Optimal Implantable Cardioverter Defibrillator Programming

Bindi K. Shah, MD

Abstract: Optimal programming of implantable cardioverter defibrillators (ICDs) is essential to appropriately treat ventricular tachyarrhythmias and to avoid unnecessary and inappropriate shocks. There have been a series of large clinical trials evaluating tailored programming of ICDs. We reviewed the clinical trials evaluating ICD therapies and detection, and the consensus statement on ICD programming. In doing so, we found that prolonged ICD detection times, higher rate cutoffs, and antitachycardia pacing (ATP) programming decreases inappropriate and painful therapies in a primary prevention population. The use of supraventricular tachyarrhythmia discriminators can also decrease inappropriate shocks. Tailored ICD programming using the knowledge gained from recent ICD trials can decrease inappropriate and unnecessary ICD therapies and decrease mortality.

Key Words: ICD programming, inappropriate shocks, primary prevention ICD, reducing ICD shocks

(*Cardiology in Review* 2017;25: 30–35)

Implantable cardioverter defibrillators (ICDs) have been shown to reduce the risk of sudden cardiac death and improve survival in patients at high risk of life-threatening ventricular arrhythmias with and without heart failure (HF; primary prevention) and in patients with a history of cardiac arrest or life-threatening arrhythmia (secondary prevention).[1–3] Although the efficacy of ICD therapy has been proven in several large randomized studies, patients with ICDs may receive unnecessary shocks, which can be associated with proarrhythmia, harmful psychological effects, and increased mortality. Unnecessary shocks can result from therapy for supraventricular tachyarrhythmia (SVT) that is misclassified as ventricular, premature treatment of nonsustained ventricular tachycardia (NSVT) that would have self-terminated, and oversensing of cardiac and noncardiac signals. These inappropriate shocks can result in anxiety, depression, a decreased quality of life, unnecessary hospitalizations, and increased mortality.[4–7]

ICD manufacturers have basic settings programmed on their devices "out of the box." Using certain principles in programming ICD parameters to minimize unnecessary shocks without compromising efficacy and the time to therapy can decrease morbidity and mortality. It is the clinician's responsibility to use these principles in tailoring ICD therapies and detection to improve outcomes.

Inappropriate shocks, as described by therapy for causes other than ventricular tachyarrhythmia [ventricular tachycardia/ventricular fibrillation (VT/VF)], have been reported to occur in up to 17% of HF patients.[7] There has been considerable concern that shock delivery can negatively impact patient survival and contribute to pump failure. In the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) and the Multicenter Automated Defibrillator Implantation trial (MADIT II), inappropriate shocks increased the risk of death by more than double. Mortality rates increased after shocks, primarily due to progressive HF.[2,3] The ALTITUDE registry of 3809 ICD recipients showed that ICD shock was an independent predictor of mortality.[8] Poole et al[7] evaluated the mortality associated with both appropriate and inappropriate shocks in a primary prevention population, and found that there was increased mortality in both groups. The most common cause of death in both groups was HF.

This review will discuss how programming of antitachycardia pacing (ATP) therapy, detection of VT/VF, SVT discriminators, and shock therapy can improve morbidity and mortality of ICD recipients.

TACHYCARDIA DETECTION PROGRAMMING

In the past, the approach to ICD detection was to attempt to accurately and quickly detect VT/VF and deliver definitive therapy to terminate the life-threatening arrhythmia as quickly as possible. Devices were preprogrammed by the manufacturers to have very short detection times. Early devices that were primarily placed for secondary prevention did not store electrograms (EGMs), so there was no documentation to develop an appreciation of the number of shocks administered for SVT or NSVT. With newer devices over the past 10–15 years, and a larger proportion of primary prevention patients, we have begun to see the overwhelming number of unnecessary shocks. Large clinical trials have reported inappropriate shock rates from 12% to 21%, and nearly 30% of all shocks delivered have been reported to be inappropriate.[7,9–12]

The majority of manufacturers have devices that were preprogrammed to have very short detection times of 2–5 seconds. Recent trials have evaluated the benefits of longer detection duration to avoid inappropriate therapy. A longer detection time gives longer episodes of nonsustained tachyarrhythmia the opportunity to self-terminate before a potentially harmful and unnecessary shock.[13–18]

TRIALS OF PROLONGED DETECTION AND DELAYED THERAPY PROGRAMMING

PREPARE

The first study to show the benefits of prolonged detection duration was the cohort controlled Prevention Parameters Evaluation (PREPARE) study.[14] This study compared outcomes in a primary prevention population (n = 700) to a historical ICD cohort programmed to "conventional detection delays." The study population had biventricular and nonbiventricular ICDs, and detection rates were set to 182 bpm with the duration set for 30–40 intervals. The historical cohort had slower rate cutoffs and a shorter detection time as was traditionally programmed. Compared with this control cohort, PREPARE study patients were less likely to receive a shock (9% vs 17%, $P < 0.01$), with no difference in the rate of arrhythmic syncope between the 2 groups. Patients enrolled from a variety of centers were followed for 1 year. The authors of this study concluded that longer detection time could safely reduce shocks and other morbidities associated with ICD therapy.[14]

RELEVANT

The Role of Long Detection Window Programming in Patients with Left Ventricular Dysfunction (RELEVANT) study was

From the Division of Cardiology, Department of Medicine, Temple University Hospital, Philadelphia, PA.
Disclosure: The author has no conflicts of interest to report.
Correspondence: Bindi K. Shah, MD, Division of Cardiology, Department of Medicine, Temple University Hospital, 3401 N Broad Street, Philadelphia PA 19140. E-mail: Bindi.shah@tuhs.temple.edu.

ISSN: 1061-5377/17/2501-0030
DOI: 10.1097/CRD.0000000000000133

a prospective, controlled, multicenter, nonrandomized trial evaluating 324 primary prevention nonischemic HF patients with cardiac resynchronization therapy-defibrillator (CRT-D) devices.[15] The experimental group and control group had the same rate cutoff for fast VT (FVT) of 330 ms (182 bpm) and VF of 240 ms (250 bpm), but the experimental group had longer detection time [number of intervals to detect (NID) 30/40] compared with the control group (NID 12/16). The experimental group showed significantly better event-free survival to first therapy and the total number of shocks was less in this group. They had significantly fewer HF hospitalizations without any increase in syncope or death. Most of the episodes in the control group were treated, while in the experimental group 66% of VF episodes and 92% of FVT episodes self-terminated. There were also significantly less inappropriate detections in the experimental group (20 vs 242, $P < 0.0001$).[15]

MADIT RIT

The Multicenter Automatic Defibrillator Implantation Trial to Reduce Inappropriate Therapy trial (MADIT RIT) randomly assigned 1500 patients to 1 of 3 programming configurations.[16] The objective was to evaluate whether a high-rate cutoff or delayed therapy would decrease inappropriate therapies compared with conventional programming with a faster detection and lower rate cutoffs. The 3 groups were identified as high-rate therapy (detection ≥ 200 bpm with a 2.5-second delay to therapy), delayed therapy (60-second delay at 170–199 bpm, 12-second delay at 200–249 bpm, and 2.5-second delay for ≥250 bpm), and conventional therapy (2.5-second delay for 170–199 bpm and 1.0-second delay for ≥200 bpm). Patients were followed for an average of 1.4 years, and the study showed that higher rate therapy zone and delayed time to therapy were associated with reductions in first inappropriate therapy (hazard ratio 0.21 and 0.24, respectively) and reductions in all-cause mortality (0.45 and 0.56, respectively) compared with conventional programming. Findings were dominated by reductions in ATP. Patients in the conventional therapy group had a 29% probability of inappropriate therapy at 2.5 years, and the high-rate and delayed-therapy groups had rates of 6% during the same follow-up period. There was no significant difference in procedure-related adverse events or syncope between the groups. The authors concluded that programming of ICD therapies for ventricular tachyarrhythmias should be for ≥200 bpm or with a prolonged detection duration at ≥170 bpm.

Of note, in the MADIT RIT study the very high rate of inappropriate ATP with conventional therapy reflected frequent atrial tachyarrhythmias occurring in the 170–199 bpm range with the device failing to appropriately discriminate between SVT and VT. There was also less frequent appropriate ATP in the high-rate and delayed-therapy groups than in the conventional therapy group, which suggests that some of the ATP in the conventional therapy group was treating NSVT that would have self-terminated and would not have needed treatment at all.[16]

ADVANCE III

The Avoid Delivering Therapies for Nonsustained Arrhythmias in ICD Patients III (ADVANCE III) trial was a randomized, single blind trial that enrolled 1902 patients with ischemic and nonischemic etiologies receiving primary and secondary prevention ICDs.[17] The patients were randomized to longer detection at 30 of 40 intervals and standard detection at 18 of 24 intervals for ventricular arrhythmias. The authors were evaluating whether this programming difference would have an effect on unnecessary therapies. This study had a similar conclusion as the PREPARE trial. When looking at ATP and shock therapy, the long detection group had a lower incident rate ratio 0.63 (95% confidence interval, 0.51–0.78; $P < 0.001$) of shocks. Mortality and arrhythmic syncope rates were not statistically different. Also of note, a lower incidence of hospitalizations was seen in the long detection group. The authors concluded that a long detection setting in both primary and secondary prevention ICDs with the capability of delivering ATP during capacitor charge significantly reduced the rate of ventricular therapies delivered and inappropriate shocks compared with standard detection settings. This reinforced the findings in PREPARE and MADIT RIT in an even larger population of patients. This study also included secondary prevention patients, patients with dual chamber devices and CRT-D, and patients with atrial fibrillation, which the prior studies did not.[17]

PROVIDE

The Programming ICDs in Patients with Primary Prevention Indication to Prolong Time to First Shock (PROVIDE) study[18] was another trial with similar findings of reduced ICD therapies and reduced mortality without increasing arrhythmic syncope in a primary prevention population with higher detection rates, longer detection intervals, empiric ATP, and optimized SVT discriminators. Patients (n = 1670) were randomized to experimental and control groups and were followed for a mean of 530 days. Time-to-first-shock and 2-year shock rates were decreased, as well as all-cause mortality. Time-to-first-shock was reduced primarily in dual chamber and CRT-D devices and not in single chamber devices.[18]

These trials all differed slightly with their specific programming rate cutoffs and detection times, but with an attempt to evaluate a similar concept (Tables 1 and 2). They primarily evaluated a primary prevention population, with only ADVANCE III including secondary prevention patients. Also of importance, these trials were performed immediately after implant. Therefore, charge times were very short and it is unclear how prolongation of charge time with battery depletion would affect these findings.[19]

A meta-analysis by Tan et al[20] analyzed the 6 large trials that looked at therapy reduction programming and mortality. The combined data evaluated 7687 patients: 77% of the patients were men and 56% had a history of ischemic heart disease. Therapy reduction programming was found to be associated with a 30% relative reduction in mortality (95% confidence interval 16–41%; $P < 0.001$) in the combined data. There was no significant difference in the risk of syncope between the experimental and conventional programming groups. The meta-analysis included the above 5 trials plus the EMPIRIC trial[13] and evaluated mostly primary prevention patients, with the exception of ADVANCE III.

ANTITACHYCARDIA PACING

It also important to understand some earlier studies that evaluated the efficacy of pacing to terminated VT/VF as a painless therapy. The PAINFREE trials[21,22] were the earliest trials looking at tailored programming of ICD therapies to reduce morbidity and mortality. Previous studies had demonstrated that ATP could terminate VT < 200 bpm in a majority of cases with low rates of acceleration. The PAINFREE Rx II trial[22] was designed to evaluate the efficacy of ATP in terminating faster VT with safety and Quality of Life measures in a randomized fashion. ICD recipients (n = 634) were randomized to standardized empirical ATP or shock for VT that was between 188 and 250 bpm with a detection of 18 out of 24 intervals. After 11 months of follow-up, ATP was effective in 81% of episodes with similar rates of acceleration, episode duration, syncope, and sudden death. Quality of life was improved in both arms, but more so in the ATP arm. In their analysis, the authors found that with every 5% increase in left ventricular ejection fraction, the odds of successful ATP for FVT was 18% higher with a P value of 0.06. This was the first randomized prospective trial to show that ATP is safe and effective compared with shocks. The study showed that ATP terminated 73% of FVT episodes with a low risk of acceleration and syncope and no difference in mortality. Previous to this study, there was great concern that ATP attempts could be deleterious in accelerating ventricular tachyarrhythmias.

TABLE 1. Therapy Programmed in Individual Trials

	Experimental Group	Control Group
PREPARE[14]	VF: 250 bpm; NID 30 of 40 FVT: 182 bpm; NID 30 of 40; ATPx1 VT-167 bpm; NID 32; monitor only	VF-rate not controlled; NID 12 of 16 or 18 of 24 FVT-rate not controlled; 25% had delivery of ATP VT-rate not controlled; 29% had delivery of ATP
RELEVANT[15]	VF-250 bpm; NID 30 of 40 FVT-182 bpm; NID 30 of 40; ATPx1 VT-167 bpm; NID 32; monitor only	VF-250 bpm; NID 12 of 16 FVT-182 bpm; NID 12 of 16; ATPx1 VT-167 bpm; NID 32; monitor only
MADIT-RIT[16] High-rate group	VF-200 bpm; 2.5-s delay; ATPx1 VT-170 bpm; monitor only	VF-200 bpm; 1-s delay; ATPx1 VT-170 bpm; 2.5-s delay; ATPx1
MADIT-RIT[16] Delayed detection group	VF-250 bpm; 2.5-s delay; ATPx1 FVT-200 bpm; 12-s delay; ATPx1 VT-170 bpm; 60-s delay; ATPx1	VF-200 bpm; 1-s delay; ATPx1 VT-170 bpm; 2.5-s delay; ATPx1
ADVANCE III[17]	VF-188 bpm; NID 30 of 40; ATPx1 VT-150 bpm; NID 32; monitor only	VF-188 bpm; NID 18 of 24; ATPx1 VT-170 bpm; NID 32; monitor only
PROVIDE[18]	VF-250 bpm; NID 12 VT 2–214 bpm; NID 18; ATPx1 VT 1–181 bpm; NID 25; ATPx2	VF-214 bpm; NID 12 VT 2–181 bpm; NID 12; ATPx VT 1–150 bpm; NID 12; monitor only

ATP indicates antitachycardia pacing; FVT, fast ventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia.

TABLE 2. ICD Programming Trial: Patient Characteristics and Findings

Trial	N	Mean FU	Mean Age	Male (%)	BB (%)	LVEF (%)	CAD (%)	Primary Prevention (%)	Single Chamber (%)	Atrial Arrhythmia (%)	Inappropriate Therapy Reduction	Syncope	Mortality Reduction
PREPARE[14]	1391	1 year	66	78	79	26	64	100	24	26	Decreased		Decreased
RELEVANT[15]	324	6 months	64	76	**77**	25	0	100	0	16	Decreased		
MADIT-RIT[16]	1500	1.4 year	63	71	94	26	53	100	0	10	HR 0.21 (0.13–0.34)*	NS	HR 0.45 (0.24–0.85)*
											HR 0.24 (0.15–0.40)†	NS	HR 0.56 (0.30–1.02)†
ADVANCE III[17]	1902	1 year	65	84	81	30	60	75	29	11	IRR = 0.55 (0.36–0.85)	NS	NS
PROVIDE[18]	1670	1.5 year	64	73	89	27	62	100	60	27	HR 0.44 (0.3–0.63)	NS	HR 0.7 (0.50–0.98)

*High-rate therapy arm.
†Delayed therapy arm.
BB indicates beta blocker; CAD, coronary artery disease; FU, follow up; HR, hazard ratio; LVEF, left ventricular ejection fraction; NS, non significant.

This study showed that ATP therapy can successfully terminate these faster VTs without an increase in syncope, duration of episodes, or mortality. Painless therapy was shown to be effective and safe, with an improvement in Quality of Life. The authors of this study also remarked on their longer detection time (18/24) resulting in 34% of the episodes terminating with aborted therapy.[21]

Consensus Statement

A recent document was published by the Heart Rhythm Society, European Heart Rhythm Association, and Sociedad Lantinoamericana de Estimulacion Cardiaca y Electrofisiologia on optimal ICD programming and testing.[19] They divided their recommendations into 4 categories, including (1) bradycardia mode and rate, (2) tachycardia detection, (3) tachycardia therapy, and (4) the intraprocedural testing of defibrillation efficacy, using the recently updated standard by the American College of Cardiology/American Heart Association.[23,24] The authors specifically state that although they are putting forth recommendations, each individual's programming requires consideration of individual patient factors and continual reassessment of needs with in-office and remote interrogations and reprogramming. They also state that these recommendations are targeting the adult ICD recipient and not the pediatric patient population.

TACHYCARDIA DETECTION AND THERAPY PROGRAMMING

The data from the trials discussed above and the consensus statement support the programming of primary and secondary prevention ICD patients with tachycardia detection to last at least 6–12 seconds or for 30 intervals before delivering therapy to avoid unnecessary episodes. The consensus statement also gives the following recommendations. The slowest therapy zone should be between 185 and 200 bpm but not lower than that unless clinically indicated for a specific patient. SVT-VT discrimination algorithms should be programmed faster than 200 bpm and up to 230 bpm to reduce inappropriate therapies. Lead failure alerts should be programmed to diagnose lead failure early. More than 1 tachycardia zone can be useful to deliver tiered therapy/detection or the effective use of SVT-VT discriminators in certain zones.[19]

DISCRIMINATING SUPRAVENTRICULAR TACHYCARDIA FROM VENTRICULAR TACHYCARDIA

One of the greatest challenges in programming ICD detection is to appropriately include all sustained VT while excluding the SVTs. ICD programming relies primarily on rate cutoff as a first criteria, and then there are other programmable features to aid in further discrimination. It has been traditionally observed that patients with secondary prevention ICDs have slower VTs, and there is more overlap between rates of SVT and VT in this population. In primary prevention patients, VT rates are higher with less overlap.[25] Programming these rate cutoffs is a balance between undertreatment of slower VTs and inappropriate treatment of faster SVTs. With recent evidence that faster rate cutoffs can be equally safe as far as rates of syncope and mortality, maximizing specificity may be acceptable despite allowing some slower VTs to go undetected. In secondary prevention patients, rate cutoffs can be tailored to the patient's specific VT rates that have been observed. In primary prevention patients, recent evidence supports rate cutoffs as high as 200 bpm. The MADIT RIT trial showed that a rate cutoff of up to 200 bpm is safe.[16] The PROVIDE study, which also looked at primary prevention patients, found that the majority of inappropriate shocks occurred at rates between 181 and 213 bpm.[18] This suggests that there may be significant overlap in the rates of SVT and VT in this population, but it may not be harmful to treat above a higher rate as slower VTs may terminate or accelerate into a faster zone.

There are multiple features available from the different ICD manufacturers that aid in discrimination of SVT from VT beyond rate cutoffs. In some devices, programming certain zones over others (VT vs VF zones) or programming multiple zones will allow for a more specific use of SVT discriminators.

Single Chamber Discriminators

Onset

This feature is used primarily to distinguish sinus tachycardia from VT. The algorithm looks at the abruptness of the change in relative risk (RR) intervals with the initiation of VT compared with the gradual change that would be observed in sinus tachycardia. This algorithm is applied in slower VT zones and not VF zones, as one would not expect to see sinus tachycardia in those higher heart rate ranges. This algorithm makes its assessment during initiation and then does not reassess. The onset algorithm can misclassify sudden onset SVTs and atrial fibrillation as VT, but can also misclassify VT with some preceding ectopy as SVT. This algorithm, as well as stability and morphology, can also be used in conjunction with the other discriminators.

Stability

This is an algorithm that differentiates atrial fibrillation from VT by looking at beat-to-beat RR intervals and variability versus the stability of those intervals. This algorithm, in contrast to the onset algorithm, will continually reassess so that if a VT were to begin irregularly and then stabilize, it would reclassify the rhythm as VT. Misclassification with this discriminator occurs with atrial fibrillation that is very rapidly conducted to the ventricle, rapid SVTs, and irregular VTs.

Morphology

This single chamber discriminator is the only algorithm that does not rely on EGM intervals. It is the primary component of most single chamber ICD algorithms. It acquires a template during a normally conducted rhythm and compares the templated EGM to that during tachycardia. If the EGM morphology during tachycardia is a "match" for the template, it classifies it as SVT. It determines a match by comparing the shapes of the EGMs and using a percentage cutoff for how similar the morphologies need to be. Each manufacturer has its own approach to comparing the tachycardia EGM to the template, but each seems to have similar efficacy and failure modes. This algorithm allows the device to reclassify a potentially misclassified irregular VT or abrupt onset SVT. This algorithm can misclassify SVT with rate related aberrancy, or conversely, if the template is taken during aberrant conduction, it can misclassify SVT without aberrancy. It can also misclassify an arrhythmia when there is a truncated EGM, errors in EGM alignment, changes in EGM over time due to lead maturation, and EGM distortion due to polarization of the lead after shock delivery. Morphology discrimination is not applied after an unsuccessful shock for this reason, but if a new episode occurs after a completed one soon after a shock, misclassification can occur. The acquisition of the initial template and updating occur automatically in most ICDs, however, in CRT devices, the template must be acquired manually.

Dual Chamber Discriminators

There is significant variability in these discriminators between ICD manufacturers, however, they all use algorithms to compare atrial and ventricular EGM relationships to improve specificity. They are comparing relative timing of atrial EGMs to ventricular EGMs during the arrhythmia. Most devices will first compare the frequency of atrial EGMs to ventricular EGMs and then apply the above discriminators. For example, Boston Scientific will apply stability after atrial fibrillation is confirmed and St. Jude Medical's algorithm will apply stability only after it is confirmed that the atrial rate is faster than the ventricular rate.[26] In many dual chamber devices, onset is applied only if the atrial rate equals the ventricular rate. The most important feature of the dual chamber algorithm takes into account that 80% of VTs have a ventricular rate that is greater than the atrial rate. The algorithms to distinguish SVT are only applied in the remaining 20% of VTs that have an equal or faster atrial rate. This reduces the risk of misclassification to fewer than 20% of true VTs.[27,28] There has not been any overwhelming evidence that dual chamber discriminators provide any benefit over single chamber algorithms in reducing inappropriate shocks. Clinical trials have reported inconsistent results.[29–33] A meta-analysis by Chen et al[34] showed that there was no significant difference in mortality or inappropriate therapy rates in dual chamber versus single chamber ICDs. The authors of the expert consensus on ICD programming state clearly that SVT-VT discriminators are not an indication for implantation of dual chamber ICDs, and that even when a dual chamber ICD is implanted for other indications, dual chamber discriminators should not be programmed on immediately. Accurate sensing on the atrial lead is critical for the proper function of these dual chamber discriminators. They can fail when there are atrial lead dislodgements, oversensing of the far field R wave on the atrial lead, and undersensing from low amplitude P waves. The authors conclude that dual chamber discriminators should only be turned on when the atrial lead becomes chronic.[19]

Supraventricular Tachycardia Limit

Another programmable feature available on some ICDs is the SVT limit rate. This is independent from the VT/VF rate on some devices, but linked to zone boundaries on others. This rate limit defines the rate under which cutoffs will be applied. Above that defined cutoff, SVT discriminators are not applied and only heart rate is considered for detection and treatment. The authors of the expert consensus statement suggest that the SVT limit not exceed 230 bpm in adults without specific patient indications to avoid misclassification of hemodynamically unstable VT.[19]

T Wave Oversensing and Programming Options

Another cause of inappropriate therapies is oversensing on the ventricular lead. This can be due to lead failure, electromagnetic interference, and T wave oversensing. T wave oversensing should be noted at implant when possible and be avoided by repositioning the lead. In cases that are discovered at a later date, there are other tools that can

be applied to minimize oversensing. One can reprogram the sensitivity on the ICD lead and change the sensing bipole. There are specific manufacturer algorithms that withhold therapy for a pattern of sensing consistent with T wave oversensing.[35] This should be used with caution, as it could result in undersensing VF. All devices provide warning alerts for lead failure when it is associated with an abrupt change in lead parameters such as impedance readings. There are also algorithms to provide alerts when ventricular cycle lengths are nonphysiologically short, suggesting oversensing. When signals are sensed on the intracardiac EGM and not the shock electrode, there can be alerts, and in some cases, withholding of therapy, which again should be used with caution.

BRADYCARDIA RATE AND MODE PROGRAMMING

There is not much scientific evidence to guide pacemaker programming in the ICD patient. Most studies evaluating pacing modes were performed in a pacemaker population who are distinct from an ICD population. However, we do have some evidence that dual chamber pacing has some benefit over single chamber pacing in patients with sinus node dysfunction and atrioventricular block. There has been no significant difference in mortality shown between the single and dual chamber modes. In addition, dual chamber pacing was associated with a lower rate of atrial fibrillation and stroke.[36] These trials did not show a difference in HF with the 2 modes. The Dual Chamber and VVI Implantable Defibrillator (DAVID) trial in ICD recipients showed poorer outcomes with DDDR pacing over VVI pacing. This was likely due to increased right ventricular pacing. The follow-up DAVID II trial demonstrated that AAI pacing was noninferior to VVI pacing.[37] Dual chamber pacing reduces the risk of pacemaker syndrome and is associated with better exercise performance than VVI pacing. Given the increased cost and complications related to dual chamber devices, and with the exception of resynchronization therapy, only ICD patients who have a pacing indication or have developed pacemaker syndrome with VVI pacing should be considered for dual chamber pacing modes. In addition, without a pacing indication, pacing should generally be avoided if possible. Basic pacemaker programming should mimic what would be done for non ICD patients, keeping in mind that although we want to minimize right ventricular pacing, long nonphysiologic atrioventricular delays with AAI/AAIR pacing have been associated with worsening HF and an increased risk of atrial fibrillation (AF).[19] Individual cases of hypertrophic cardiomyopathy and long QT are exceptions and should be evaluated on a case-by-case basis. When programming CRT pacing in the HF patient, much attention should be given to producing the highest percentage of biventricular pacing. With each 1% increase in CRT after 90%, there is a significant improvement in HF and death.[38]

CONCLUSIONS

There is significant evidence that ICD shocks, both necessary and unnecessary, are associated with an increased risk of death. Although the majority of these deaths are due to HF, the populations studied were on an adequate HF regimen so the shocks may be associated with worsened HF. There is also evidence that tailored programming can reduce shocks and improve outcomes. This seems to be a finding in both ischemic and nonischemic patients, and a primary prevention patients and secondary prevention patients. Programming ICDs, using the simple framework reviewed here, will potentially decrease harmful ICD therapies and reduce mortality safely and efficaciously. Optimal programming techniques should be used by all physicians implanting and managing ICD patients.

REFERENCES

1. Zipes, DP, Wyse DG, Friedman PL, et al. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. *N Engl J Med.* 1997;337:1576–1584.
2. Moss AJ, Zareba W, Hall WJ, et al; Multicenter Automatic Defibrillator Implantation Trial II Investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med*. 2002;346:877–883.
3. Bardy GH, Lee KL, Mark DB, et al; Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *N Engl J Med*. 2005;352:225–237.
4. Sears SF Jr, Todaro JF, Lewis TS, et al. Examining the psychosocial impact of implantable cardioverter defibrillators: a literature review. *Clin Cardiol*. 1999;22:481–489.
5. Sweeney MO, Sherfesee L, DeGroot PJ, et al. Differences in effects of electrical therapy type for ventricular arrhythmias on mortality in implantable cardioverter-defibrillator patients. *Heart Rhythm*. 2010;7:353–360.
6. Schron EB, Exner DV, Yao Q, et al. Quality of life in the antiarrhythmics versus implantable defibrillators trial: impact of therapy and influence of adverse symptoms and defibrillator shocks. *Circulation*. 2002;105:589–594.
7. Poole JE, Johnson GW, Hellkamp AS, et al. Prognostic importance of defibrillator shocks in patients with heart failure. *N Engl J Med*. 2008;359:1009–1017.
8. Powell BD, Saxon LA, Boehmer JP, et al. Survival after shock therapy in implantable cardioverter-defibrillator and cardiac resynchronization therapy-defibrillator recipients according to rhythm shocked. The ALTITUDE Survival by Rhythm Study. *J Am Coll Cardiol*. 2013;62:1674–1679.
9. Powell BD, Asirvatham SJ, Perschbacher DL, et al. Noise, artifact, and oversensing related inappropriate ICD shock evaluation: ALTITUDE NOISE Study. *Pacing Clin Electrophysiol*. 2012;35:863–869.
10. Daubert JP, Zareba W, Cannom DS, et al; MADIT II Investigators. Inappropriate implantable cardioverter-defibrillator shocks in MADIT II: frequency, mechanisms, predictors, and survival impact. *J Am Coll Cardiol*. 2008;51:1357–1365.
11. Kadish A, Dyer A, Daubert JP, et al; Defibrillators in Non-Ischemic Cardiomyopathy Treatment Evaluation (DEFINITE) Investigators. Prophylactic defibrillator implantation in patients with nonischemic dilated cardiomyopathy. *N Engl J Med*. 2004;350:2151–2158.
12. Saxon LA, Hayes DL, Gilliam FR, et al. Long-term outcome after ICD and CRT implantation and influence of remote device follow-up: the ALTITUDE survival study. *Circulation*. 2010;122:2359–2367.
13. Wilkoff BL, Ousdigian KT, Sterns LD, et al; EMPIRIC Trial Investigators. A comparison of empiric to physician-tailored programming of implantable cardioverter-defibrillators: results from the prospective randomized multicenter EMPIRIC trial. *J Am Coll Cardiol*. 2006;48:330–339.
14. Wilkoff BL, Williamson BD, Stern RS, et al; PREPARE Study Investigators. Strategic programming of detection and therapy parameters in implantable cardioverter-defibrillators reduces shocks in primary prevention patients: results from the PREPARE (Primary Prevention Parameters Evaluation) study. *J Am Coll Cardiol*. 2008;52:541–550.
15. Gasparini M, Menozzi C, Proclemer A, et al. A simplified biventricular defibrillator with fixed long detection intervals reduces implantable cardioverter defibrillator (ICD) interventions and heart failure hospitalizations in patients with non-ischaemic cardiomyopathy implanted for primary prevention: the RELEVANT [Role of long dEtection window programming in patients with LEft VentriculAr dysfunction, Non-ischemic eTiology in primary prevention treated with a biventricular ICD] study. *Eur Heart J*. 2009;30:2758–2767.
16. Moss AJ, Schuger C, Beck CA, et al; MADIT-RIT Trial Investigators. Reduction in inappropriate therapy and mortality through ICD programming. *N Engl J Med*. 2012;367:2275–2283.
17. Gasparini M, Proclemer A, Klersy C, et al. Effect of long-detection interval vs standard-detection interval for implantable cardioverter-defibrillators on antitachycardia pacing and shock delivery: the ADVANCE III randomized clinical trial. *JAMA*. 2013;309:1903–1911.
18. Saeed M, Hanna I, Robotis D, et al. Programming implantable cardioverter-defibrillators in patients with primary prevention indication to prolong time to first shock: results from the PROVIDE study. *J Cardiovasc Electrophysiol*. 2014;25:52–59.
19. Wilkoff BL, Fauchier L, Stiles MK, et al. 2015 HRS/EHRA/APHRS/SOLAECE expert consensus statement on optimal implantable cardioverter-defibrillator programming and testing. *Heart Rhythm*. 2016;13:e50–e86.
20. Tan VH, Wilton SB, Kuriachan V, et al. Impact of programming strategies aimed at reducing nonessential implantable cardioverter defibrillator therapies on mortality: a systematic review and meta-analysis. *Circ Arrhythm Electrophysiol*. 2014;7:164–170.
21. Wathen MS, Sweeney MO, DeGroot PJ, et al; PainFREE Investigators. Shock reduction using antitachycardia pacing for spontaneous rapid ventricular tachycardia in patients with coronary artery disease. *Circulation*. 2001;104:796–801.

22. Wathen MS, DeGroot PJ, Sweeney MO, et al; PainFREE Rx II Investigators. Prospective randomized multicenter trial of empirical antitachycardia pacing versus shocks for spontaneous rapid ventricular tachycardia in patients with implantable cardioverter-defibrillators: pacing fast ventricular tachycardia reduces shock therapies (PainFREE Rx II) trial results. *Circulation*. 2004;110:2591–2596.
23. Jacobs AK, Anderson JL, Halperin JL, et al; ACC/AHA TASK FORCE MEMBERS. The evolution and future of ACC/AHA clinical practice guidelines: a 30-year journey: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *Circulation*. 2014;130:1208–1217.
24. Anderson JL. Evolution of the ACC/AHA Clinical Practice Guidelines in Perspective: Guiding the Guidelines. *J Am Coll Cardiol*. 2015;65:2735–2738.
25. Wilkoff BL, Hess M, Young J, et al. Differences in tachyarrhythmia detection and implantable cardioverter defibrillator therapy by primary or secondary prevention indication in cardiac resynchronization therapy patients. *J Cardiovasc Electrophysiol*. 2004;15:1002–1009.
26. Madhavan M, Friedman PA. Optimal programming of implantable cardiac-defibrillators. *Circulation*. 2013;128:659–672.
27. Wilkoff BL, Kühlkamp V, Volosin K, et al. Critical analysis of dual-chamber implantable cardioverter-defibrillator arrhythmia detection: results and technical considerations. *Circulation*. 2001;103:381–386.
28. Glikson M, Swerdlow CD, Gurevitz OT, et al. Optimal combination of discriminators for differentiating ventricular from supraventricular tachycardia by dual-chamber defibrillators. *J Cardiovasc Electrophysiol*. 2005;16:732–739.
29. Theuns DA, Klootwijk AP, Goedhart DM, et al. Prevention of inappropriate therapy in implantable cardioverter-defibrillators: results of a prospective, randomized study of tachyarrhythmia detection algorithms. *J Am Coll Cardiol*. 2004;44:2362–2367.
30. Olshansky B, Day JD, Moore S, et al. Is dual-chamber programming inferior to single-chamber programming in an implantable cardioverter-defibrillator? Results of the INTRINSIC RV (Inhibition of Unnecessary RV Pacing With AVSH in ICDs) study. *Circulation*. 2007;115:9–16.
31. Friedman PA, McClelland RL, Bamlet WR, et al. Dual-chamber versus single-chamber detection enhancements for implantable defibrillator rhythm diagnosis: the detect supraventricular tachycardia study. *Circulation*. 2006;113:2871–2879.
32. Almendral J, Arribas F, Wolpert C, et al; DATAS Steering Committee; DATAS Writing Committee; DATAS Investigators. Dual-chamber defibrillators reduce clinically significant adverse events compared with single-chamber devices: results from the DATAS (Dual chamber and Atrial Tachyarrhythmias Adverse events Study) trial. *Europace*. 2008;10:528–535.
33. Friedman PA, Slusser J, Hodge DO, et al. Prospective randomized trial of dual chamber vs. single chamber ICDs to minimize shocks in optimally programmed devices. *Heart Rhythm*. 2012;9:1583.
34. Chen BW, Liu Q, Wang X, et al. Are dual-chamber implantable cardioverter-defibrillators really better than single-chamber ones? A systematic review and meta-analysis. *J Interv Card Electrophysiol*. 2014;39:273–280.
35. Cao J, Gillberg JM, Swerdlow, CD. A fully automatic, implantable cardioverter-defibrillator algorithm to prevent inappropriate detection of ventricular tachycardia or fibrillation due to T-wave oversensing in spontaneous rhythm. *Heart Rhythm*. 2012;9:522–530.
36. Wilkoff BL, Cook JR, Epstein AE, et al; Dual Chamber and VVI Implantable Defibrillator Trial Investigators. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) Trial. *JAMA*. 2002;288:3115–3123.
37. Wilkoff BL, Kudenchuk PJ, Buxton AE, et al; DAVID II Investigators. The DAVID (Dual Chamber and VVI Implantable Defibrillator) II trial. *J Am Coll Cardiol*. 2009;53:872–880.
38. Ruwald AC, Kutyifa V, Ruwald MH, et al. The association between biventricular pacing and cardiac resynchronization therapy-defibrillator efficacy when compared with implantable cardioverter defibrillator on outcomes and reverse remodelling. *Eur Heart J*. 2015;36:440–448.