing, therapy is withheld.

With dual-zone programming, the shock zone uses rate as the sole method for rhythm analysis. In contrast, the conditional shock zone uses a stepwise discrimination algorithm to distinguish shockable from nonshockable rhythms. The conditional shock zone has a morphology analysis process based on a normal rhythm transthoracic QRS:T wave template. The template uses up to 41 fiduciary points to reconstruct morphology for the template as well as the programmed targeted heart rate zones. The comparison of the template to the high-rate rhythm electrocardiogram for discrimination constitutes the static waveform analysis. A good template match designates a sensed beat as supraventricular, thereby preventing a shock. A poor match to the static QRS:T morphology template moves the algorithm to a dynamic waveform analysis that compares single-beat morphologies in groups of 4 beats for uniformity. A consistent dynamic waveform match adjusts the sensing to evaluate QRS width. If a tachycardia has a prolonged QRS width compared with the template width (>20 ms) and is of sufficient duration, it will lead to a shock.

The system uses an initial 18 of 24 duration criteria (nonprogrammable) prior to initiating capacitor charging; however, this duration is automatically extended following nonsustained ventricular tachyarrhythmia events. A confirmation algorithm is also used at the end of capacitor charging to ensure persistence of the ventricular arrhythmia prior to shock delivery. Shocks for spontaneous (noninduced) episodes are delivered at a nonprogrammable 80 J regardless of the therapy zone of origination.

When programmed to include a conditional shock zone, the S-ICD VT detection algorithm has been demonstrated to be more effective than transvenous ICD systems programmed at nominal settings to prevent the detection of induced supraventricular arrhythmias.[153] Furthermore, in the clinical evaluation of the conditional shock zone, the S-ICD system was strongly associated with a reduction in inappropriate shocks from supraventricular arrhythmias and did not result in prolongation of detection times or increased syncope.[154]

Integrating Tachycardia Detection Data Into Programming Recommendations

When taking data from specific single-manufacturer studies and producing generic guidelines applicable across all ICDs, some compromises and potential pitfalls have been encountered. Nevertheless, it is our intention to convey the general principles of good quality evidence (e.g., extending detection time) to apply to ICD programming in general. Thus, attempts have been made to translate interval-based detection to time-based detection and to provide a range of reasonable heart rate cutoffs that are inclusive of those proven in good-quality trials. We encourage programming ICDs to manufacturer-specific therapies of proven benefit; however, when evidence is lacking, the guidelines provide a framework for programming within the evidence base. See online Appendix B for manufacturer-specific examples of optimal ICD programming.

Tachycardia Detection Programming Recommendations	Class of Recommendation	Level of Evidence
For primary prevention ICD patients, tachyarrhythmia detection duration criteria should be programmed to require the tachycardia to continue for at least 6–12 seconds* or for 30 intervals before completing detection, to reduce total therapies. **Tachyarrhythmia detection duration is directly related to the tachyarrhythmia rate. Direct evidence to support a delay $>$2.5 seconds for rates over 250 bpm is not available, but can be inferred from evidence that 30 detection intervals are safe at that rate.*	I	A
For primary prevention ICD patients, the slowest tachycardia therapy zone limit should be programmed between 185 and 200bpm*, to reduce total therapies. **Higher minimum rates for detection might be appropriate for young patients or for those in whom SVT-VT discriminators cannot reliably distinguish SVT from VT, provided there is no clinical VT below this rate.*	I	A
For secondary prevention ICD patients, tachyarrhythmia detection duration criteria should be programmed to require the tachycardia to continue for at least 6–12 seconds* or for 30 intervals before completing detection, to reduce total therapies. **Tachyarrhythmia detection duration is directly related to the tachyarrhythmia rate. Direct evidence to support a delay $>$2.5 seconds for rates over 250 bpm is not available, but can be inferred from evidence that 30 detection intervals are safe at that rate.*	I	B-R
Discrimination algorithms to distinguish SVT from VT should be programmed to include rhythms with rates faster than 200 bpm and potentially up to 230 bpm (unless contraindicated*) to reduce inappropriate therapies. **Discrimination algorithms and/or their individual components are contraindicated in patients with complete heart block or if the algorithm/component is known to be unreliable in an individual patient. Dual-chamber discriminators that misclassify VT as SVT if the atrial lead dislodges are discouraged in the perioperative period. Dual-chamber discriminators are contraindicated in patients with known atrial lead dislodgment, atrial undersensing or oversensing of far field R waves, and in those with permanent AF.*	I	B-R
It is recommended to activate lead-failure alerts to detect potential lead problems.	I	B-NR
For secondary prevention ICD patients for whom the clinical VT rate is known, it is reasonable to program the slowest tachycardia therapy zone at least 10 bpm below the documented tachycardia rate but not faster than 200bpm*, to reduce total therapies. **Higher minimum rates for detection might be appropriate for young patients or for those in whom SVT-VT discriminators cannot reliably distinguish SVT from VT, provided there is no clinical VT below this rate.*	IIa	C-EO
It can be useful to program more than one tachycardia detection zone to allow effective use of tiered therapy and/or SVT-VT discriminators and allow for a shorter delay in time-based detection programming for faster arrhythmias.	IIa	B-R
When a morphology discriminator is activated, it is reasonable to reacquire the morphology template when the morphology match is unsatisfactory, to improve the accuracy of the morphology discriminator.	IIa	C-LD
It is reasonable to choose single-chamber ICD therapy in preference to dual-chamber ICD therapy if the sole reason for the atrial lead is SVT discrimination, unless a known SVT exists that may enter the VT treatment zone, to reduce both lead-related complications and the cost of ICD therapy.	IIa	B-NR
For the S-ICD, it is reasonable to program 2 tachycardia detection zones: 1 zone with tachycardia discrimination algorithms from a rate $\leq$200 bpm and a second zone without tachycardia discrimination algorithms from a rate $\geq$230 bpm, to reduce avoidable shocks.	IIa	B-NR
Programming a nontherapy zone for tachycardia monitoring might be considered to alert clinicians to untreated arrhythmias.	IIb	B-NR
It may be reasonable to disable the SVT discriminator timeout function, to reduce inappropriate therapies.	IIb	C-EO
It may be reasonable to activate lead “noise” algorithms that withhold shocks when detected VT/VF is not confirmed on a shock or other far-field channel to avoid therapies for nonphysiologic signals.	IIb	C-EO
It may be reasonable to activate T-wave oversensing algorithms, to reduce inappropriate therapies.	IIb	C-LD
It may be reasonable to program the sensing vector from bipolar to integrated-bipolar in true-bipolar leads at risk for failure of the cable to the ring electrode to reduce inappropriate therapies.* **This is not intended as a long-term solution when a cable fracture has been identified.*	IIb	C-EO

Tachycardia Therapy Programming

Although therapies delivered by the ICD can abort SCD, appropriate and inappropriate ICD shocks have been associated with a considerable increase in the risk of mortality.[82,83,155–158] In the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT), the risk of mortality was 5-fold higher in patients who received appropriate ICD shocks and 2-fold higher in patients who received inappropriate shocks.[83] Similarly, pooling data from 4 studies of 2135 ICD patients, shocked VT was associated with a 32% increase in the risk of mortality. In that analysis, shocked patients had poorer survival than patients treated with ATP only.[155] ICD shocks are likely a marker of more advanced heart disease and subsequent death, but defibrillation therapies have been associated with troponin release and increased LV dysfunction with the potential of further mortality risk.

The incidence of appropriate and inappropriate ICD shocks depends on the patient's characteristics, including the indication for the device, concomitant medical therapies including antiarrhythmic medications, programming of the ICD, and the duration of follow-up. With regard to ICD programming, faster VT/VF detection rates, longer detection durations, use of a single zone, use of SVT discriminators, and delivery of ATP have been shown to reduce both appropriate and inappropriate shocks and to improve quality of life.[91,101,126,129,130,133,159,160] This programming might improve survival.[126] Indeed, several studies have shown that ATP is effective at terminating slow and fast VT with exceedingly low rates of adverse events like syncope.[93,135,161–165] The initial bias of the ICD community was to reserve ATP therapy for those patients in whom the therapy was demonstrated to be effective, usually during an electrophysiologic study. However, the approach of physician-directed programming based on the knowledge of induced arrhythmias was found to be significantly inferior to the routine strategic (EMPIRIC) programming of ATP. It is not reflective of the arrhythmias experienced outside the electrophysiology laboratory for primary and secondary prevention patients with ischemic and nonischemic substrates.[101,166] Although the ideal number of ATP bursts has not been definitively determined, current data support the use of up to 2 ATP attempts, given additional attempts yield very little additional efficacy.[93,135,161–165,167,168] In one study, up to 5 attempts were found to be safe.[168] The most effective ATP duration is likewise uncertain; however, in the ATP Delivery for Painless ICD Therapy (ADVANCE-D) trial—a prospective RCT of 925 patients—8-pulse ATP was as effective and safe as 15-pulse ATP.[169] The PITAGORA ICD clinical trial randomized 206 patients with an ICD to 2 ATP strategies: an 88% coupling interval burst vs a 91% coupling interval ramp. The results of the trial showed that over a median follow-up of 36 months and compared with ramp pacing, burst pacing was more effective for terminating fast VT episodes (between CL 240 and 320 ms).[170] In a prospective study of 602 patients, a strategy of tiered ATP and low-energy shock was efficacious and safe in patients with VT CL greater than 250 ms, with extremely low syncope rates.[171] However, a "real-world" retrospective study on 2000 patients with 5279 shock episodes from the LATITUDE remote monitoring system showed that the success rate of first shock as first therapy was approximately 90%, but the success rate was lower after failed ATP. Therefore, that study recommended programming a higher level of energy after ATP.[172] Finally, a substudy of the Effectiveness and Cost of ICD Follow-Up Schedule with Telecardiology (ECOST) study, which randomly assigned 433 patients to remote monitoring (n = 221; active group) vs ambulatory follow-up (n = 212; control group), showed that remote monitoring was highly effective in the long-term prevention of inappropriate ICD shocks through early detection and prevention of AF with a rapid ventricular rate, nonsustained VT, or diverted VT episodes.[173]

Benefits and Risks

The goal of ICD therapy is to prolong life while causing as little morbidity as possible. Although survival is quantifiably objective, morbidity is more subjective and includes both physical and emotional components. Clearly, shocks are usually painful to the patient, whereas ATP is typically not uncomfortable. However, there can be other morbidities related to both therapies, including mild to extreme emotional distress, syncope, palpitations, and proarrhythmia yielding more therapies and occasionally leading to death. Paradoxically, the need for life-saving therapies, including shocks and potentially ATP, might also be associated with increased mortality; however, the causal relationships are unclear. Also, the prevalence of tachycardia amenable to ATP or hemodynamic significance varies with the mechanism of the risk (e.g., long QT vs ischemic cardiomyopathy). In addition, although the risk of having a hemodynamically important or life-threatening arrhythmia can vary from patient group to patient group, the largest proportion of patients in whom ICD therapy is applied has yet to have a previously recorded arrhythmia, and we must therefore strategically choose on the basis of other factors how we will treat the first event and subsequent events.

Classification of Therapy

The literature uses definitions of therapies that differ from each other and that impact their results and conclusions. The occurrence rates of these events not only are dependent on their definition but also are highly dependent on the programming of the defibrillation system. Both shock and nonshock therapies can be categorized as being appropriate, inappropriate, and avoidable. Whereas appropriate and inappropriate therapies refer to therapies that were actually delivered, avoidable therapies are theoretical events in the future. These potential future tachycardia therapies, delivered for either appropriately or inappropriately detected events, can frequently be avoided by establishing programming to either prevent the initiation of the arrhythmia or to allow the condition to pass without therapy.

Appropriate

A response to a sustained ventricular arrhythmia (VT, VF) or hemodynamically poorly tolerated arrhythmias (e.g., associated with syncope, rate over 200 bpm, or hemodynamically compromising supraventricular arrhythmias).

Inappropriate

A response to signals generated by something other than sustained ventricular arrhythmias or hemodynamically poorly tolerated arrhythmias. Possible signals include supraventricular rhythms such as sinus tachycardia, AF, atrial flutter, reentrant SVT, atrial tachycardia, or instances of signal misinterpretation. Signal misinterpretation includes multiple counting of single events (e.g., atrial, T-wave or R-wave), environmental signals such as electromagnetic interference, frequent premature ventricular contractions (PVCs) and nonsustained ventricular arrhythmias, extracardiac physiologic signals (e.g., diaphragmatic or pectoral myopotentials), other implantable electronic devices (e.g., pacemakers, LV assist devices, nerve stimulators), inappropriate lead placement or dislodgment, conductor or insulation failures, header connection instability, and pulse generator failure.

Avoidable

Programming of detection and therapy parameters and algorithms so that shock or ATP therapy is withheld from arrhythmias that would be expected to be hemodynamically tolerated. Examples include self-terminating ventricular arrhythmias, ATP-susceptible ventricular arrhythmias, and overdrive suppression responsive rhythms. Many appropriate and most inappropriate therapies are also potentially avoidable.

Phantom

These are not true therapies; however, there is the patient's perception that a therapy was delivered. Interrogation of the ICD and/or coincident rhythm monitoring does not identify a tachycardia or therapy.

Unintended Consequences of ICD Therapy and ICD Therapy Programming

In the SCD-HeFT and MADIT II trials, inappropriate shocks more than doubled the risk of death. Mortality rates were substantially higher after shocks: 10% within days after the first shock, 25% within 1 year, and 40% by 2 years. The leading cause of death was progressive HF. In an analysis of the MADIT-CRT trial, the patients with appropriate shocks experienced increased mortality when compared with the patients without ICD shocks, after accounting for mechanical remodeling effects; this was not the case for patients who received appropriate ATP only.[156] ICD shocks have also been associated with independent predictors of mortality in the large ALTITUDE registry of 3809 ICD recipients and in a meta-analysis of ICD trials in which ATP was applied.[155,157] Emotional morbidities associated with ICD shocks are well recognized and include anxiety, depression, and posttraumatic stress disorders.[174–176] Phantom shocks can result from fear and/or anxiety and have a reported incidence of 5% in a European study of ICD recipients over 35 months of follow-up.[177] If possible, and when safe, it is best to avoid both the discomfort and psychological impact of shocks for ventricular arrhythmias, supraventricular arrhythmias, noise events including lead failures, and for self-terminating arrhythmias, as is discussed in the section on tachycardia detection. The 1500-patient MADIT-RIT study demonstrated a mortality reduction by changing both tachycardia detection criteria and tachycardia therapy (shocks and ATP). Therefore, it is difficult to assign the outcome result to ATP, shocks, or both when compared with older, more conventional programming.[126] In addition, in a randomized study of remote follow-up of ICDs, home monitoring showed an incidence of 52% fewer inappropriate shocks, 72% fewer hospitalizations due to inappropriate shocks, 76% fewer capacitor charges, and a significant positive impact on battery longevity.[178]

ATP

Several large clinical trials have established the safety and efficacy of ATP as a first-line therapy to treat even very fast VTs.[93,95,101,133] The use of first-line ATP involving VT at rates between 188 and 250 bpm in the PainFREE Rx II trial resulted in a 71% relative shock reduction.[93] In the PREPARE study, a primary prevention cohort of 700 patients was programmed with 30 of 40 detection intervals with ATP-first for VT between 182 and 250 bpm with SVT discriminators active up to 200 bpm. The results demonstrated a robust absolute risk reduction for shocks at 1 year from 17% to 9% without an increase in arrhythmic syncope when compared with historical controls.[133] Similar findings were noted in the RELEVANT study, which evaluated a cohort of patients with nonischemic heart disease and cardiac resynchronization defibrillators.[95] In the earlier EMPIRIC study, standardized VT detection and ATP therapy parameters demonstrated a reduction in shocks when compared with physician-tailored treatment in a randomized assessment of 900 primary prevention patients.[101] The use of ATP during ICD capacitor charging has been clinically validated as safe and effective.[163] It is important to recognize that inappropriate therapies including inappropriate ATP, delivered primarily in the setting of supraventricular arrhythmias, have been associated with increased mortality in the MADIT-RIT and MADIT-CRT trials.[156,179] However, the overall safety of ATP and its role as a contributor to improved survival are well established, particularly in terms of preventing avoidable ICD shocks.

Customized vs Strategic Programming

Because primary prevention patients have no prior ventricular arrhythmias, programming individual devices on implant is largely empiric. There are more data for secondary prevention patients, but how the patient will behave in the future is still uncertain. The ability to individualize the

antitachycardia programming for patients with both primary and secondary prevention indications was tested in the EMPIRIC trial and found to be an inferior approach to prevent these therapy events.[101] The application of standardized programming and borrowing data from the Pain-FREE Rx II and PREPARE studies resulted in a comprehensive review of programming and its application across manufacturers.

Secondary Prevention

For the secondary prevention ICD patient, specific knowledge of the patient's arrhythmia history facilitates the creation of an effective antitachycardia programming strategy. Using what is known about the ventricular arrhythmia, including any electrocardiograms, available telemetry strips, and EMS recordings, provides insight into the arrhythmia mechanism. In cases of monomorphic VT, discerning the rate (CL) and the hemodynamic impact is useful in making choices, particularly for detection at a minimum; the device must be programmed with active VT detection zones sufficient to cover the clinical arrhythmia. Slower, monomorphic VT that is better tolerated hemodynamically favors a robust approach using ATP termination with at least 2–3 sequences and at least 8 pulses. The use of a second burst of ATP has also been shown to increase effectiveness from 64% to 83% in the fast VT range of 188 to 250 bpm.[167] Although a second burst has clear value, value beyond 2 bursts is limited, except in rare situations.[101] The use of ICDs in patients with implanted LV assist devices allows prolongation of detection times and programming of multiple ATP attempts without significant risk to the patient, and it reduces the opportunity for shock therapies. Adjunct medications and ablation of VT (or SVT) might also be considered for cases in which slow VT occurs or if there is an overlap between the SVT and VT rates, leading to ICD therapies.

Tachycardia Therapy Programming Recommendations	Class of Recommendation	Level of Evidence
It is recommended in all patients with structural heart disease and ATP-capable ICD therapy devices that ATP therapy be active for all ventricular tachyarrhythmia detection zones to include arrhythmias up to 230 bpm, to reduce total shocks except when ATP is documented to be ineffective or proarrhythmic.	I	A
It is recommended in all patients with structural heart disease and ATP-capable ICD therapy devices that ATP therapy be programmed to deliver at least 1 ATP attempt with a minimum of 8 stimuli and a cycle length of 84%–88% of the tachycardia cycle length for ventricular tachyarrhythmias to reduce total shocks, except when ATP is documented to be ineffective or proarrhythmic.	I	A
It is indicated to program burst ATP therapy in preference to ramp ATP therapy, to improve the termination rate of treated ventricular tachyarrhythmias.	I	B-R
It is reasonable to activate shock therapy to be available in all* ventricular tachyarrhythmia therapy zones, to improve the termination rate of ventricular tachyarrhythmias. **Rarely, to limit patient discomfort and anxiety, hemodynamically stable slow VT can be treated without programming a backup shock.*	IIa	C-EO
It is reasonable to program the initial shock energy to the maximum available energy in the highest rate detection zone to improve the first shock termination of ventricular arrhythmias unless specific defibrillation testing demonstrates efficacy at lower energies.	IIa	C-LD

Intraprocedural Testing of Defibrillation Efficacy

The efficacy of the ICD for the primary and secondary prevention of SCD has been well established in several landmark clinical trials.[180–185] Most of these trials have required induction, detection, and termination of VF at the time of implantation as a measure of defibrillation efficacy and as a surrogate of the ICD's ability to prevent SCD. Testing defibrillation efficacy has been considered an integral part of ICD implantation for many years, and it is performed to establish the appropriate connection of high-voltage electrodes and to test the ability of the ICD to detect and terminate VF with a shock. However, identifying system failures or high defibrillation thresholds is difficult, mainly due to the low prevalence, which also depends on the definition employed, about 5% combined. Significant improvements over the past 2 decades have reduced energy requirements for defibrillation.[186–189] Similarly, current transvenous ICD technology is capable of delivering energies of 35–40 J, raising the question of the value of routine defibrillation testing (DT). Physicians have therefore gravitated to implanting ICDs with minimum or no DT with wide variability in practice, despite a paucity of rigorous data. DT is currently being performed during ICD implant in only about half the procedures.[190–195] Studies evaluating DT are summarized in Table 4.

Table 4 Defibrillation testing

Study (n)	Patients (DT/no DT)	Results and remarks
CREDIT[193] (361) Prospective multicenter registry	64%/36%	More frequent DT for new implants vs generator replacements (71% vs 32%, $P = .0001$), DT for primary and secondary prevention indications (64% vs 63%, P = NS). Reasons for no DT were as follows: unnecessary (44%); persistent atrial fibrillation (37%); no anesthetist (20%); and patient or physician preference (6%). DT was not performed in a third of ICD implants, usually due to a perceived lack of need or relative contraindication. Nonconsecutive patients, single manufacturer.
Ontario DT Registry[225] (2173) Prospective multicenter registry	PP: 65%/45% SP: 67%/43% GR: 24%/ 76%	Multivariate predictors for DT included new ICD implant (OR 13.9; $P < .0001$), DCM (OR 1.8; $P < .0001$), amiodarone (OR 1.5; $P = .004$), and LVEF >20% (OR 1.3; $P = .05$). History of AF (OR 0.58; $P = .0001$) or OAC use (OR: 0.75; $P = .03$) was associated with a lower likelihood of having DT. Complications, including death, were similar: DT 8.7% vs no DT 8.3% ($P = .7$). All consecutive implants at 10 centers in Ontario
NCDR[223] (64,277) Prospective multicenter registry	71%/29%	No DT; older, higher incidence of HF, lower LVEF, atrial arrhythmias, and a primary prevention indication; hospital adverse events; DT 2.56% vs. 3.58% no DT ($P < .001$). Death or any no DT complication (OR [95%CI] 1.46 [1.33–1.61]; $P < .001$), DT is not performed on many (29%) patients in clinical practice. Generator replacement excluded.
Israel DFT Registry[226] (3596) Prospective multicenter registry	17%/83%	Variables associated with ICD testing: implantation for secondary prevention (relative risk [RR] 1.87), prior ventricular arrhythmias (RR 1.81), use of AADs (RR 1.59), and sinus rhythm (RR 2.05). No significant differences in the incidence of mortality, malignant ventricular arrhythmias, or inappropriate ICD discharges were observed between patients who underwent DT compared with those who were not tested. All consecutive implants during 1 year at 22 centers: HOCM: 6.2% DT, 6.3% no DT; ARVC: 0.6% DT, 0.5% no DT; congenital heart disease: 0.8% DT, 2.1% no DT; Long QT: 1.2% DT, 0.26% no DT; Brugada syndrome: 0.3% DT, 0.44% no DT; family history cardiac death: 5.3% DT, 4.7% no DT.
SAFE-ICD[210] 2120 Prospective observational study	836 DT 1284 no DT	Followed up for 24 months. Primary endpoint was composite of severe implant complications, sudden cardiac death, or resuscitation at 2 years. Primary endpoint: Of 34 patients, 12 intraoperative complications (8 in DT; 4 in no DT) and 22 during follow-up (10 in DT; 12 in no DT). Estimated yearly incidence: DT 1.15% (0.73 to 1.83) and no DT 0.68% (0.42 to 1.12); no difference. In 41 Italian centers. The only exclusion criterion was refusal to provide consent. Other ICD indications: 15% DT, 12% no DT.
Healey JS, et al[212] (145) Randomized Multicenter subgroup study	75 DT 70 no DT	All patients in DT arm achieved a successful DT ($\leq$25 J); 96% without requiring any system modification. No patient experienced perioperative stroke, myocardial infarction, HF, intubation, or unplanned ICU stay. The composite of HF hospitalization or all-cause mortality occurred in 10% of no DT vs. 19% of the DT arm (HR: 0.53; 95% CI 0.21–1.31; $P = .14$). Conclusions: Perioperative complications, failed appropriate shocks, and arrhythmic death are uncommon regardless of DT. There was a nonsignificant increase in the risk of death or HF hospitalization with DT. Excluded: intracardiac thrombus, persistent or permanent AF without appropriate anticoagulation, right-sided implant, or felt ineligible for DT.

SIMPLE[215]	1253 DT	Primary outcome: arrhythmic death or failed appropriate shock occurred in fewer patients (90 [7% per year]) in no DT vs DT (104 [8% per year]; HR 0.86; 95% CI 0.65–1.14; *P* noninferiority $<.0001$). The first safety composite outcome occurred in 69 of 1236 patients (5.6%) with no DT and in 81 of 1242 patients (6.5%) with DT ($P = .33$). The second safety composite outcome, including only events most likely to be directly caused by DT, occurred in 3.2% of patients without DT vs 4.5% with DT ($P = .08$). Routine DT at the time of ICD implantation is generally well tolerated but does not improve shock efficacy or reduce arrhythmic death.
2500 Randomized multicenter trial	1247 no DT	Single manufacturer, excluded patients on active transplantation list, ICD expected to be right-sided implant. HOCM: 4.2% DT, 3.4% no DT; long QT, Brugada syndrome, or catecholaminergic polymorphic VT: 2.3% DT, 1.9% no DT.
NORDIC ICD[216] 1077 Randomized multicenter trial	540 DT 537 no DT	ICD shocks were programmed to 40 J in all patients. Primary endpoint: first shock efficacy for all true VT and fibrillation episodes during 22.8 months of follow-up. Noninferior with or without DT. First shock efficacy 3.0% in favor of no DT. A total of 112 procedure-related serious adverse events occurred within 30 days in 94 DT patients (17.6%) and 89 events in 74 no-DT patients (13.9%). Excluded were the following: survived an episode of VF due to acute ischemia or potentially reversible causes, listed for heart transplant, life expectancy less than the study duration due to malignant conditions, terminal renal insufficiency, any conditions precluding DT (e.g., left atrial or ventricular thrombus), preexisting or previous ICD or CRT-D, or if the device was intended to be implanted on the right side.

AAD = antiarrhythmic drug; ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = cpronary heart disease; HOCM = hypertrophic obstructive cardiomyopathy; ICU = intensive care unit; OAC = oral anticoagulant

Intraprocedural Testing of Defibrillation Efficacy Recommendations	Class of Recommendation	Level of Evidence
Defibrillation efficacy testing is recommended in patients undergoing a subcutaneous ICD implantation.	I	C-LD
It is reasonable to omit defibrillation efficacy testing in patients undergoing initial left pectoral transvenous ICD implantation procedures where appropriate sensing, pacing, and impedance values are obtained with fluoroscopically well-positioned RV leads.	IIa	B-R
Defibrillation efficacy testing is reasonable in patients undergoing a right pectoral transvenous ICD implantation or ICD pulse generator changes.	IIa	B-NR
Defibrillation efficacy testing at the time of implantation of a transvenous ICD should not be performed on patients with a documented nonchronic cardiac thrombus, atrial fibrillation or atrial flutter without adequate systemic anticoagulation, critical aortic stenosis, unstable CAD, recent stroke or TIA, hemodynamic instability, or other known morbidities associated with poor outcomes.	III (Harm)	C-LD

One of the most important reasons to avoid DT at the time of ICD implantation is that testing might result in complications or even death. The risks of DT include (1) those related to VF itself, which can lead to circulatory arrest and hypoperfusion, (2) risks related to the shocks delivered to terminate VT, and (3) risks related to anesthetic drugs that are required for heavier sedation, which are used to provide patient comfort during testing.

Periprocedural Mortality

Although improved ICD technology has led to the need for fewer inductions of VF at the time of implantation testing, procedure-related mortality has not been completely eliminated. Using modern ICD technology with transvenous systems and biphasic waveforms, the perioperative mortality rate within 30 days of implantation is reported to be 0.2% to 0.4%.[191,196] Recent data from the National Cardiovascular Data Registry (NCDR) demonstrated an in-hospital mortality of 0.03% following ICD implantation, with death occurring in the laboratory in 0.02%.[196] A Canadian report from 21 implanting centers estimates that 3 of 19,067 deaths (0.016%) are related to DT.

DT-Related Complications

Complications occurring during ICD implantation procedures are infrequent, and many can be directly or indirectly related to DT. Adverse effects related to DT include myocardial injury, depression of contractile function leading to worsening of HF, persistent hypotension, central nervous system injury, thromboembolic events, or respiratory depression.

Transient central nervous system hypoperfusion and cerebral ischemic changes can be demonstrated during intraoperative electroencephalographic (EEG) monitoring at the time of DT. However, EEG recovery occurs within less than 30 seconds, with a slightly longer time to the return of middle cerebral blood flow.[197–199] However, the clinical relevance of this transient finding is unclear because DT does not appear to cause cognitive dysfunction 24–48 hours following ICD implantation.[200,201] Although an increase in biochemical markers of myocardial injury can be observed during ICD implantation or after spontaneous clinical shocks, true intraoperative myocardial infarction (MI) is rare, even when extensive DT is performed.[202–205] In 2 recent studies using transvenous ICDs and a more abbreviated testing protocol, there was no significant increase in CK, CK-MB, myoglobin, and NT-proBNP before and after DT, whereas elevated levels of high-sensitive troponin T were observed after DT.[206,207] In the NCDR ICD Registry, the incidence of MI during ICD implantation was reported to be 0.02%.[196]

Defibrillator shocks and VF transiently depress contractile function, although fatal pulseless electrical activity is rare at the time of ICD implantation.[202,206,208–210] Refractory VF has been reported to occur during DT, but this is also uncommon, particularly with contemporary devices. One study reported that all tested ICD shocks failed and at least 3 external rescue shocks were required in 0.5% of patients.[203] A Canadian study reported that 27 of 19,067 implants (0.14%) required prolonged resuscitations during DT.[211]

Thromboembolic complications can occur during DT in the presence of intracardiac thrombus or when there are less than 3 weeks of therapeutic and uninterrupted anticoagulation in the setting of AF. Stroke or transient ischemic attack (TIA) is reported to occur in 0.026%–0.05% of cases.[204,211] Multiple strategies have been employed, but none were documented to reduce the incidence of thromboembolism, including the avoidance of DT. These include preprocedure transesophageal echocardiography to exclude left atrial appendage thrombus and deferring testing when a thrombus is identified, or using transthoracic echocardiography to detect LV thrombi.

Anesthetic agents can contribute to complications related to a depressant effect on myocardial contractility or can lead to respiratory depression if oversedation occurs. Heavier sedation is typically used in patients undergoing DT. Although patients with underlying chronic obstructive pulmonary disease or sleep apnea might be at increased risk, oversedation and respiratory depression could occur in any patient. Randomized trial data can help to identify which adverse events are directly (or indirectly) related to DT. For example, stroke or TIA might be "directly" related to DT due to dislodgment of intracardiac thrombus during conversion of AF in the absence of therapeutic anticoagulation, and an episode of prolonged hypotension could result in reduced cerebral perfusion. Respiratory depression, respiratory failure requiring intubation, or hypotension might be direct results of DT or might be due to the drugs required to perform testing. Pulseless electrical activity or even death can occur with hemodynamic complications related to induction of VF or multiple external shocks. In contrast, DT can indirectly increase the risk for pneumothorax, perforation, tamponade, lead dislodgment, or infection as more leads are inserted, or the procedure might be prolonged due to the system modifications required to improve defibrillation efficacy; however, all these complications can also occur in the absence of DT. In addition, due to the rates and types of adverse events reported in the literature, it appears that overall complication rates are primarily driven by mechanical complications or infection, most of which are not related to DT.

In a substudy of the Resynchronization for Ambulatory Heart Failure Trial (RAFT), in which 145 patients were randomized to DT compared with no DT at the time of initial ICD implantation, the risk of perioperative complications was extremely low, regardless of DT performance.[212] There was, however, a nonsignificant increase in the risk of death or HF hospitalization in the group that underwent DT. Likewise, no significant difference in implant-related complications was demonstrated in DT compared with the groups without DT in the Safety of Two Strategies of ICD Management at Implantation (SAFE-ICD) study, a prospective observational study of 2120 patients performed at 41

centers.[213] Similar findings were observed in the prospective randomized Test-No Test Implantable Cardioverter Defibrillator (TNT-ICD) pilot study on 66 patients, in which there was no difference in adverse events between patients who underwent testing compared with those who did not.[214]

The Shockless Implant Evaluation (SIMPLE) trial is the largest randomized study assessing the effect of DT on clinical outcomes.[215] This large-scale study randomized 2500 patients to DT or not at the time of ICD implantation; 1253 patients were randomly assigned to DT and 1247 were assigned to no-testing, and were followed for a mean of 3.1 years (SD 1.0). The primary outcome of arrhythmic death or failed appropriate shock was noninferior (90 [7% per year]) in the no-testing group compared with patients undergoing DT (104 [8% per year]; HR 0.86; 95% CI 0.65–1.14; P noninferiority $<.0001$). The first safety composite outcome occurred in 69 of 1236 patients (5.6%) with no testing and in 81 of 1242 patients (6.5%) with DT ($P = .33$). The second, prespecified safety composite outcome, which included only events most likely to be directly caused by testing, occurred in 3.2% of patients with no testing and in 4.5% with DT ($P = .08$). Heart failure needing intravenous treatment with inotropes or diuretics was the most common adverse event (in 20 of 1236 patients [2%] in the no-testing group vs 28 of 1242 patients [2%] in the testing group, $P = .25$). In summary, routine DT at the time of ICD implantation is generally well tolerated without a statistically significantly increased rate of complications, but it also does not improve shock efficacy or reduce arrhythmic death.

Finally, the No Regular Defibrillation Testing In Cardioverter Defibrillator Implantation (NORDIC-ICD) trial, another prospective randomized parallel group multicenter noninferiority trial conducted in 48 centers in Europe, assessed the effects of DT at the time of ICD implantation on first shock efficacy.[216] The primary endpoint was different from the SIMPLE trial and assessed the average first-shock efficacy for all true VT and VF episodes occurring in any patient during follow-up. NORDIC-ICD randomized 540 patients to DT and 537 to no DT at the time of ICD implantation. During a median follow-up of 22.8 months, the first shock efficacy was demonstrated to be noninferior in the patients undergoing ICD implantation without DT, with a difference in first shock efficacy of 3.0% in favor of the no-DT test group (95% CI 3.0%–9.0%; P noninferiority $<.001$). Overall, 112 procedure-related serious adverse events were reported within 30 days of ICD implantation in 94 patients (17.6%) undergoing DT compared with 74 patients (13.9%) not undergoing DT ($P = .095$). The authors concluded that defibrillation efficacy without DT was noninferior to ICD implantation with DT in left-sided ICD implants. Because no major benefit or harm associated with DT was detected, in patients with a left-sided pectoral implantation it is reasonable to omit routine VF induction and DT during ICD implantation, assuming stable ICD lead position and good sensing and capture function.[217–220] This approach is particularly applicable to patients with ischemic and idiopathic dilated cardiomyopathy, given these entities were well represented in the studied cohort. Patients well represented within the cohort included those with implantation in the left pectoral location, those indicated for primary and secondary prevention of SCD, and patients with ischemic and nonischemic cardiomyopathies. Fewer data are available regarding other cardiomyopathies, such as patients with hypertrophic obstructive cardiomyopathy, congenital channelopathies, patients undergoing generator replacement, and procedures in the right pectoral location. In these instances, and when there is any question of the adequacy of the lead position or function, DT is reasonable. It is worth emphasizing that a nontesting strategy requires an anatomically well-positioned defibrillation lead in the RV with adequate sensing of intrinsic R waves (>5–7 mV), adequate pacing thresholds, and a thorough verification of proper lead connection.

Other important considerations include the use of alternative RV defibrillation lead sites such as the mid-septum. Pooled data from 2 randomized studies do not indicate a clinically relevant elevation of energy required for defibrillation with mid-septal sites. Positioning of the RV defibrillation lead in other positions such as the RV outflow tract has not been systematically addressed.[221]

The SIMPLE trial data were consistent between subgroups, both from patients with single- or dual-coil ICD leads and with or without the use of amiodarone. More recently, the Multicenter Comparison of Shock Efficacy Using Single vs Dual-Coil Lead Systems and Anodal vs Cathodal Polarity Defibrillation in Patients Undergoing Transvenous Cardioverter-Defibrillator Implantation (MODALITY) study was reported.[222] This was a multicenter registry that prospectively followed 469 consecutive patients undergoing DT at the time of implant; 158 (34%) had dual-coil and 311 (66%) had single-coil lead systems configuration, 254 (54%) received anodal shock, and 215 (46%) received cathodal shock. In 35 patients (7.4%), the shock was unsuccessful. No significant differences in the outcome of DT using a single- vs dual-coil lead were observed, but the multivariate analysis showed an increased risk of shock failure using cathodal shock polarity (odds ratio [OR] 2.37; 95% CI 1.12–5.03). These and other registry data support the use of either single- or dual-coil leads, preferably programmed to deliver anodal shocks.[211,213,223]

Performing DT has not been determined to be harmful or inappropriate. One reason to perform DT in specific populations is that high defibrillation thresholds have been reported in 2.2% to 12% of subjects undergoing DT. The probabilistic nature of DT with the failure of a single shock 10 J below the maximum ICD output does not necessarily imply long-term ICD failure. Determinations of DT using multiple shock protocols have reported that a safety margin of only 5.2 ± 1.1 J has a 97.3% rate of successful VT/VF conversion[224]; however, the inability to convert VF at maximum output occurs in approximately 1% of procedures during DT. The long-term outcomes of these patients have not been evaluated without modification of the lead system. Further supporters of DT suggest that routine testing is

necessary to identify system integrity and sensing failures. R-wave amplitude ≤5–7 mV at implant almost invariably reliably sense VF.[190,221] Failure to sense and some inner insulation failures might only be detected by DT. This situation has not been systematically evaluated.

Contraindications to Defibrillation Threshold Testing

A great paucity of systematic data limits the assessment of the literature regarding contraindications to DT. Most implanters tend to avoid DT in patients perceived to be at high risk. Information derived from an NCDR-ICD registry identified advanced age, impaired LVEF, NYHA Class IV HF, atrial fibrillation/flutter, need to withhold warfarin, and several other factors as high-risk situations. Unfortunately, the strength of these associations was weak, given the ORs were under 2.[223] Other registries have identified patients with broader QRS durations, advanced NYHA class, and CRT as reasons for not performing DT.[213] There are no convincing data to identify high-risk patients, and clinical judgment has likely kept the highest-risk patients, particularly those who were hemodynamically unstable, from being tested in the current literature.

S-ICD

Patients receiving a nontransvenous ICD system should routinely undergo DT, given there are no current data regarding the safety and efficacy of not performing DT with this lead configuration and device.

Conclusion

In providing focused recommendations for ICD programming and DT of patients implanted with a device we have intentionally left many questions unanswered. There are hundreds of choices for which there are inadequate data to provide evidence or consensus-based recommendations. This document is a long overdue effort to provide analysis and guidance to the clinician as to how to make strategic programming choices in the implementation of ICD therapy. The four continental electrophysiology societies limited the discussion and recommendations to four areas for which there was sufficient consensus and data. In the review process, clearly articulated opinions pointed out that additional recommendations are desirable. However, there is an information gap of insufficient data filled with opinions and logical arguments. Generalizations and inferences were made from the existing data, e.g., taking data from pacemaker trials and applying to this to ICD bradycardia programming, logical arguments bridging the differences between primary and secondary prevention patients for tachycardia detection and therapy, and the use of non-inferiority data to make decisions about DT. This document is a beginning, necessary because there are now sufficient data to support recommendations that improve the safety, morbidity, and mortality of patients with ICDs.

Appendix Disclosures

Tables A1, A2

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Table A1 Author Disclosure Table

Writing Group Member	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
Bruce L. Wilkoff, MD, FHRS, CCDS (Chair)	Cleveland Clinic, Cleveland, OH	1: St. Jude Medical, Boston Scientific Corp. 2: Spectranetics Corporation, Medtronic, Inc.	None	None	None	None	Equity interests: 1 CardioMEMS; Royalty Income:2 Medtronic, Inc.
Laurent Fauchier, MD, PhD (Co-Chair)	Centre Hospitalier Universitaire Trousseau, Tours, France	1: Bayer HealthCare, LLC; Boehringer Ingelheim; Boston Scientific Corp.; Brital Meyers Squibb; Medtronic, Inc.; Novartis Pharmaceuticals Corp; Sanofi Aventis; Daiichi; Sorin Group	None	None	None	None	None
Martin K. Stiles, MBCHB, PhD (Co-Chair)	Waikato Hospital, Cardiology, Hamilton, New Zealand	1: Medtronic, Inc.; Boston Scientific Corp.; Biotronik	None	None	2: Medtronic, Inc. 3: Biosense Webster, Inc. St. Jude Medical	None	None
Carlos A. Morillo, MD, FRCPC, FHRS (Co-Chair)	McMaster University, Hamilton, Canada	1: Sanofi Aventis; Biotronik; 2: Boehringer Ingelheim; Marck Pharmaceuticals	1: Boehringher Ingelheim; Sanofi Aventis; 2: Merck Pharmaceuticals	3: St. Jude Medical; 4: Medtronic, Inc.; Boston Scientific Corp.	None	None	None
Sana Al-Khatib, MD, MHSc, FHRS, CCDS	Duke University Medical Center, Durham, NC	None	None	None	None	None	None
Jesús Almendral, MD, PhD, FESC	Hospital General Alicante, Alicante, Spain	1: Boston Scientific, St. Jude Medical, Medtronic	None	None	2: St. Jude Medical	None	None
Luis Aguinaga, MD, PhD, FACC, FESC	Centro Privado De Cardiologia, Tucuman, Argentina	None	None	None	None	None	None
Ronald D. Berger, MD, PhD, FHRS	Johns Hopkins University, Baltimore, Maryland	2: Boston Scientific Corp;	None	None	3: Medtronic, Inc; 4: St. Jude Medical	None	Royalty Income:3: Zoll Medical Corporation
Alejandro Cuesta, MD, PhD, FESC	Montevideo, Uruguay	None	None	None	None	None	None

Table A1 (*continued*)

Writing Group Member	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
James P. Daubert, MD, FHRS	Duke University Medical Center, Durham, North Carolina	1: Medtronic, Inc.; St. Jude Medical; Boston Scientific Corp.; Sorin Group; CardioFocus, Inc.; Gilead Sciences, Inc.; Biosense Webster, Inc.; Biotronik; Sanofi Aventis	None	5: Boston Scientific Corp.; Biosense Webster, Inc.; Medtronic, Inc.; Gilead Sciences Inc.	3: Medtronic, Inc.; Boston Scientific Corp.; Biotronik; St. Jude Medical; Biosense Webster, Inc.; Bard Electrophysiology	None	None
Sergio Dubner, MD, FACC	Clinica y Maternidad Suizo Argentina and De Los Arcos Sanatorio, Buenos Aires, Argentina	1: Boehringer Ingelheim	None	None	None	None	0; Officer, trustee, director or any other fiduciary role: Cardiology Journal
Kenneth A. Ellenbogen, MD, FHRS	Virginia Commonwealth University Medical Center, Richmond, VA	1: Biotronik; St. Jude Medical; Cameron Health Corp.; American College of Cardiology; American Heart Association; CardioNet, Inc.; Daiichi; Janssen Pharmaceuticals; Biosense Webster, Inc.; AtriCure, Inc.; Pfizer, Inc. 2: Boston Scientific Corp.; Medtronic, Inc.; Sentreheart; Endosense; Elsevier	None	1: National institutes for Health; St. Jude Medical; 2: Biosense Webster, Inc. 3: Medtronic, Inc.; Boston Scientific Corp.	2: Medtronic, Inc. 3: Boston Scientific Corp; Biosense Webster, Inc.	None	None
N.A. Mark Estes III, MD	New England Medical Center, Boston, Massachusetts	1: Medtronic, Inc.; St. Jude Medical; 2: Boston Scientific Corp.	None	4: Boston Scientific Corp.	4: Medtronic, Inc.; Boston Scientific Corp.; St. Jude Medical	None	None
Guilherme Fenelon, MD, PhD	Federal University of São Paulo, São Paulo, Brazil	None	None	2: Biosense Webster, Inc.	None	None	None

Table A1 (*continued*)

Writing Group Member	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
Fermin C. Garcia, MD	Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania	None	1: St. Jude Medical; Biotronik; 2: Boston Scientific Corp.	1: Biosense Webster, Inc.	None	None	None
Maurizio Gasparini, MD	Humanitas Research Hospital, Milan, Italy	None	1: Boston Scientific Corp.; Medtronic, Inc.	None	None	None	None
David E. Haines, MD, FHRS	William Beaumont Hospital Division of Cardiology, Royal Oak, Michigan	None	None	None	None	1: nContact, Inc.	None
Jeff S. Healey, MD, MSc, FRCPC, FHRS	Population Health Research Institute, McMaster University, Hamilton, Canada	1: Boehringer Ingelheim; St. Jude Medical; Bayer HealthCare, LLC	None	4: Bristol Meyers Squibb; 5: Boston Scientific Corp.; St. Jude Medical; Boehringer Ingelheim; Medtronic, Inc.; Bristol Meyers Squibb; Pfizer, Inc.	None	None	None
Jodie L. Hurtwitz, MD	North Texas Heart Center, Dallas, Texas	1: Biosense Webster, Inc.	1: Boehringer Ingelheim; Medtronic, Inc.; St. Jude Medical	None	None	None	None
Roberto Keegan, MD	Hospital Privado del Sur, Bahia Blanca, Argentina	None	None	None	None	None	None
Christof Kolb, MD	Deutsches Herzzentrum Munchen, Munich, Germany	1: Biotronik; Boston Scientific; Medtronic; Sorin Group; St. Jude Medical; German Cardiac Society	None	1: Biotronik, Sorin Group; St. Jude Medical	None	None	None
Karl-Heinz Kuck, MD, FHRS	Allgemeines Krankenhaus St. Georg, Hamburg, Germany	1: Biotronik; St. Jude Medical; Stereotaxis, Inc.; Biosense Webster, Inc.	None	1: Biotronik; St. Jude Medical; Biosense Webster, Inc.; Stereotaxis, Inc.	None	1: Endosense	None

Table A1 (*continued*)

Writing Group Member	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
Germanas Marinskis, MD, FESC	Vilnius University, Clinic of Cardiac and Vascular Diseases, Lithuania	None	None	None	None	None	None
Martino Martinelli, MD, PhD	Instituto do Coração, Universidade de São Paulo, São Paulo, Brazil	1: Biotronik	None	1: St. Jude Medical	None	None	None
Mark McGuire, MBBS, PhD	Royal Prince Alfred Hospital, Sydney, Australia	None	1: Medtronic, Inc.; St. Jude Medical; Astra Zeneca Pharmaceuticals; Sanofi Aventis	None	None	None	None
Luis G. Molina, MD, DSc	Mexico's National University, Mexico's General Hospital, Mexico City, Mexico	3: Medtronic, Inc.	None	None	None	None	None
Ken Okumura, MD, PhD	Hirosaki University Graduate School of Medicine, Hirosaki, Aomori, Japan	1: Bayer/Schering Pharma; Boehringer Ingelheim; Bristol Meyers Squibb; Daiichi; Biosense Webster, Inc.	None	2: Biosense Webster, Inc.	None	None	None
Alessandro Proclemer, MD	Azienda Ospedaliero Universitaria S. Maria della Misericordia–Udine, Udine, Italy	1: Medtronic, Inc.; Boston Scientific Corp.	None	1: Medtronic, Inc.; Sorin Group; Boston Scientific Corp.	None	None	None
Andrea M. Russo, MD, FHRS	Cooper University Hospital, Camden, New Jersey	1: Medtronic, Inc.; Boston Scientific Corp.; St. Jude Medical; Biotronik	None	1: Boston Scientific Corp.; Biotronik; 2: Medtronic, Inc.; Cameron Health Corp.	2: Medtronic, Inc.; Boston Scientific Corp.	None	None
Jagmeet P. Singh, MD, DPhil, FHRS	Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts	1: Boston Scientific Corp.; Biotronik; Medtronic, Inc.; CardioInsight Technologies; St. Jude Medical; Respicardia; Sorin Group; Guidepoint Global Advisors	None	4: Boston Scientific Corp.; Medtronic, Inc.; 5: Biotronik; St. Jude Medical	None	None	None

Table A1 (*continued*)

Writing Group Member	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
Charles D. Swerdlow, MD, FHRS	Cedars-Sinai Medical Center, Beverly Hills, California	1: St. Jude Medical; Sorin Group; 2: Medtronic, Inc.	2: Medtronic, Inc.	None	None	None	Intellectual Property Rights: 3: Medtronic, Inc.
Wee Siong Teo, MBBS, FHRS	National Heart Centre Singapore, Singapore, Singapore;	1: Biosense Webster, Inc.; Sanofi Aventis; Medtronic, Inc.; Bayer HealthCare, LLC	1: Biosense Webster, Inc.; Boehringer Ingelheim; St. Jude Medical	None	None	None	None
William Uribe, MD	Cardiologia Ces Y Centros Especializados San Vicente Fundacion, Medellin y Rionegro, Colombia	1: St. Jude; Bayer; Sanofi; Boehringer Ingelheim	None	None	None	None	None
Sami Viskin, MD	Tel Aviv Sourasky Medical Center and Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel	0: Boston Scientific Corp.	None	None	None	None	None
Chun-Chieh Wang, MD	Chang Gung Memorial Hospital, Taipei, Taiwan	None	None	None	None	None	None
Shu Zhang, MD	National Center for Cardiovascular Disease and Beijing Fu Wai Hospital, Peking Union Medical College and China Academy of Medical Sciences, Beijing, China	1: Medtronic, Inc.; Johnson and Johnson; St. Jude Medical; Boston Scientific Corp.	None	None	None	None	None

Number Value: 0 = $0; 1 = <$10,000; 2 = >$10,000 to <$25,000; 3 = >$25,000 to <$50,000; 4 = >$50,000 to <$100,000; 5 = >$100,000.

Table A2 Reviewer Disclosure Table

Peer Reviewer	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
Giuseppe Boriani, MD, PhD	University of Bologna, Italy	1: Medtronic, Inc.; Boston Scientific Corp.; St. Jude Medical	None	None	None	None	None
Michele Brignole, MD, FESC	Ospedali del Tigullio, Lavagna, Italy; University of Wisconsin, Madison, WI	None	None	None	None	None	None
Alan Cheng, MD, FHRS	Johns Hopkins University School of Medicine, Baltimore, MD	1: Boston Scientific Corp.; Medtronic, Inc.; St. Jude Medical	None	None	None	None	None
Thomas C. Crawford, MD, FACC, FHRS	The University of Michigan Health System, Ann Arbor, MI	None	None	None	None	None	None
Luigi Di Biase, MD, PhD, FACC, FHRS	Montefiore-Einstein Center for Heart & Vascular Care, Bronx, NY	1: Stereotaxis; St. Jude Medical; Boston Scientific Corp.; Medtronic, Inc.; EpiEP, Inc.; Janssen; Pfizer 2: Biosense Webster, Inc.; Biotronik	None	None	None	None	None
Kevin Donahue, MD	University of Massachusetts Medical School, Worcester, MA	None	None	0: Medtronic, Inc.; Boston Scientific Corp.; St. Jude Medical	None	None	1: NIH (salary)
Andrew E. Epstein, MD, FAHA, FACC, FHRS	Philadelphia VA Medical Center, Philadelphia, PA	1: Medtronic, Inc. 2: Boston Scientific Corp.; St. Jude Medical	None	4: Boston Scientific Corp.; Biotronik, Medtronic, Inc.; St. Jude Medical	4: Boston Scientific Corp.; Biotronik; Medtronic; St. Jude Medical	None	None
Michael E. Field, MD, FACC, FHRS	University of Wisconsin School of Medicine and Public Health, Madison, WI	None	None	None	None	None	None
Bulent Gorenek, MD, FACC, FESC	Eskisehir Osmangazi University, Eskisehir, Turkey	None	None	None	None	None	None

Table A2 (*continued*)

Peer Reviewer	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
Jin-Long Huang, MD, PhD	School of Medicine, National Yang-Ming University, Taichung City, Taiwan	1: Pfizer; Sanofi-Aventis; AstraZeneca; Boehringer Ingelheim	None	None	None	None	None
Julia H. Indik, MD, PhD, FACC, FAHA, FHRS	University of Arizona, Sarver Heart Center, Tucson, AZ	None	None	None	None	None	None
Carsten W. Israel, MD	Chefarzt der Klinik für Innere Medizin - Kardiologie, Diabetologie und Nephrologie des Ev. Krankenhauses Bielefeld Privatdozent der J.W. Goethe-Universität Frankfurt	0: European Society of Cardiology 1: Biotronik; Boston Scientific Corp.; Medtronic, Inc.; Sorin Group; St. Jude Medical	1: Biotronik; Boston Scientific Corp.; Medtronic, Inc.; Sorin Group; St. Jude Medical	1: Medtronic, Inc.; Sorin Group; St. Jude Medical	None	None	None
Mariell L. Jessup, MD, FACC, FAHA, FESC	University of Pennsylvania School of Medicine, Philadelphia, PA	None	None	None	None	None	None
Christophe Leclercq, MD, PhD	CHU Pontchaillou, Rennes, France	1: Biotronik; Medtronic, Inc.; Sorin Group; Boston Scientific Corp.; Bayer; Bristol Meyers Squibb 2: St. Jude Medical	None	None	None	None	None
Robert J. MacFadyen, MD, PhD	Ballarat Health and University of Melbourne, Victoria, Australia	None	None	None	None	None	None
Christopher Madias, MD, FHRS	Rush University Medical Center, Chicago, IL	None	None	None	None	None	None
Manlio F. Marquez, MD, FACC	Sociedad Mexicana de Electrofisiología y Estimulación Cardíaca (SOMEEC), Mexico City, Mexico	None	None	None	None	None	None

Table A2 (*continued*)

Peer Reviewer	Institution	Consultant/Advisory Board/Honoraria	Speakers' Bureau	Research Grant	Fellowship Support	Stock Options/ Partner	Board Mbs/Other
Brian Olshansky, MD, FACC, FAHA, FHRS	University of Iowa Carver College of Medicine, Iowa City, IA	1: Daiichi Sankyo; Boston Scientific Corp.; Medtronic, Inc.; Biotronik; BioControl; Amarin; On-X; Boehringer Ingelheim; Lundbeck	1: Daiichi Sankyo	None	None	None	None
Kristen K. Patton, MD	University of Washington, Seattle, WA	None	None	None	None	None	0: American College of Cardiology; American Heart Association
Marwan M. Refaat, MD, mMBA, FACC, FAHA, FHRS, FASE, FESC, FACP, FAAMA	American University of Beirut Faculty of Medicine and Medical Center, Beirut, Lebanon	None	None	None	None	None	None
Cynthia M. Tracy, MD, FACC, FAHA	George Washington University, Washington, DC	None	None	None	None	None	None
Gaurav A. Upadhyay, MD	University of Chicago Medicine and Biological Sciences, Chicago, IL	None	None	4: Medtronic, Inc.	None	None	None
Diego Vanegas, MD, FHRS	Electrophysiology Unit. Hospital Militar Central, Bogotá, Colombia	None	None	None	None	None	None
Paul J. Wang, MD, FHRS, CCDS	Stanford School of Medicine, Stanford, CA	1: Janssen, Medtronic, Inc.	None	3: Biosense Webster; Johnson & Johnson; St. Jude Medical; Medtronic, Inc; Boston Scientific Corp.	None	1: VytronUS	None

Number Value: 0 = $0; 1 = <$10,000; 2 = >$10,000 to <$25,000; 3 = >$25,000 to <$50,000; 4 = >$50,000 to <$100,000; 5 = > $100,000.