T
he implantable cardioverter-defibrillator (ICD) was devised to satisfy the unmet need for an effective, instantaneous therapy to prevent sudden cardiac death (SCD) due to ventricular fibrillation (VF) in at-risk, ambulatory patients. That therapy was a high-voltage electric shock delivered directly into the heart muscle. More than 3 decades later, shocks are still the defining operating characteristic of ICDs, and no other instantaneously effective therapy for VF exists. This elite status was clinched by large randomized clinical trials1,2 which demonstrated that ICDs improved mortality in patients with reduced left ventricular ejection fraction, regardless of pathogenesis or accompanying symptoms of heart failure (HF), by primary prevention of SCD due to ventricular tachyarrhythmia (VTA). Like bradycardia pacemakers for asystole, the ICD resides as a therapy genre of one, with no peer, and no competitor on the horizon. These sibling therapies for lethal heart rhythm disturbances will stand prominently among the greatest medical achievements of the 20th century.

The ICD is a mature technology, and neither the technique nor the tools have changed much for several decades. Despite a certain evolutionary elegance of the operating system, the ICD is still a blunt instrument. Although it is true that some innovation has occurred, it is still a matter of a shock delivered by insulated metal conductors residing somewhere in direct proximity to the heart. No innovation beyond the fundamental of a timed shock for VF has proven to enhance mortality benefit. The basic design persists simply because no one can think of a suitable alternative and the self-satisfying aphorism that “shocks save lives.”

Yet there is a growing intellectual dissatisfaction with the unintended consequences of this powerful, irreplaceable therapy. The stimulus for this self-inspection is an awareness of the very high morbidity risk overhead borne by the primary prevention patient, in particular, leveraged against very low annualized need for life-saving therapy. Consequently, strategies to reduce the morbidity penalties of ICD therapy without sacrificing mortality benefit now dominate the intellectual landscape and are the theme of this treatise.

The singularity of purpose of the ICD defines the standard by which any ancillary enhancement to the basic and essential apparatus must satisfy or risk elimination: does it increase mortality benefit? Exemptions may exist for complex features that cost-effectively reduce HF or improve quality of life (eg, resynchronization therapy), but these should not detract from the primary mission that is mortality.

A systematic approach to minimizing ICD morbidity necessarily spares no single aspect of therapy delivery and implementation. The basic architecture seeks to eliminate or minimize: (1) nonessential hardware for terminating VTA, (2) unnecessary atrial and ventricular pacing, (3) shocks by targeting (a) inappropriate therapies for supraventricular tachycardia (SVT), (b) premature therapies for VTA that are clinically inconsequential or destined to terminate spontaneously, (c) shock-free termination of non-VF VTA (antitachycardia pacing, ATP), (d) routine implant defibrillation threshold (DFT) testing. As with any integrated therapy delivery system, many of these constituents are cross-serviced by common hardware elements. This creates efficiencies such that elimination of 1 hardware element eliminates multiple inefficiencies and nonessential behaviors.

**Hardware for VTA Termination**

Current ICD systems exploit the outer housing of the pulse generator as a defibrillation electrode. This is a robust electrode design because it is simple, resides outside the vascular system, and is almost indestructible. Therefore, only 1 additional high-voltage electrode near the heart is absolutely necessary to deliver a life-saving shock. The second electrode invariably is a multicomponent design incorporating ≥1 high-voltage coils for shocks and ≥1 low-voltage electrodes for sensing, ventricular bradycardia pacing, and ATP. This typically resides within the right ventricle (RV), but subcutaneous arrangements also exist. A dual shocking coil–dedicated true bipolar pacing lead has been the most popular design for more than a decade. These complex quadripolar designs require multiple lumens and insulating layers to accommodate 4 independent conductors (2 high voltage, 2 low voltage). A variation is a single-coil, true bipolar lead arrangement with 3 independent conductors. Alternately, a minimalist design is achieved by a single pacing electrode (tip) and a single shocking electrode (coil);

![Image](http://circ.ahajournals.org)
the shocking electrode also serves as the low-voltage anode for sensing and pacing (integrated bipolar lead). The virtue of this design is that it reduces the minimum number of electric conductors from 4 to 2. This matters because the opportunity set for biomechanical stress-failure mechanisms may be related to design complexity including the number of conductors. Recent disastrous worldwide experience with downsized quadripolar leads indicates that the lower boundary for structural reliability has been exceeded given current technology.

The popularity of the complex quadripolar lead is driven by 2 enduring misperceptions. The first misperception is that dual-coil leads deliver a clinically important reduction in DFT. The fact is that DFT is low (mean 12–15 J) and not significantly different between single- and dual-coil arrangements, although dual coil leads may reduce minority outliers with higher DFTs. The second misperception is that sensing is clinically superior with true bipolar leads. The fact is that sensing is equally robust with either design. Moreover, there is no evidence that differences in DFT or sensing between simple versus complex leads influences mortality benefit. Risk of catastrophic SVC laceration during extraction is reduced by single-coil leads.

The ICD Minimalist's Approach to Hardware for VTA Termination

Lead conductors and structural complexity should be minimized by use of the simplest design to reduce the odds of failure. Single-coil integrated bipolar (or true bipolar) leads are recommended for primary prevention ICDs.

Hardware Selection for Pacing

The singular purpose of an ICD is served by a 1 ventricular lead. Perception of a need for bradycardia pacing support, improved rhythm discrimination, atrial arrhythmia surveillance, and atrial therapies motivated the development of the dual-chamber ICD. Clinical adoption was immediate, sustained, and unexplained. Dual-chamber systems account for 65% of all conventional ICD implants in the National ICD Registry for years 2006 to 2009 despite the complete lack of clinical evidence that the additional capabilities provided by the atrial lead improve mortality or quality of life, or reduce risks of inappropriate ventricular therapies, HF, or atrial arrhythmia.4,5 Because critical appraisal of the dual-chamber ICD, in comparison with the single-chamber ICD, is not favorable, the enduring use is most likely accounted for by physician bias.

Bradycardia Pacing

The vast majority of ICD-qualifying patients do not have a bradycardia pacing requirement, and later emergence of a class 1 pacing indication is uncommon. Four randomized clinical trials compared dual-chamber or atrial pacing against ventricular-only backup pacing (nominally VVI 40 bpm) in typical ICD patients.4–7 A minority (4%–14%) assigned to VVI-40 were reprogrammed to dual-chamber (or atrial-only) pacing because of sinus node dysfunction or atrioventricular block. In the largest ICD pacing mode trial, a need for pacing emerged in 5.5% (mostly sinus node dysfunction), and was 2 times higher in the VVI-40 group.5 This exceeds the 3% 5-year estimated need for pacing in Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) but is similar to the Dual Chamber and VVI Implantable Defibrillator (DAVID) trial (4%) and less than DAVID II (14%).4 These are likely biased overestimates, because all patients had dual-chamber ICDs, and crossover only required reprogramming instead of surgical hardware upgrade.

Moreover, conventional dual-chamber pacing is associated with increased risks of atrial arrhythmia, VTA, HF, and death,6,8–11 which exhibit an increasing risk relationship with cumulative percent RV pacing.8–10 and, if sufficiently intense, eliminate the mortality benefit of ICD therapy.10,11 These adverse effects are attributed to ventricular electromechanical desynchronization imposed by RV stimulation in all pacing modes, which may induce hypertrophy, negative volumetric remodeling, mitral regurgitation, and reduced pump function.12 The clinical magnitude is greatest in patients with the high-risk substrate (low ejection fraction, myocardial infarction, HF) that characterizes ICD patients9 and is reduced by minimization of RV pacing.4

Consequently, expert guidelines have emphasized minimization of RV pacing in all patient groups. This is equally achieved with simple single-lead, ventricular-only backup pacing, or newly developed complex dual-chamber pacing algorithms that prioritize atrial pacing by eliminating constraints on the atrioventricular (AV) interval.5 The assumption of these designs is that necessary or desired atrial pacing is not directly harmful, whereas RV pacing is directly adverse. A background concern is that atrial pacing is indirectly harmful because of unrestrained increases in AV time when RV pacing is eliminated.13

The Managed Ventricular Pacing (MVP) trial5 unexpectedly failed to demonstrate that nominal atrial pacing at 60 bpm by use of a new dual-chamber algorithm that severely reduces RV pacing is noninferior to single-lead VVI-40 backup pacing in ICD patients with no need for pacing. This failure was due to a 2.7 increased risk of death and HF during atrial pacing among the 10% of patients with baseline PR interval ≥230 ms, whereas outcomes were similar between pacing strategies for patients with PR <230 ms. The risk of death and HF increased ∼8% for every 10-ms increase in baseline PR interval above the mean of 184 ms. This prompts concern that increases in already prolonged PR intervals (AV desynchronization) during atrial-only pacing may be adverse in some low ejection fraction HF patients.13 An effect of higher heart rates imposed by nonobligatory pacing has not been excluded. DAVID II observed no relationship between atrial pacing at 70 bpm and risk of HF; however, patients with baseline PR >240 ms were excluded by design because of concern for worsening AV desynchronization.4 A direct comparison with Inhibition of Unnecessary RV Pacing With AVSH in ICDs (INTRINSIC RV) is not possible, because hardware limitations excluded patients with long PRs.7 No guidance regarding any of these possibilities has been provided by any study to date.

Bradycardia Pacing Induced Ventricular Proarrhythmia

Up to ∼30% of VTA in ICD patients are initiated by short-long-short ventricular sequences (pauses).14 Critically
timed delivery of ventricular pacing stimuli during normal bradycardia operation in any mode may actively facilitate short-long-short VTA (pacing-induced ventricular proarhythmia). A causal relationship between pause-terminating ventricular pacing stimuli and initiation of VTA in single-chamber ICDs has been established. The common element among all pacing modes is an abrupt change in ventricular cycle length and activation sequence that is initiated and terminated by ventricular premature depolarizations and/or ventricular premature depolarization surrogates in the form of single ventricular pacing stimuli. A comparison of pacing-facilitated VTA between modes (ventricular-only, conventional dual-chamber, and dual-chamber minimal ventricular pacing) revealed that the incidence of pacing facilitated short-long-short VTA as the sole observed VTA onset sequence was very low (≈1%) overall. However, among patients with any VTA, pacing proarrhythmia (1) accounted for 8% to 15% of all VTA, (2) was observed in 11% to 28% patients, (3) was the only VTA onset sequence in ≈4% to 10% of patients, and (4) is invoked differently but observed in all pacing modes.

The ICD Minimalist’s Approach to Management of Pacing

Most ICD-qualifying patients do not need bradycardia support and are well served with ventricular-only backup pacing in single-chamber ICD systems. There is no evidence that empirical use of any dual-chamber pacing approach improves mortality, quality of life, or reduces HF, VTA, or atrial arrhythmia. Moreover, real-world pulse generator longevity is about one-third less in dual- versus single-chamber ICDs. In the case of obligatory pacing for symptomatic sinus node dysfunction, a dual-chamber strategy for minimal pacing at all chamber levels is recommended. Without question, RV pacing should be suppressed by strategic extension of pacemaker AVIs or dedicated dual-chamber minimal ventricular pacing modes. Atrial pacing should be practiced conservatively (50–55 bpm during waking hours, 35–40 bpm during sleep), and rate response should be eliminated to minimize exaggeration of AV timing problems that might contribute to increased risk of HF. Additionally, rate-responsive pacing has not been shown to improve functional status or quality of life and is associated with reduced ICD pulse generator longevity. The management of obligatory pacing in ICD patients with baseline AV conduction delay unaccompanied by left bundle branch block is especially difficult, because both atrial and ventricular pacing can induce or exaggerate chamber-level and cross-chamber timing abnormalities that may worsen HF. There is some interest in the use of conventional biventricular pacing to correct spontaneous or atrial pacing-induced AV timing delays despite normal ventricular conduction (narrow QRS HF), but there is no clinical evidence to support this approach. Furthermore, even biventricular pacing will disrupt normal ventricular conduction that may worsen ventricular pump function forcing a trade-off, and biventricular ICDs have the shortest real-world battery longevity of any system. In case of obligatory ventricular pacing, preemptive biventricular pacing may reduce RV pacing–related remodeling, but there is little evidence that this prevents HF or improves mortality in ICD patients.

Overcoming pacing-induced proarrhythmia is difficult. Switching modes may not eliminate the problem, because it can occur in any mode. For typical ICD patients, ventricular-only backup pacing should be programmed to the lowest possible rate; some will still have pacing-facilitated VTA because of nonobligatory isolated ventricular paced beats after pauses, particularly during sleep. Elimination of nonobligatory ventricular backup pacing altogether (OVO mode) may be effective.

The recently emerged shock-only subcutaneous ICD expresses a theme of hardware minimization by eliminating transvenous leads altogether. This arrangement also eliminates the possibility of conventional bradycardia and antitachycardia pacing, although neither are essential to the primary mission of terminating life-threatening VTA.

Shock Reduction

The mortality benefit of simple ICDs is due entirely to reduction of SCD owing to VTA. Clinical trials that confirmed this causal effect consistently provide a signal of something else important happening: an immediate and accelerating risk of HF and death after registration of a VTA shock. The increased risk of death after VTA shocks (1) persists after adjustment for all other mortality predictors, (2) increases with number of VTA shocks, and (3) exceeds that of all other mortality predictors. The increased magnitude effect of VTA shocks for adjusted risk of death ranges from 2.4 to >10 times. In Multicenter Automatic Defibrillator Implantation Trial (MADIT) II and SCD-HeFT, survival after first VTA shock was ≤80% at 1 year, which was significantly less than survival before first VTA shock. Consequently, 20% to 23% of patients were dead within 1 year of their first life-saving VTA shock. In the case of the Defibrillation in Acute Myocardial Infarction Trial (DINAMIT) and Immediate Risk-Stratification Improves Survival (IRIS) trial, the SCD reduction early post–myocardial infarction was completely offset by a parallel increase in nonarrhythmic death confined to the ICD minority subgroups that recorded electric therapies (mostly shocks) for VTA. Consequently, no survival difference was observed between treatment arms.

This consistently duplicating relationship between shocked VTA episodes and increased mortality risk can be summarized as follows: After adjusting for all other mortality predictors, patients with VTA and shocks have higher mortality than otherwise similar patients with neither, and patients with more VTA and more shocks have higher mortality than patients with less of both.

The association between VTA shocks and mortality is further influenced by VTA subtype. The increased mortality risk is greatest for VF (rate ≥250 bpm) shocks, intermediate for fast ventricular tachycardia (FVT, rate 190–250 bpm) shocks, and lowest for slow VT (rate <190 bpm) shocks. Mortality increases as a dramatic step function after VF shocks. Consequently, survival time after VF shocks was much shorter than VT or FVT shocks, such that 17% of patients in MADIT II and 40% in DINAMIT were dead.
within 1 year of a VF shock. These facts imply that VTA rhythm severity influences shocked mortality risk, and the effect is greatest for VF.

The association between VTA shocks and mortality is also influenced by cardiac substrate. The risk increase after VTA shocks is \( \geq 3 \) times greater in ischemic than in nonischemic HF. Additionally, survival time after VTA shocks is inversely related to HF class. The consequence of these effects is that a mortality benefit was not recorded in some subgroups (eg, class III HF) despite VTA shocks.

Risk of HF hospitalization doubled after single VTA shocks, and the risk of multiple HF hospitalizations increased by 74% in MADIT II. Similar to the effects of shocked VTA subtype on mortality risk, the risk of HF hospitalization was greater for VF versus VT shocks. At 1 year, the probability of HF hospitalization without VTA shocks was 19% in comparison with 26% after VT shocks and 31% after VF shocks.

The association between VTA shocks and increased risk of HF and death is not debatable. The meaning of this association, however, is uncertain. A widely held view is that the emergence of VTA identifies a patient at higher mortality risk, unrelated to shocks. This argument emphasizes that a mortality benefit occurred in the moment of the VTA event (not necessarily true) which vindicates the decision to implant; the fact that some patients record no meaningful survival extension is a patient failure, not a therapy failure. In MADIT II, SCD-HeFT, and DINAMIT, the sickest patients were the most likely to have VTA therapies, generating the adage “the sickest patients benefit the most”; then again, the sickest are the most likely to die. This argument is conveniently extended to the increased risk of HF after VTA shocks, which is explained as the unnatural progression of disease granted by life-prolonging shocks. In other words, the VTA shock prolongs suffering but not necessarily survival in some patients, and neither HF events nor death are a direct consequence of the shocked VTA episode itself.

There are 2 certainties regarding the paradox of appropriate shocks and increased mortality: (1) a cause-and-effect relationship has not been established, and (2) the possibility that shock-induced cardiac electric trauma directly contributes to the increased risk of HF and death in some patients has not been excluded. The latter possibility must be considered, because shocks at therapeutic levels for defibrillation cause temporary or permanent damage to the heart, whereas weaker shocks cause less damage but do not defibrillate. There are numerous biophysical injury mechanisms of electric shock trauma; irreversible electroporation of the cell membrane is probably most important, because its structure is highly vulnerable to electric field trauma induced by clinically relevant current field intensities for defibrillation.

More than 45 years of experimental evidence has established the adverse cellular, tissue, and cardiac mechanical responses to large electric currents during sinus rhythm and defibrillation shocks in animals and humans. Acute myocardial necrosis varying directly with proximity to the shocking electrode and shock strength has been demonstrated. Shocks cause reductions in pump function and reduced oxidative metabolism proportional to shock strength. The severity of post-VF myocardial depression increases and survival time decreases with shock strength independently of preshock VF in animal models.

Randomized clinical trials to establish whether shocks cause myocardial damage have not been conducted. Observational experience shows that repetitive shocks can cause clinical deterioration. The increased mortality risk of VTA shocks is greatest in patients with more severe reduction in LV function. Death due to electromechanical dissociation may occur during implant testing or shock storms. Defibrillation-level shock energies cause release of cardiac-specific injury biomarkers that increase with size of electric trauma (shock strength, number of shocks, total joules delivered), transvenous transthoracic shocks, recent myocardial infarction, and time in VTA. Despite revascularization, implant shocks generated a 10-times higher cardiac biomarker release among patients with recent myocardial infarction.

Particularly relevant to DINAMIT and IRIS is evidence that ischemia dramatically enhances the negative effects of shocks on recovery of LV pump function after VF termination. The instantaneous emergence of a transient local injury current on the right ventricular electrogram (EGM) after an induced VF shock, not present during the immediately preceding sinus rhythm, identified patients who were at \( \approx 2.5 \) times increased risk of HF and death following spontaneous VTA shocks.

Two related observations raise concern for mortality effects of shocks. First, inappropriate shocks increase mortality risk by 2 to 4 times. This argues that shock effects persist independent of VTA, which fits with animal and human experiments. Second, shock-free termination of VTA by ATP did not increase mortality risk in MADIT II. A more illuminating exploration of the differences in effects of electric therapy type (shocks versus ATP) was provided by recent analysis of 2135 ICD patients from 4 trials that broadly incorporated ATP for VT (<190 bpm) and fast VT (FVT; 190–250 bpm). Of adjudicated VTA, 59% was VT, 37% was FVT, and 4% was VF. ATP terminated 92% of VT and 68.2% of FVT, whereas all VF received shocks. Consequently, 80% of non-VF VTA (eg, VT + FVT) was terminated with ATP, whereas 20% of all VTA (primarily VF) received shocks. Although VF mandates shocks, these were the minority contributor to overall shocked VTA episode burden. Adjusted models indicated that (1) each shocked VTA episode increased mortality risk by \( \approx 20\% \), whereas ATP-treated VTA did not; (2) mortality risk was highest in shocked patients and equivalently lowest in those without VTA or only ATP-treated VTA; and (3) VTA occurrence rates, durations, and electric therapy burden of both types were highest among patients who were shocked and died.

**Rhythm Discrimination**

Selective identification and treatment of VTA remains frustratingly problematic. Sensing of ventricular electric activity is generally robust, and rate alone would be adequate for VF, because virtually no other cardiac rhythm disturbance is capable of achieving similarly high and sustained ventricular rates. Ventricular rate-based detection alone cannot distinguish between other VTAs and SVT at coexisting rates.
Detection enhancements refine recognition of true VTA for appropriate therapy and reject SVT to avoid indiscriminate therapy. A simple waiting period (in units of time or ventricular intervals) before therapy after rate has been satisfied can reduce premature therapies for VTA and inappropriate therapies for SVT, because either may spontaneously slow or terminate. Time duration alone has safety limitations and is incapable of distinguishing between VTA and SVT, which requires rhythm discrimination.

The basis for discrimination is identifying and exploiting distinguishing characteristics of VT similar to using surface electrocardiography in which more information accurately applied generally increases the probability of a correct diagnosis. Such information includes onset and offset characteristics, atrial-ventricular timing relationships, ventricular regularity, and distinguishing features of the ventricular EGM complex (shape, notching, etc) relative to sinus rhythm. However, the availability of information for analysis in ICDs is influenced by the hardware system. The ventricular signal for sensing and detection is typically recorded from a single site (usually the RV). This is sufficient for calculating tachycardia onset and regularity, which is useful for discrimination because sinus tachycardia is usually of gradual onset, whereas VT starts suddenly, and atrial fibrillation (AF) is characterized by irregular ventricular intervals, whereas VT is typically regular.

Onset and stability alone may yield specificity as high as 96% for rejecting sinus tachycardia and AF at ventricular rates <180 to 190 bpm in single-chamber ICDs. The specificity for AF with use of interval stability alone declines significantly at higher ventricular rates (>190 bpm) because of interval regularization. In either situation, the penalty for improved specificity with use of this simple approach is reduced sensitivity for detection of VTA to 80% to 90%, which is fundamentally unacceptable. The addition of a therapy inhibitor override (high rate time-out), after which therapy is delivered regardless of rhythm discrimination if ventricular rate criteria are still satisfied, guarantees 100% detection sensitivity for true VT but erodes detection specificity and increases inappropriate therapies.

Because VT is characterized by a change in ventricular activation sequence, morphological features of the ventricular EGM could also be useful in diagnosing VT. This can be achieved with single-chamber or dual-chamber ICDs by use of various EGM signal sources. Atrial rhythm analysis and atrial-ventricular timing relationships might also enhance rhythm discrimination but usually require a dual-chamber ICD. Properly classifying SVTs with a 1:1 AV relationship is paradoxically a particularly difficult problem for dual-chamber ICD systems that analyze AV timing. These rhythms are often sudden onset (simulating VT) and regular (also mimicking VT and rendering ventricular interval stability criteria useless). The situation is even more complex when there is a varying AV timing relationship owing to discontinuous AV nodal behavior at high atrial rates, which mimics AV dissociation, a hallmark of VT. Although the use of ventricular EGM morphology would seem particularly useful in this situation, specificity may be as low as 70%. Rhythm discrimination using ventricular EGM template matching commonly fails when the template sinus rhythm EGM changes or the VT EGM is insufficiently different from the sinus template; other failure mechanisms exist.

Against a >30-year background of increasingly ornate detection enhancements, the specificity for ventricular rate–based detection still resides at ~70% to 90%. No advantage of dual-chamber versus single-chamber detection enhancements has been proven. The most recent attempt to reverse these failures reported that dual-chamber enhancements reduced overall inappropriate detections by nearly 50% in comparison with single-chamber detection. However, inappropriate shocks were not reduced, which was the point of the experiment. Equally troubling, only 93% of VTA episodes were detected; the remaining were misclassified as SVT, and, because no inhibitor override was applied by design, no therapies were delivered, which is operationally a device failure.

More information about the distinguishing characteristics of atrial versus ventricular tachyarrhythmia using dual-chamber ICDs has not been shown to meaningfully increase detection specificity versus single-chamber ICDs without jeopardizing safety. Consistently >30% to 40% of ICD patients have inappropriate therapies, regardless of the hardware arrangement. This reality is unacceptable, because every inappropriate detection risks exposing the patient to a full and repeating sequence of shocks ("unloading the clip"). The lifetime likelihood of an inappropriate therapy significantly exceeds the likelihood of an appropriate VTA therapy for VT in primary-prevention patients.

An argument for dual-chamber ICDs solely to suppress inappropriate shocks cannot be made based on published evidence; this practice should be eliminated. In contrast, strategic detection programming dramatically reduces the odds of appropriate and inappropriate shocks in typical primary-prevention ICD patients without compromising safety and requires only a ventricular lead. This strategy has 4 constituents: (1) clinically consequential slow sustained VT (<180/min) is rare in primary-prevention patients; (2) at least one-third of PFTs (rates 180–250 minutes) terminate spontaneously during capacitor charging (eg, 10–15 seconds); (3) ≥70% to 80% of all VTA in the VF zone are actually FVT that can be terminated with ATP; and (4) inappropriate SVT detections are registered in the VF zone in ≥20% of patients. In most ICD systems, all ventricular detections >180 to 190 bpm are classified monotonically as VF, detection enhancements are not applied, and all episodes are shocked. This historical practice should be abolished because ~60% to 70% of all VTA in the VF zone can be terminated without shocks, and extension of detection enhancements into the VF zone could further reduce inappropriate detections.

Therefore, the 2 most powerful discriminators, rate and time, can be leveraged to suppress inappropriate shocks for VT or nonsustained fast VT. A high detection rate (>180–200 bpm) increases the odds of detecting only clinically consequential rapid VTA; a longer detection delay (in units of time or ventricular intervals) reduces premature therapies for rapid, self-terminating VTA and for rapidly conducted SVT with transient rate excursions into the detection zone. Aggressive use of ATP terminates ~60% to 70% of all
FVT; consequently, shocks are limited to true VF (≈5% of all VTA in primary-prevention patients) and ATP failures (see Shock Reduction). ATP may also prevent shocks for SVT by multiple mechanisms (termination of AV nodal-dependent SVTs, delaying shocks long enough to permit spontaneous slowing or termination of SVT and slowing of SVTs by concealed retrograde penetration of the AV node).

Preliminary effectiveness of this evidence-based, simplified approach to detection and therapy programming for reducing all-cause shocks in primary-prevention patients has been provided.43 Elimination of a slow VT zone (<180 bpm), longer detection time in the FVT/VF zone, and ATP before shock for FVT resulted in a 50% reduction in all shocks, appropriate shocks, and inappropriate shocks in comparison with control patients with traditional programming, despite no increase in untreated VT or syncope, and no signal of increased mortality.43 This effect was registered across all hardware systems (single chamber, dual chamber, biventricular). Greater gains in shock reduction likely could have been obtained had detection enhancements with no time-out override been applied to rates >200 bpm. Many of these issues will be clarified by the ongoing Multicenter Automatic Defibrillator Trial-Reduce Inappropriate Therapy (MADIT-RIT).

The Minimalist’s Approach to Rhythm Discrimination (see also Shock Reduction)

Single-zone VTA detection programming is recommended for primary-prevention patients. A detection rate of 180 to 200 bpm has proven to be safe in clinical trials.2,43 Detection delays of 10 to 20 seconds, and perhaps longer in patients with AF or arrhythmia substrate prone to rapid, self-terminating VTA (eg, long QT) are contemplated. All episodes that satisfy detection should be screened with detection enhancements; therapy inhibitor overrides should be deactivated or extended to several minutes to maximally reduce inappropriate therapies. ATP should be applied to all detections before shock. Computer modeling demonstrated that application of such strategies to SCD-HeFT hypothetically reduced the number of shocked VTA episodes by 59% and shocked non-VTA episodes by 82%.44 It should be noted, however, that the obligatory use of ATP has not been validated in large clinical trials, whereas the mortality sparing effects of shocks have.

A second monitor-only zone (150–190 bpm) is highly useful for ambient arrhythmia surveillance. AF surveillance can be reliably achieved in single-chamber ICDS by use of far-field EGM analysis. A rapid, irregular rhythm with an EGM template match to sinus rhythm is almost certainly AF. This provides sufficient information for assessing burden, rate control, and response to therapy without an atrial lead. Infrequently, FVT or VF is initiated by a slow VT that is captured by a monitor-only zone and re-targeted for therapy.

Whether detection and therapy programming should be modified after the first episode of rapid VTA sufficient to result in therapy is recorded is uncertain. The emergence of sustained VTA does not alter the future risk of inappropriate therapies; therefore, the logic applied to detection programming from this perspective is unchanged. However, near-syncope or syncope before termination of rapid VTA would probably invite consideration for reducing the time to therapy by shortening the intentional detection delay. Similarly, the discovery of sustained VTA in the slower monitor-only zone should prompt consideration of tiered detection and therapy programming. Both of these modifications will force a difficult trade-off against increased risk for inappropriate therapies.

The Minimalist’s Approach to Shock Reduction

The distribution of VTA type and electric therapy applied is consistent across contemporary ICD trials that excluded immediate post–myocardial infarction patients.2,42,43 The substantial majority of VTA is either VT or FVT, whereas VF is the minority. ATP is highly effective in reducing shocked-episode burden, such that ~70% of all non-VF VTA episodes can be terminated without shocks. Consequently, the majority of appropriate shocks should be reserved for true VF and ATP-unresponsive FVT and VT. This means that the vast majority of non-VF VTA should be targeted for shock-free termination. Strategies to minimize VTA shocks with ATP and shorten shocked-episode durations without sacrificing ATP should be used in all patients.

Implant Defibrillation Threshold Testing

No topic incites more emotive debate among electrophysiologists than implant DFT testing, despite the absolute lack of clinical evidence that routine use improves mortality or any other meaningful outcome in ICD patients. The practice of implant testing originated as a consequence of 2 pragmatic realities in the early era of ICD therapy: (1) literal doubt whether the equipment would work at all levels of execution, and (2) treatment of an extremely high-risk group of cardiac arrest survivors in whom the best available drug therapy had failed.

This practice endures as a habit of history, even though vast improvements in the equipment have virtually eliminated these concerns and cardiac arrest survivors are the substantially minority of the implant population. The out of the box DFT with current systems is 10 to 15 J (>50% safety margin for a typical 35-J device). The modern fact is that ICD systems are remarkably robust at saving lives by terminating VTA, regardless of whether implant testing is performed.

Why does the practice of implant testing persist? One commonly cited reason is that guidelines state implant testing is mandatory. In reality, neither evidence-based expert guidelines nor the Center for Medicare and Medicaid Services offers an official recommendation for or against DFT testing, because there is no published evidence that implant testing improves survival, nor evidence that lack of implant testing degrades survival. Moreover, most shocks are delivered for VT and FVT, and the cardioversion energy requirement for VT and FVT is always less than the DFT. This explains why patients in whom DFT exceeds maximum energy output (eg, cannot defibrillate) still have a survival benefit with an ICD in comparison with otherwise similar patients who leave the hospital without an ICD because of inability to defibrillate.45

Another commonly held reason for implant testing is that some patients have high DFTs and that identification of such...

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patients for system revision and restoration of an adequate DFT is important to guarantee a survival benefit. This is the black swan argument, where the search for the rare patient who would otherwise elude observation is a justification for continuing a practice with little or no evidentiary value. A single-center analysis indicated that a 10-J implant safety margin was not met in 6% patients. Long-term follow-up indicated no measurable difference in mortality between patients with low DFT, high DFT corrected by system revision, high DFT uncorrected by system revision, or those with no DFT testing whatsoever.

An integral aspect of this dilemma is that even if implant testing was proven to enhance mortality benefit, there is no expert consensus for a DFT testing standard. Likewise, there is no published evidence that achieving a DFT margin of any value improves survival, or that any arbitrarily lower safety margin worsens survival. The best evidence against any added mortality benefit from implant testing is provided by a randomized evaluation of 1997 ICD patients showed that wireless remote monitoring with automatic clinician alerts, in comparison with standard in-office evaluation, reduced median time from clinical event to decision from 22 days in the in-office arm to 4.6 days in the remote arm. The importance of collaborative care with a HF specialist cannot be overstated, because typical primary-prevention patients face a relentlessly competing threat to survival from HF. This is particularly essential to overcome the troubling complacency that widely exists in response to shocks. Many clinicians casually view appropriate shocks as vindication to implant the ICD. However, the immediate and accelerating risk of HF and death after registration of a VTA shock indicates impending doom in some patients. A VTA shock should sponsor a thorough evaluation for treatable causes of cardiac decompensation. There is no doubt that residual ischemia influences the mortality trajectory of ICD patients. Particular attention to coronary revascularization is critical, because lack of adequate revascularization may actively counterbalance ICD mortality benefit in a negative direction, whereas robust revascularization may passively neutralize mortality benefit by suppressing cardiac arrest.

The Minimalist’s Approach to Implant Defibrillation Threshold Testing

There is no argument of logic or data to support implant DFT testing in the modern era. A few patients will die during nonobligatory implant testing. The possibility that implant shocks increase mortality risk, similar to spontaneous appropriate and inappropriate shocks, has not been excluded. The only published analysis of the effect of implant shocks on mortality was fatally flawed because all patients had implant shocks. Implant DFT testing should be eliminated in contemporary primary-prevention patients. Many implanters favor implant DFT testing in secondary-prevention patients and other situations generally associated with higher DFTs (eg, right-sided implants, amiodarone exposure, etc). Maximum output shocks are recommended for high first-shock efficacy to shorten VTA episode duration, particularly after failed ATP. Many of these issues will be addressed by the ongoing Shockless Implant Evaluation (SIMPLE) trial.

Patient Follow-Up

The relative ease of a successful simple ICD implant does not mean that no other skills are necessary to maximize mortality benefit. Simply managing to implant the hardware is now the easiest implementation aspect of ICD therapy; patient selection, strategic programming, interpretation of spontaneous events and therapies, and system troubleshooting require significant training and experience.

Periodic assessment of system performance is routine; however, the format and frequency of such assessments is not standardized. The primary purpose of such evaluations is to establish structural integrity with the goal of anticipating system failures before patient consequence. A parallel purpose is to review ambient arrhythmia and therapies to inform programming instructions. Until recently, such assessments were conducted in real-time clinic visits. Although the human encounter is indispensable, this approach is inherently inefficient. Contemporary ICDs have enhanced early warning notification for all aspects of system performance (ambient arrhythmia, delivered therapies, lead integrity, power source and capacitor status, automatically determined sensing and stimulation thresholds). Such information is ideally placed in hierarchical storage and automatically reported at regular intervals, irrespective of a scheduled clinic visit. Critical events would be electronically flagged and reported immediately. For these reasons, the emergence of remote monitoring has greatly enhanced ICD follow-up. A recent prospective, randomized evaluation of 1997 ICD patients showed that wireless remote monitoring with automatic clinician alerts, in comparison with standard in-office evaluation, reduced median time from clinical event to decision from 22 days in the in-office arm to 4.6 days in the remote arm.

Disclosures

Dr Sweeney is a paid consultant to, and has received honoraria for speaking from, Medtronic, Inc, a manufacturer of implantable electric devices for cardiac rhythm management and heart failure therapy, in compliance with the relevant policies and guidelines of the Brigham and Women’s Hospital, Inc. Dr Sweeney and the Brigham and Women’s Hospital, Inc. own, and have received royalties for, intellectual property including US patents in the area of electric device therapy for cardiac pacing, defibrillation, cardiac resynchronization therapy, and heart failure therapy.


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