Current Status of Antitachycardia Devices

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Management of hemodynamically significant supraventricular and ventricular tachyarrhythmias is often a difficult problem for the practicing cardiologist. Not only is there considerable controversy regarding the appropriate technique to guide the selection of therapy (i.e., noninvasive versus invasive techniques), but the available therapeutic options continue to expand. Antitachycardia therapy now includes not only pharmacological agents but also ablative techniques (surgical or catheter) and implanted devices. This is the latter antitachycardia treatment modality that forms the basis for this review.

Much of the impetus for the development of effective antitachycardia device therapy has risen from the limitations of the other therapeutic options available. Pharmacological antiarrhythmic therapy, whether guided by empirical trial or invasive electrophysiological testing, identifies an agent capable of suppressing tachycardia occurrence in a minority of patients. Of considerable concern in patients with life-threatening arrhythmias is the occurrence of arrhythmia recurrence despite predicted success during rigorous testing before discharge.1–5 The problem of serious proarrhythmic effects of antiarrhythmic drugs, most recently raised by the CAST trial,6 also may limit this option in addition to less serious but frequent side effects. For sustained ventricular tachyarrhythmias, amiodarone has been perceived as the most efficacious pharmacological option, but its use has been limited by its significant side-effect profile and the 30–40% recurrence rate in 1–2-year follow-up studies, which cannot be predicted.7–10 Although catheter ablation techniques are promising for patients with circus movement tachycardias, particularly those using posteroseptal bypass tracts11 and dual atrioventricular (AV) nodal pathways,12 they carry some risk and appear to be limited in usefulness for ventricular tachycardia (VT).13 Similarly, surgical ablative techniques have become accepted therapy in patients with supraventricular tachycardia (SVT) using accessory AV connections and in patients with uniform VT; however, the widespread application in the latter group of patients has been limited by an operative mortality of 9–17%14–17 and the detailed mapping required for surgical therapy.

This review will focus on reported experience with implanted antitachycardia devices as primary or adjunctive therapy of supraventricular and ventricular tachyarrhythmias. Experience with antitachycardia pacing and internal defibrillation and cardioversion will be discussed as will future directions for device therapy.

Antitachycardia Pacing

Pacing for Tachycardia Prevention

Although pacing therapy has been used primarily as a means of tachycardia termination, it has also played a limited role in tachycardia prevention. Although overdrive pacing has been shown to decrease spontaneous ventricular ectopy18–19 when used as a primary antiarrhythmic modality in a group of patients with malignant ventricular arrhythmias, the results were disappointing.18 Overdrive pacing has also been used for long-term suppression of torsades de pointes in patients with congenital QT interval prolongation, with favorable results in a limited number of patients.20–21 Its use in preventing bradycardia-dependent torsades de pointes associated with high-grade AV block has also been established, although this is a relatively uncommon clinical problem.

Novel approaches to tachycardia suppression by using pacing therapy have also been developed in the clinical electrophysiology laboratory. Sung et al22 used simultaneous atrial and ventricular pacing to abolish the previously reproducible induction of AV nodal reentrant tachycardia during programmed stimulation. Coumel and colleagues23 and Spurrell and Sowton24 had previously demonstrated suppression of SVT with experimental pacing systems capable of simultaneous or nearly simultaneous atrial and ventricular pacing. Although theoretically attractive as a means of rendering both limbs of the AV nodal circuit refractory, the hemodynamic disadvantages are obvious. In a similar manner, Marchlinski and coworkers25 recently demonstrated that pacing at the site of VT origin during right ventricular programmed stimulation prevented the reproducible induction of VT nearly 50% of the time. The precise mechanism underlying this observation is unclear. Additionally, the frequent finding of multiple VT
morphologies in individual patients and the inability to maintain a stimulating electrode at a left ventricular site of VT origin limits the potential clinical applicability of this technique.

Pacing for Tachycardia Termination

Physiology of pacing termination. Most clinically important tachyarrhythmias are thought to be due to a reentrant mechanism in which regions of unidirectional conduction block and slow conduction allow the establishment of a circuit-like wave of depolarization that perpetuates the tachycardia. For a paced extrastimulus to terminate a tachycardia, it must traverse the myocardium between the pacing site and tachycardia circuit and then interact with the wave of depolarization. The ability to reach the reentrant circuit is facilitated by the introduction of multiple extrastimuli that act to “peel back” intervening tissue refractoriness.

When premature paced extrastimuli reach a reentrant circuit, penetration into the circuit requires an area of excitable tissue between the head and tail of the wavefront propagating in the circuit. This is termed the “excitable gap.” At relatively long coupling intervals, paced extrastimuli may enter the excitable gap and collide in the retrograde direction with the head of the wavefront propagating in the circuit but be allowed to depolarize the circuit in an antegrade direction without terminating it. In this way, the tachycardia is advanced. This phenomenon is termed “resetting.” When this same phenomenon occurs with continuous pacing, it is termed “entrainment.” At closer extrastimulus coupling intervals, the premature impulse may collide with the propagating wavefront in both the antegrade and retrograde direction, thus extinguishing it and terminating the tachycardia (Figure 1).

The resetting response may aid in establishing the mechanism of pacing induced tachycardia termination. Most tachycardias terminated by pacing techniques are usually reset at coupling intervals or paced cycle lengths longer than those resulting in tachycardia termination.26–28 The relation between the coupling intervals of paced premature extrastimuli and the subsequent return cycle has been used to construct “resetting curves,” allowing characterization of the relative refractoriness of tissue in the excitable gap.29,30 Preliminary data from our laboratory suggests that the relative refractoriness of the tissue within the excitable gap, as determined by the slope of the increasing limb of the resetting curve, is a major determinant of the ability of paced extrastimuli to terminate sustained uniform VT.31 Additionally, the ability of extrastimuli to reset a tachycardia may be a marker for subsequent termination and may potentially play a role in antitachycardia pacing algorithms.28

Pacing modalities. A wide variety of pacing algorithms have been devised to facilitate the ability of paced beats to reach and terminate clinical reentrant tachycardias. These may range from the introduction of fixed bursts of rapid pacing at a predetermined cycle length to the introduction of extrastimuli that scan electrical diastole with increasing prematurity until the tachycardia is extinguished. Pacing modalities in which pacing bursts or premature extrastimuli are introduced as a percentage of the sensed tachycardia cycle length (i.e., adaptive modes) are thought to be superior to modalities with predetermined coupling intervals or pacing cycle lengths.32 Adaptive modes lessen the significance of spontaneous variations in tachycardia cycle length or changes in tachycardia cycle length.
cardia cycle length induced by pacing. Studies comparing individual pacing modalities are limited, but generally, techniques using multiple extrastimuli or pacing bursts are superior to those using only one or two extrastimuli for tachycardia termination.26,32–35 Although the number of extrastimuli introduced during a trial of antitachycardia pacing may be predetermined, den Dulk et al33 have suggested the possible superiority of a “universal” pacing mode during which extrastimuli are automatically added sequentially, at coupling intervals that are preset percentages of the tachycardia cycle length, until termination occurs.33

Newer pacing techniques have been investigated but await widespread clinical application. Ultrarapid trains of stimuli with frequencies up to 100 Hz have been used to obtain a single ventricular capture just after the end of ventricular refractoriness to terminate sustained uniform VT in humans.36 The efficacy of this technique appears limited, resulting in termination of approximately 50–60% of tachycardias reported. Subthreshold stimulation has been shown to inhibit depolarization of premature atrial and ventricular extrastimuli in the canine37 and human38 heart, and has also been shown to cause termination of AV reentry and sustained uniform VT in humans.39–41 This technique is limited by the requirement for application of subthreshold stimulation in very close proximity to the reentrant circuit itself.

Clinical efficacy of pacing for tachycardia termination. Most of the literature on antitachycardia pacing for termination is anecdotal, preventing accurate estimations of short- or long-term effectiveness. Fisher et al42 recently combined the results of several available reports on antitachycardia pacing for SVTs and VTs to obtain an estimate of long-term efficacy. At an average follow-up of 12–15 months, subjectively “good to excellent” results were reported in the vast majority of patients with only 3.7% of results in patients with VT termed “poor” and 3.6% of results in patients with VT deemed “poor.” Obviously, such an accumulation of anecdotal reports would lead to potentially biased reporting and underestimation of unsatisfactory outcome. More interesting is the report of Fisher and coworkers43 regarding their group’s experience with antitachycardia pacing at Montefiore Medical Center. Actuarial efficacy was estimated for patients with SVT to be 93% at 1 year, 86% at 4 years, and 68% at 8 years. In patients with tolerated uniform VT, actuarial efficacy was 78% at 1 year, 72% at 3 years, and 55% at 5 years.

Limitations and complications of antitachycardia pacemakers. Tachycardia detection and differentiation from sinus tachycardia and rhythms not amenable to pacing therapy (e.g., atrial fibrillation) is an ongoing area of concern and clinical research. Whereas patient-activated devices depended on the patient’s accurate perception that his clinical tachycardia had started, the automatic antitachycardia pacemakers depend on predetermined algorithms to accurately identify a clinical tachycardia and trigger pacing therapy. Although a high-rate detector may be sensitive, overlap between the rate of physiological and pathological tachycardias can seriously limit its specificity. Additional criteria such as suddenness of tachycardia onset, sustained high rate, and rate stability have been used in conjunction with high-rate criteria to minimize inappropriate device activation without compromising the ability to detect pathological tachycardias.44,45 As will be discussed in more detail, other investigational tools to increase the sensitivity and specificity of tachycardia detection include the use of extrastimulus testing to detect tachycardia resetting,46 electrogram analysis,47 and the use of electrogram recording at multiple cardiac sites.48

Antitachycardia pacing may inadvertently induce tachycardias or cause tolerated tachycardias to degenerate to more rapid, hemodynamically significant tachyarrhythmias. Inappropriate activation of patient-activated devices during sinus rhythm may produce the patient’s clinical arrhythmia or atrial fibrillation.49 Similarly, activation of automatic devices due to failure of the detection algorithm or oversensing of noncardiac signals (e.g., myopotentials) can inappropriately trigger pacing with potential proarrhythmic effects.50 The production of atrial fibrillation or flutter during pacing therapy for VT is common; Waldecker et al51 noted this during 8% of trials of attempted pacing termination of VT involving 30% of the patient group. In 75% of instances, the atrial fibrillation or flutter was sustained. In patients capable of rapid antegrade conduction over an accessory AV connection, this can be a potentially lethal problem.52

In patients with tolerated uniform VT, the potential proarrhythmic effects of pacing therapy have curtailed the use of this therapy as a sole antiarrhythmic modality. It is well known from experience in the clinical electrophysiology laboratory that pacing to terminate uniform VT can cause tachycardia acceleration or degeneration to ventricular fibrillation (VF).34,35 The incidence increases from approximately 1% with the use of single ventricular extrastimuli to as high as 35% with bursts of rapid ventricular pacing.34,35 This problem has not been eliminated by newer pacing techniques that had seemed promising in limiting this problem.32,33,53

Selection of Patients for Antitachycardia Pacemakers

The clinical role of permanent antibradycardia pacemakers for tachycardia prevention is limited. Rare patients presenting with congenital QT prolongation and torsade de pointes may benefit from this modality as may patients with bradycardia-dependent malignant ventricular arrhythmias. Otherwise, antibradycardia permanent pacing has little use in patients with significant clinical tachycardias.

The use of currently available antitachycardia pacemakers designed for tachycardia termination is almost exclusively limited to patients with refractory reentrant SVT in whom invasive electrophysiological
testing has demonstrated reproducible tachycardia termination with pacing techniques without proarrhythmic effects. The findings during programmed stimulation can be used to develop a “pacing prescription” to allow for selection of the appropriate antitachycardia response. The decision to proceed with pacing therapy over ablative therapy may be difficult; however, we believe that patients with reentrant SVT using an accessory AV connection should initially be considered for surgical or catheter ablative therapy. This is especially true in patients with demonstrated rapid antegrade bypass tract conduction in whom pacing therapy is contraindicated. Recent advances in treating patients with AV nodal reentrant SVT with surgical or catheter ablation techniques should further limit the potential application of antitachycardia pacing in this subgroup of patients as well.

As mentioned previously, antitachycardia pacemakers do not play a role as sole management for patients with tolerated uniform VT. The risk for proarrhythmic effects of pacing therapy in this patient population has precluded the use of automatic antitachycardia pacemakers alone; they may be used in conjunction with a separate unit capable of internal defibrillation if extensive invasive electrophysiological testing has demonstrated safe and reproducible tachycardia termination. The potential for significant interdevice interaction is considerable, however. Externally activated antitachycardia pacemakers may still play a limited role in patients with tolerated VT; however, device activation is only recommended under physician supervision in a setting with readily available external cardioversion capabilities.

Internal Cardioversion and Defibrillation

**Physiology of Cardioversion and Defibrillation**

Cardioversion of VT may be accomplished with electrical discharges delivered by extrathoracic, epicardial, or intracavitary electrodes. In that sustained VT is due to reentry in most clinical circumstances, the mechanism of tachycardia termination involves penetration of the electrical discharge into the circuit with depolarization of fully or partially excitable tissue within the reentrant circuit. Saksena et al\(^{54}\) demonstrated that low-energy discharges (<0.5 J) delivered by an intracavitary catheter captured myocardium locally with muscle-to-muscle spread to the site of VT origin, whereas higher energy discharges (>0.5 J) instantaneously captured sites distant from the catheter including the site of VT origin.\(^{54}\) Once tissue within the reentrant circuit is depolarized by the electrical discharge, termination may occur if conduction block occurs within the circuit.\(^{54,55}\) As described previously with pacing termination of reentrant tachyarrhythmias, resetting of VT can also occur if retrograde, but not antegrade, block occurs within the VT circuit. Cardioversion of VT is generally performed with electrical discharges synchronized to the QRS complex to avoid delivery of energy during the vulnerable period of the cardiac cycle.\(^{56}\) This period is a time of nonuniform recovery of excitability (corresponding to the upstroke of the T wave on the electrocardiogram) and may provide a milieu for electrical induction of other reentrant arrhythmias (e.g., VF) or reinduction of VT.\(^{56}\)

Defibrillation is performed by delivering a high-energy discharge to instantaneously depolarize a large mass of ventricular myocardium to allow reemergence of a stable, hemodynamically tolerated rhythm. Although not all cells will be excitable at the time of delivery of the defibrillating discharge, presumably enough cells would be instantaneously depolarized to reduce the mass of fibrillating tissue below that required to sustain VF. Although it has been proposed that a critical mass of ventricular myocardium must be depolarized during defibrillation to terminate VF,\(^{57}\) Chen and colleagues\(^{58}\) have shown that in canines, there is also a threshold energy that must be exceeded during successful defibrillation to avoid reinitiation of VF in partially refractory tissue. Energy below this threshold could reinitiate VF in these tissues, leading to inability to terminate VF.

**Implantable Automatic Cardioverter/Defibrillator Therapy**

In 1970, Mirowski and colleagues\(^{59}\) and Schuder and coworkers\(^{60}\) independently reported on the first animal experiments in which internal defibrillation was attempted with an implanted device. After another decade of clinical research, Mirowski et al\(^{61}\) reported on the first three patients to receive the prototype automatic implantable defibrillator for treatment of sudden cardiac death due to VF. After further studies documenting the effectiveness and safety of the device as well as technical refinements allowing detection and synchronized cardioversion of VT, an automatic implantable antitachycardia device capable of cardioversion and defibrillation was approved for clinical use in the United States in 1985. In 1990, there is one FDA-approved series of automatic implantable cardioverter/defibrillators (AICDs) (Cardiac Pacemakers, St. Paul, Minnesota) available for clinical use. The currently available device consists of a hermetically sealed unit containing a lithium silver vanadium pentoxide battery, capacitors, and circuitry. The device is large (240–250 g) and is implanted in the abdominal wall where it is connected to a lead system that allows for sensing of tachycardia onset and serves as a conduit for delivery of energy for tachycardia termination (Figure 2). Rate sensing for tachycardia detection is generally accomplished by a bipolar screw-in ventricular epicardial lead or, less commonly, by an endocardial transvenous right ventricular lead. Energy for cardioversion/defibrillation is delivered by one of two separate lead systems, a superior vena cava spring electrode–epicardial patch electrode combination or a two epicardial patch electrode system. In
Figure 2. A representation of the components of the current automatic implantable cardioverter defibrillator (AICD) system and their location. The generator unit is placed in an abdominal pocket, usually located in the left upper quadrant. Rate sensing leads are usually screwed in epicardially and tunneled to the abdominal unit. Defibrillation and cardioversion is most commonly achieved by two epicardial patch electrodes placed through a thoracotomy. They may be placed in the pericardial fat pads and the attached leads are then tunneled to the device in the abdominal wall. (Reprinted with permission from JAMA 1989;262:1363, American Medical Association.)

1990, the two epicardial patch electrode configuration is used much more commonly due to its greater efficacy in achieving cardioversion and defibrillation with lower energy requirements.\(^{65}\) The energy delivery lead system is also used for an additional sensing algorithm termed "the probability density function;" this algorithm detects the amount of time the recorded electrogram is off the electrocardiographic baseline and is used to enhance the specificity of tachycardia detection by aiding in the differentiation of narrow and wide complex tachycardias. It was the sole means of VF detection in the original automatic internal defibrillator.

The currently available device requires a thoracotomy for implantation of the epicardial lead system. Median sternotomy, left anterior, subxiphoid, and subcostal approaches have all been used with general success, although the subxiphoid and subcostal approaches provide minimal visualization of the heart and are less practical in patients with prior cardiac or thoracic surgical procedures.

Tachycardia detection was a significant problem with the early implantable devices due to the fact that the rate thresholds were fixed and nonprogrammable. As a result, changes in the patient's VT rate during subsequent follow-up could compromise tachycardia detection. This has been addressed by the latest FDA-approved model of the AICD, which incorporates programmability of the rate cutoff for tachycardia detection. Additionally, the option to add the probability density function to the sensing algorithm is also programmable and can be used in patients in whom rate sensing alone may lack specificity (e.g., the patient with atrial fibrillation and malignant ventricular arrhythmias).

The currently available AICD also is somewhat limited by its energy delivery function. Nonsustained VT could satisfy the device's detection criteria but terminate before energy delivery, leading to device discharge in sinus rhythm. The currently approved AICD model and its predecessors incorporated a tachycardia detection window of 2.5 seconds after the detection algorithm was satisfied to confirm continuation of VT or VF and to lessen the chance of inappropriate device discharge. An investigational model, awaiting FDA approval at this writing, has a programmable tachycardia detection window (2.5–10 seconds) to prevent inappropriate device discharge in patients with longer episodes of nonsustained VT. The incorporation of algorithms that confirm the presence of continued tachycardia during capacitor charging to prevent inappropriate device discharge after tachycardia cessation is currently being evaluated in a number of investigational devices.

The amount of energy delivered per discharge was also originally predetermined at the time of manufacture in the earlier internal cardioverter/defibrillators. Generally, energies between 28 and 37 J were available in early models of the AICD, with the devices capable of recycling to deliver up to four discharges. This lack of flexibility did not address the fact that some VTs can be cardioverted with very low energy, whereas other patients require higher energy for reliable defibrillation. The currently available device provides limited first-shock flexibility (26 or 30 J), whereas newer investigational devices will attack this problem with extensive shock output programmability (0.1–40 J). Such programmability can overcome variations in individual patient energy requirements for tachycardia termination.

Clinical Efficacy of the Automatic Implantable Cardioverter Defibrillator

Several studies have examined the impact of the AICD on the incidence of sudden cardiac death in patients who have had the device implanted. The reported incidence of sudden cardiac death at 1 year of follow-up in patients with the AICD is 1–4.6%\(^{65-68}\); whereas at 5 years of follow-up, it is 4–20%\(^{66-68}\). This compares with historical control groups of patients in whom the incidence of recurrent sudden death in cardiac arrest survivors is approximately 20–30% at 1–2-year follow-up.\(^{69-70}\) When actual sudden deaths are combined with the number of patients experienc-
ing an AICD discharge to arrive at a “total” sudden death incidence, Mercando et al.\(^6\) reported an actuarial sudden cardiac death-free survival at 1 year of 56% and, at 4 years, of 14% in their AICD recipients. Although the occurrence of inappropriate device discharges must be taken into consideration, the incidence of patients in the overall AICD experience who have had at least one AICD discharge for presumed VT of VF is 34%.\(^6\) In view of these statistics, some have called for the AICD as a first-line therapy of survivors of sudden cardiac death.\(^7\)

Alternatively, it could be argued that a device’s nonusage rate of 66% might indicate overuse of the device in patients with a relatively good survival (e.g., inducible polymorphic VT) with artificial enhancement of recent survival statistics.

**Limitations and Complications of Implantable Cardioverter/Defibrillator Therapy**

Although the mortality associated with AICD implantation is relatively low (1–4% in most series),\(^6\)–\(^6\) it is significant. A number of periprocedural complications have also been noted. The major problem encountered has been infection, with an incidence of 1–20%.\(^6\)–\(^6\) Troup and coworkers\(^7\) described the insidious nature of AICD system infections; there was a latency period of 8.5 months between implantation and clinical presentation with fever and leukocytosis frequently being absent. Generally, conservative therapy with antibiotics is unsuccessful and explantation of the system is necessary. Other less commonly described periprocedural complications include perioperative myocardial infarction, coronary artery laceration, venous thrombosis, pneumonia, large pericardial and pleural effusions, cerebrovascular accidents, and late constrictive pericarditis.\(^6\)–\(^6\)\(^6\),\(^6\)\(^6\)\(^6\),\(^6\)\(^7\)\(^8\)\(^8\)\(^8\) Migration of the superior vena cava electrode was also reported in earlier patient series in which this electrode was used more commonly.\(^6\)\(^3\)\(^6\)\(^3\)\(^6\)

Several clinical complications with the AICD have arisen from design limitations in previous and current models of the device. The current detection algorithm is relatively rudimentary; rate threshold does not differentiate VT from SVTs such as atrial fibrillation. The probability density function can sometimes be satisfied by SVT with wide QRS complexes or, conversely, may be insensitive to relatively narrow or “spikey” VT. As a result, the device can discharge during tachycardias other than VT. As mentioned previously, nonsustained ventricular tachyarrhythmias can also trigger device discharge due to the “committed” nature of the current AICD. Due to these problems, questionably appropriate or spurious device discharges occur commonly with an incidence of 8–41% in different series.\(^6\)\(^4\)\(^5\),\(^6\)\(^7\)\(^8\),\(^9\)

The lack of telemetry in the current generation of the AICD makes determination of the appropriateness of a discharge in a conscious patient very difficult. Interactions with antibradycardia pacemakers have largely

![Diagram of AICD](image-url)
arisen from sensing difficulties. Unipolar pacemaker spikes as well as paced complexes can be sensed by the AICD, leading to double counting and satisfaction of rate criteria for device discharge. In some instances, continued delivery of pacing stimuli during VT or VF can prevent AICD recognition of the underlying ventricular rhythm due to sensing of the pacing spikes instead of the ventricular electrograms. This occurs due to normal function of the device’s automatic gain control.

A number of potential interactions between antiarrhythmic medications and the AICD may occur. In patients with early models, the rate threshold for tachycardia detection was fixed. Therefore, nonsensing of VT could occur if antiarrhythmic therapy were initiated after device implantation and tachycardia slowing below the rate cutoff resulted. Pharmacological therapy has also been shown to have varied effects on the energy requirements for successful internal cardioversion/defibrillation, with several agents increasing this requirement. As a result, repeat electrophysiological testing with tachycardia induction and confirmation of successful device function is recommended after any alteration in pharmacological antiarrhythmic therapy.

Selection of Patients for Implantable Cardioverter/Defibrillator Implantation

Once the AICD was approved for clinical use, the FDA recognized two groups of patients who were candidates for AICD therapy. They were 1) patients who had survived an episode of cardiac arrest due to malignant ventricular arrhythmias without concomitant myocardial infarction and 2) patients with recurrent ventricular tachyarrhythmias in whom hemodynamically significant sustained VT or VF was induced during electrophysiological study. The Health Care Financing Administration, however, established Medicare reimbursement guidelines that allowed AICD implantation only for patients with inducible VT or VF and only after all other options had been considered. Although in our practice to perform electropharmacological testing in cardiac arrest survivors with inducible VT before using the implantable cardioverter defibrillator, we believe these guidelines pose a problem in the cardiac arrest survivor who has no identifiable cause for his episode and has no inducible ventricular tachyarrhythmias at electrophysiological study. Although initially the prognosis in this group of patients was thought to be favorable, longer follow-up has shown the incidence of recurrent sudden death to be 10–30%. In view of this, we use the AICD as primary therapy in this class of patients with concomitant myocardial revascularization when appropriate.

The AICD has a limited role in the patient who presents with sustained tolerated VT. Although electropharmacological testing yields a clinically effective drug in a minority of patients and amiodarone therapy is frequently limited by inefficacy or toxicity, exposing conscious patients to the often painful discharge of the AICD is avoided if possible. If pharmacological therapy is deemed a clinical failure, it is our practice to next consider ablative surgical or catheter techniques in suitable candidates with tolerated sustained VT before turning to the AICD. Patients with clinical sustained tolerated VT but with multiple morphologies of inducible VT, some of which are not hemodynamically tolerated, may be considered for AICD therapy with the understanding that the induced VT may not have clinical relevance and that device discharge could occur in the conscious state.

Presently, the role of AICD implantation in patients with clinical nonsustained VT who have inducible sustained VT that is refractory to electropharmacological suppression is not established. Clearly, a small but definite percentage of these patients may benefit by AICD therapy. Further studies to stratify these patients into high- and low-risk groups for subsequent cardiac arrest or clinical sustained VT are necessary. Although Buxton and colleagues demonstrated that patients with nonsustained VT who had coronary artery disease, a low left ventricular ejection fraction (<40%), and inducible sustained VT at the time of electrophysiological study were at high risk for subsequent sudden cardiac death, this was a relatively small study. Larger studies must be done before widespread application of implantable device therapy to even a high-risk group of patients with nonsustained VT should be done. Until then, it is premature if not irresponsible to broadly apply such an approach.

Future Directions in Antitachycardia Device Therapy

Nonthoracotomy Systems

The original internal defibrillator described by Mirowski et al. in 1970 used a transvenous right ventricular electrode and chest wall electrode to deliver energy. As noted, however, the device that was eventually developed for use in humans required some form of thoracotomy for placement of a minimum of one epicardial patch electrode. Because much of the perioperative risk associated with AICD implantation stems from the performance of a thoracotomy in patients with significant left ventricular dysfunction, research efforts have focused on nonthoracotomy approaches to cardioversion and defibrillation.

Low-energy cardioversion of VT using a right ventricular catheter was demonstrated to be feasible in humans by several investigators. Ciccone and coworkers described a 62% success rate for low-energy catheter cardioversion of sustained VT. Success correlated with longer VT cycle length, higher energy per shock, and a shorter time from tachycardia onset to energy delivery. An 8% tachycardia acceleration rate was reported. Kallok et al. described an 84% efficacy rate for transvenous cardioversion of VT with a 5% incidence of acceleration. This led to the development of a prototype implant-
able transvenous low-energy cardioverter described in clinical trials by Zipes and colleagues. Although the device was implanted in patients with a mean cardioversion threshold of 0.43 J, the risk of VT acceleration or degeneration to VF limited its clinical usefulness in the absence of backup defibrillation capabilities. Moreover, low-energy cardioversion of VT may result in atrial fibrillation, which may be associated with ventricular rates in excess of the rate threshold of the implanted device. Transvenous catheter defibrillation by using an 11F tripolar catheter and high-energy shocks was efficacious in only 45% of patients tested intraoperatively by Winkle et al. Therefore, catheter-only systems were largely abandoned.

Newer systems that utilize a nonthoracotomy system incorporating a transvenous catheter and chest wall electrode for cardioversion and defibrillation have been developed and tested clinically. Saksena and Parsonnet reported the first implantation of such a device in 1988, with a subsequent report from Saksena and coworkers, detailing a total of nine patients with implanted systems. A 10F tripolar catheter was used, which incorporated a right ventricle sensing electrode as well as right atrial and right ventricular defibrillating electrodes. A third defibrillating electrode was placed submuscularly on the left thorax (Figure 3). Defibrillation was accomplished with *bidirectional* energy delivery (right ventricle to right atrium and right ventricle to chest wall patch) with two separate vectors of energy delivered simultaneously. The concept of bidirectional energy delivery for cardioversion/defibrillation and its efficacy by using a similar electrode configuration had previously been demonstrated by Lindsey et al. In the clinical series reported by Saksena and coworkers, this nonthoracotomy, bidirectional trielectrode system produced first-shock defibrillation efficacy intraoperatively in all patients, with four patients subsequently experiencing appropriate device discharge as outpatients. Although clinical trials with a nonthoracotomy system are currently suspended due to problems with the endocardial lead, this promises to be an area of active clinical investigation in the immediate future.

**New Modalities of Energy Delivery**

Attempts have been made to assess the efficacy of different modalities of energy delivery to decrease the energy requirement of cardioversion/defibrillation. As mentioned previously, bidirectional energy delivery has been demonstrated to be efficacious in performing internal defibrillation and has made thoracotomy systems feasible. Presumably, more myocardium is depolarized in this manner than with monodirectional energy delivery using such a system.

The configuration of the energy waveform also has been shown to affect the ability to successfully perform internal defibrillation. The currently available device uses a monophasic, truncated exponential waveform with a fixed tilt of approximately 60°. Several investigators have reported on the superior defibrillating ability of biphasic shocks compared with the monophasic pulses currently used.

Chapman et al reported that the configuration of the biphasic pulse itself was also crucial for lowering defibrillation threshold with a greater efficacy occurring with a longer initial positive phase relative to the negative phase.

The efficacy of defibrillation using sequential multiple pulses rather than a single shock or simultaneous bidirectional shocks has also been extensively investigated. These systems have used both multiple epicardial patch electrodes (up to four) or combinations of epicardial patch electrodes and transvenous catheter electrodes to accomplish defibrillation. Generally, lower defibrillation thresholds have been found with sequential pulse delivery systems using three or four electrodes than with single-pulse delivery systems using two electrodes. Kallo and coworkers showed that in dogs, a three-patch sequential system produced defibrillation thresholds superior to a three-patch simultaneous system with bidirectional energy delivery. Further work is necessary to define the energy delivery system that yields the lowest defibrillation threshold.

**Improved Tachycardia Detection Algorithms**

The inadequacy of a simple rate threshold for tachycardia detection has been previously discussed. Although adequate sensitivity can be achieved, specificity is severely limited. Specificity has been improved with a combination of criteria such as high rate, sudden onset of tachycardia, rate stability, and sustained high rate without impairing sensitivity for tachycardia detection.

Newer criteria for pathological tachycardia detection have been proposed. The introduction of atrial extrastimuli during tachycardia has been advocated as a means of differentiating sinus tachycardia (which is usually reset) from AV nodal reentrant tachycardia or VT (which are frequently not reset). Electrogram analysis by using digitized atrial signals has allowed differentiation of antegrade and retrograde atrial signals. Similarly, template analysis of right ventricular apical electrograms has shown promise in differentiating sinus tachycardia from VT. Analysis of simultaneous atrial and ventricular recordings has been shown to be helpful in identifying AV dissociation, which would be useful in the diagnosis of VT without ventriculoatrial conduction. Finally, Mancini and Furman demonstrated that recordings from different ventricular sites can differentiate the activation sequence of primary supraventricular and ventricular activation in individual patients.

**Tiered Therapy**

Ultimately, antitachycardia devices used for the treatment of malignant ventricular arrhythmias will incorporate the capability for antitachycardia pacing, low-energy cardioversion, and high-energy cardioversion/defibrillation. An antitachycardia “prescription” will need to be developed from extensive electrophysiological testing. Programmable criteria to allow appropriate automatic selection of antitachycardia
therapy will need to be developed. Predetermined rate criteria or actual hemodynamic data could be used during a tachycardia occurrence to allow the device to “select” the appropriate modality for tachycardia termination. Bardy et al\(^9\) demonstrated that a system using a transvenous catheter could detect hypotensive episodes of VT or VF by measuring changes in right ventricular impedance. Preliminary reports of tiered antitachycardia therapy have been promising.\(^9\)

Several miscellaneous features would ideally be incorporated into future antitachycardia devices. The capability for memory and storage of telemetry data would not only be useful in determining the appropriateness of a device discharge but could have usefulness for long-term electrocardiographic monitoring and assessment of efficacy of concomitant pharmacological therapy for tachycardia suppression. Backup antibradycardia pacing would be essential to prevent interactions with separate demand-pacing systems, and to treat bradycardias that could occur after tachycardia termination. Newer devices will have the ability to perform noninvasive programmed electrical stimulation to allow documentation of the efficacy of pharmacological or device therapy without hospital admission. Ultimately, with possible advances in capacitor technology, the size of the device could be decreased as well.

**Summary**

With the limitations of currently available modalities for treating clinically important tachycardias, the role of implanted antitachycardia devices will continue to expand. The challenge of the future will not only involve continued technological advances but the socioeconomic impact of this efficacious but expensive mode of therapy in an era of increasing financial restraints. Further studies to definitively prove the efficacy of more widespread use of antitachycardia device therapy will be needed.

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