A prospective randomized study of the clinical efficacy and safety of transvenous cardioversion for termination of ventricular tachycardia

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ABSTRACT The clinical efficacy and safety of transvenous cardioversion for termination of sustained ventricular tachycardia (VT) were examined by a prospective randomized study design in 22 patients (19 men, three women; mean age 64 ± 9 years) with organic heart disease and sustained VT. Patients were randomly assigned to undergo an incremental low-energy protocol from 0.03 to 2.2 J (group A, 11 patients) or an incremental high-energy protocol from 0.5 to 10.0 J (group B, 11 patients). Transvenous cardioversion was performed during electrophysiologic studies in the control (drug-free) state and during serial antiarrhythmic drug testing in all patients. Both groups were comparable for demographic, disease and functional status, and electrophysiologic parameters. A total of 77 episodes of VT (group A, 45; group B, 32) were analyzed. The overall efficacy of transvenous cardioversion for termination of VT was 62% (group A 56% vs group B 72%; p < .01). Antiarrhythmic drug therapy did not significantly enhance efficacy of transvenous cardioversion (control 59% vs drug 65%; p > .2). Stepwise discriminant analysis correlated successful transvenous cardioversion with longer VT cycle length (p < .0005), higher energy (p < .025), lower energy waveform tilt (p < .025), shorter time to initial cardioversion attempt (p < .025), and shorter QRS duration in sinus rhythm (p < .05). Acceleration of VT was frequent (8% incidence per delivered shock). Thirty-one percent of all incremental shock protocols were terminated because of this complication. After cardioversion, transient arrhythmias were common (bradyarrhythmias 23%, supraventricular tachyarrhythmias 12%). Displacement of electrode catheters after transvenous cardioversion was uncommon (3%). We conclude that transvenous cardioversion has limited efficacy for termination of VT in unselected patients. The clinical efficacy of the technique can be enhanced by careful patient selection with respect to influencing variables. Acceleration of VT further limits efficacy and requires the availability of defibrillation capabilities.


THE EFFICACY of intracavity cardioversion in terminating sustained ventricular tachycardia (VT) has been demonstrated in experimental and clinical studies.1-6 Recent reports have emphasized the feasibility of low-energy transvenous cardioversion for the termination of sustained VT in selected patients.7-10 However, there has been no systematic controlled study of the efficacy, energy requirements, and safety of this therapeutic technique. We performed a prospective, randomized study to evaluate the efficacy and safety of transvenous cardioversion with two different incremental energy protocols in unselected patients with recurrent and sustained VT.

Methods

Patients. All patients entering this study satisfied the following selection criteria: (1) Patients had recurrent sustained VT and were undergoing clinically indicated electrophysiologic procedures. For this study, "recurrent" was defined as three or more spontaneous episodes and "sustained" as VT of greater than 30 sec duration or requiring earlier electrical or pharmacologic termination for hemodynamic compromise; (2) absence of recent (less than 30 days) myocardial infarction; (3) reproducible induction of sustained VT during electrophysiologic studies; and (4) written informed consent to undergo transvenous cardioversion.

Twenty-two patients satisfied all inclusion criteria. There were 19 men and three women, mean age 64 ± 9 years. All patients had organic heart disease, with a mean left ventricular ejection fraction of 31%. Coronary artery disease was present in 18 patients and cardiomyopathy in four patients.

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Catheter and cardioversion system. The cardioversion lead was a No. 9.5F Medtronic 6880 temporary electrode catheter. This is a tripolar catheter with distal and intermediate electrodes, each with a surface area of 1.25 cm² separated by an interelectrode distance of 5 mm. These electrodes were positioned in the right ventricular apex and were used for sensing the ventricular electrogram and for bipolar ventricular pacing. During cardioversion both became electrically common, forming the cathode. The two proximal electrodes, each 1.25 cm² in surface area, were also separated by 5 mm and were 125 mm proximal to the distal electrode pair. These were usually located at the superior vena cava–high right atrial junction, were common, and formed the anode during cardioversion. Lead position was verified by fluoroscopy at the time of electrophysiologic study. The electrodes were connected by three coil-wound, multifilar, drawn brazed-strand, low-impedance, wire electrical conductors in a coaxial configuration to three connector pins. The conductors were insulated by sleeves of polyurethane. The connector pins at the proximal end of the catheter were connected by a cable to a Medtronic model 5350 external cardioverter-defibrillator. This device is a battery-operated generator capable of delivering graded energy shocks when the storage capacitor is discharged. It has sensing capabilities, allowing shocks to be synchronized with the sensed electrogram. The energy output ranges from 0.03 to 28.0 J. Three different waveforms controlled by tilt settings can be selected. The cardioverter can also be interfaced with a programmed stimulator by an external input that connects directly to the distal catheter electrodes for performance of programmed electrical stimulation and/or bipolar pacing.

Study design. Patients were prospectively randomized to undergo one of two incremental transvenous cardioversion protocols. Group A patients entered a low-energy protocol consisting of eight specified incremental shocks ranging from 0.03 to 2.2 J (0.03, 0.05, 0.06, 0.11, 0.27, 0.56, 1.1, and 2.2). Group B patients entered a high-energy protocol with six specified incremental shocks ranging from 0.5 to 10.0 J (0.5, 1.0, 1.5, 2.0, 5.0, and 10.0). Groups A and B included 11 patients each, all of whom completed the entire study protocol.

After randomization, patients underwent initial electrophysiologic studies in the control, drug-free state. All patients were studied in the nonsedated, postabsorptive state. Multipolar catheters were inserted by standard percutaneous introduction techniques as previously described. Intracardiac electrograms were usually monitored at high and mid-right atrium, His bundle, and right ventricular apex. Surface electrocardiographic leads I, aVF, and V₆ were recorded simultaneously with intracardiac electrograms. Arterial blood pressure was monitored with an indwelling femoral arterial sheath connected to a Statham P23 transducer. A multichannel display recorder (Electronics for Medicine VR-12, White Plains, NY) was used to amplify and display the electrograms. Hard-copy recordings were obtained at paper speeds of 50 to 250 mm/sec. All records were stored on magnetic FM tape. Programmed electrical stimulation was performed with a custom-made programmed stimulator (Bloom Associates., Ltd., Norbeth, PA), which delivered rectangular pulses of 1 or 2 msec duration at twice diastolic threshold. Programmed stimulation was performed with a standard protocol for our laboratory as previously described.

After sustained VT was induced, immediate transvenous cardioversion was attempted with the preselected protocol. The protocol was terminated if successful transvenous cardioversion of VT was achieved or if VT accelerated or degenerated into ventricular fibrillation (VF). Unsuccessful transvenous cardioversion was defined as inability to terminate VT at the highest energy level in a particular protocol or if VT accelerated or degenerated into VF.

Patients were subsequently restudied one or more times during serial testing with an antiarrhythmic drug. Identical techniques were used as in the control study. Transvenous cardioversion was attempted according to the previously assigned protocol once sustained VT was induced.

Data analysis. Several factors were analyzed for possible importance in determining the efficacy of transvenous cardioversion. These included age and sex, disease and functional status, characteristics of induced VT (morphology, cycle length, and induction mode), cardioversion lead threshold for ventricular pacing, time to initial attempt of cardioversion, right ventricular effective refractory period, and presence or absence of antiarrhythmic therapy. Statistical analysis included data description, analysis of variance, and stepwise discriminant analysis.

Results

Comparison of study groups (Table 1)

Clinical characteristics. There was no statistically significant difference in mean age, sex, disease status, or left ventricular ejection fraction between the two groups.

Electrophysiologic characteristics. QRS duration during sinus rhythm, mean corrected QT interval, and right ventricular effective refractory period were comparable in groups A and B. Of the 45 episodes of VT in group A, 19 were of left bundle branch block QRS morphology, 24 were of right bundle branch block QRS morphology, and two had a polymorphic configuration. Of the 32 episodes in group B, 16 were of left bundle branch block QRS morphology and 16 were of right bundle branch block morphology. One patient had VT of both morphologies. There was no significant difference in this variable between the two groups.

Comparison of transvenous cardioversion attempts. A total of 77 episodes of VT were analyzed. Forty-five occurred in group A and 32 in group B patients. Two

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Clinical and electrophysiologic characteristics of patient groups</th>
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<tbody>
<tr>
<td></td>
<td>Group A</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>66 ± 10</td>
</tr>
<tr>
<td>Sex</td>
<td>9 M, 2 F</td>
</tr>
<tr>
<td>Disease status</td>
<td>CAD 9, CM 2</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>33 ± 20</td>
</tr>
<tr>
<td>QRS complex duration in sinus rhythm (msec)</td>
<td>110 ± 22</td>
</tr>
<tr>
<td>QTc in sinus rhythm (msec)</td>
<td>451 ± 37</td>
</tr>
<tr>
<td>VTCL (msec)</td>
<td>259 ± 83</td>
</tr>
<tr>
<td>ERPV (msec)</td>
<td>241 ± 28</td>
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LVEF = left ventricular ejection fraction; QTc = corrected QT interval; VTCL = ventricular tachycardia cycle length; ERPV = ventricular effective refractory period; CAD = coronary artery disease; CM = cardiomyopathy.
hundred eighty-five shocks were delivered, 196 to group A patients and 89 to group B patients. In group A, 18 episodes of VT occurred during the control study and 27 after treatment with antiarrhythmic drugs. An average of 4.1 shocks, 1.6 at control study and 2.5 after antiarrhythmic therapy, was delivered in these patients. In group B, 16 episodes of VT occurred during the control study and 16 after treatment with antiarrhythmics. An average of 2.9 shocks, 1.45 at control study and 1.45 after antiarrhythmic therapy, was delivered in these patients. There was no significant difference in the number of transvenous cardioversion attempts in either group.

Antiarrhythmic therapy. An average of 1.6 ± 1.0 antiarrhythmic agents per patient was used in group A compared with 1.5 ± 0.7 per patient in group B. Antiarrhythmic agents used in group A patients included procaainamide (three patients), lidocaine (seven patients), quinidine (one patient), mexiletine (one patient), amiodarone (three patients), and cibenzoline (two patients). In group B patients, procainamide (eight patients), lidocaine (one patient), quinidine (two patients), mexiletine (two patients), and amiodarone (two patients) were used.

Efficacy of transvenous cardioversion

Effect of energy protocol on efficacy of transvenous cardioversion. The overall efficacy of transvenous cardioversion, defined as the percentage of all episodes of VT successfully terminated, was 62%: 56% in group A and 72% in group B. The difference in efficacy between the two groups was statistically significant (p < .01).

Effect of antiarrhythmic therapy. The efficacy of transvenous cardioversion for both groups in the control state was 59%; after antiarrhythmic drug therapy efficacy of cardioversion was 65%. This difference was not statistically significant (p > .2).

Factors influencing successful transvenous cardioversion. Table 2 summarizes the results of a stepwise discriminant analysis that attempted to correlate results of transvenous cardioversion with different parameters. Successful transvenous cardioversion correlated with five variables, including VT cycle length, mean time to initial attempt of transvenous cardioversion, tilt of the waveform, energy of the delivered shock, duration of ventricular depolarization during sinus rhythm.

Mechanisms of termination (figures 1A to 1C). Immediate termination of VT by the delivered shock was the most frequent mechanism of termination (figure 1A). Overall, 45 of 48 (94%) successfully cardioverted episodes of VT were immediately terminated by the delivered shock. One patient had transient slowing with change in QRS morphology from left bundle branch block to right bundle branch block before termination (figure 1B). Only two of 48 (4%) successful cardioversions displayed transient acceleration before cardioversion (figure 1C). Figure 2 shows data from catheter endocardial mapping performed during a transvenous countershock. This 5 J shock depolarized both ventricles, reset the tachycardia circuit, which slowed, and changed ventricular activation pattern and QRS complex morphology before termination.

Complications of transvenous cardioversion

Acceleration of VT or degeneration into VF. Twenty-four (8%) of all transvenous cardioversion shocks resulted in sustained acceleration of VT (figure 3). However, 31% of all episodes of VT accelerated during the course of a particular cardioversion protocol. Seventy-six percent of all failed transvenous cardioversion protocols in groups A and B were terminated because of acceleration of VT. Shocks were synchronized with the QRS complex in all instances. The location of the shock within the QRS complex did not correlate with acceleration of VT. In the low-energy cardioversion protocol, patients who were successfully cardioverted did not experience prior transient acceleration of VT regardless of drug therapy. Two group B patients at control study demonstrated transient acceleration of VT before successful cardioversion. Therefore, only two of 24 episodes (8%) displaying acceleration of VT resulted in successful cardioversion.

Table 2

<table>
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<th>Table 2 Discriminant analysis of factors influencing results of transvenous cardioversion</th>
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<tr>
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<tr>
<td>Age (yr)</td>
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<td>LVEF (%)</td>
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<td>VTCL (msec)</td>
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<tr>
<td>Energy (J)</td>
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<td>Tilt (%)</td>
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<td>TTCV (sec)</td>
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TTCV = time to control cardioversion attempt; other abbreviations as in table 1.
the different energy protocols. Acceleration of VT at control studies in both groups occurred at a mean shock energy of 0.5 ± 1.5 J. After antiarrhythmic therapy the mean shock energy resulting in acceleration of VT increased significantly to 1.1 ± 1.5 J (p < .05).

Lead complications. In two patients the electrode catheter was displaced after a transvenous cardioversion attempt for an overall incidence of 3%. The lead was left in situ for 1 to 10 days and major local complications were not observed. The mean pacing threshold with this catheter before cardioversion was 4.0 ± 1.1 mA at 1 msec pulse width and remained unchanged at 4.0 ± 1.2 mA after cardioversion.

Postcardioversion rhythm. Transient bradyarrhythmias were observed after transvenous cardioversion. Eight of 45 episodes of VT (18%) in group A and 10 of 32 episodes (31%) in group B manifested a variety of bradyarrhythmias. These included sinus bradycardia (seven episodes), sinus rhythm with high grade atrioventricular block (six episodes), junctional rhythm (two episodes), idioventricular rhythm (two episodes), and asystole for 6 sec (one episode). Supraventricular tachyarrhythmias were also noted. The incidence of

FIGURES 1A to 1C. Mechanisms of termination of VT after transvenous countershock.

FIGURE 1A. Immediate termination by delivered transvenous cardioversion shock. ECVD = external cardioverter/defibrillator; HRA = high right atrium; MRA = mid right atrium; H-P = His bundle, proximal; H-D = His bundle, distal; Ao = aortic pressure; CL = cycle length.

FIGURE 1B. Change in VT morphology before termination by transvenous countershock. LBBB = left bundle branch block; RBBB = right bundle branch block; NSR = normal sinus rhythm; HBE = His bundle electrogram; other abbreviations as in figure 1A.
supraventricular tachyarrhythmias in the two groups was comparable (group A, four [9%]; group B, five [16%]). These included persistent sinus tachycardia (one episode), atrial fibrillation with rapid ventricular response (five episodes), and atrial flutter with varying block (three episodes). Two patients with chronic atrial fibrillation (one each in groups A and B) converted to sinus rhythm after transvenous cardioversion.

Patient tolerance. Patient tolerance for transvenous cardioversion shocks showed wide individual variability but was also related to absolute shock energy. Shocks in the energy range of 0.03 to 0.5 J were perceived as tolerable (painless or minimally uncomfortable) by 56% of patients, and intolerable (moderately or severely painful) by 44%. From 0.51 to 1.0 J, 43% considered the discomfort to be tolerable, while 57% experienced moderate or severe discomfort. Above 1.0 J, only 24% considered the experience tolerable. The highest energy shock reported to be tolerable by a patient in this study was 2.2 J.

FIGURE 1C. Acceleration of VT with polymorphism before termination. AF = atrial fibrillation; other abbreviations as in figures 1A and 1B.

FIGURE 2. Catheter endocardial mapping during transvenous countershock with the external cardioverter/defibrillator (ECVD). RV = right ventricle; RVOT-p = right ventricular outflow tract, proximal; RVOT-d = right ventricular outflow tract, distal; LV-p = left ventricle, proximal; LV-d = left ventricle, distal.
Discussion

Electrical cardioversion of sustained VT is performed with external transthoracic electrodes over a wide range of energy. The feasibility of internal transvenous electrical defibrillation has been experimentally and clinically demonstrated.1-10 The automatic implantable defibrillator currently under clinical investigation for internal cardioversion and defibrillation uses energy discharges from 20 to 30 J.12 Recently, Zipes et al.7 reported successful transvenous cardioversion of sustained VT with the Medtronic 6880 electrode catheter with energies ranging from 0.03 to 2.0 J in five of seven patients studied. They also noted acceleration of VT and degeneration into VF in one patient. Yee et al.9 used a conventional defibrillation unit providing a damped sinusoidal discharge waveform and higher countershock energies ranging from 2.5 to 40 J. Although they were able to terminate 87% of episodes of VT with this technique, four of the six patients studied experienced both successful and failed transvenous cardioversion. Therefore, although uncontrolled studies in small groups of patients suggest that transvenous cardioversion is valuable in termination of VT, a controlled prospective trial to determine its clinical efficacy and safety is currently unavailable. In addition, the factors underlying successful and failed cardioversion are currently unknown. This study was designed to clarify these issues.

Clinical efficacy. The overall efficacy of transvenous cardioversion in this prospective study was moderate (62%) in unselected patients with VT. This is comparable to the findings of a prior report by Waspe et al.,10 but is substantially lower than the results of some other reports.7-9 The differences in efficacy rate are clearly explained by the unselected controlled study design and larger experience in this report as compared with selected patients in the prior reports. Additionally, an analysis of factors influencing efficacy of this technique can account for different efficacy rates. VT cycle length is a major determinant of efficacy. Longer VT cycle lengths are associated with greater efficacy. The mean VT cycle length in the successfully cardioverted patients was significantly longer than that in the patients who could not be cardioverted in this study (316 ± 70 vs 265 ± 51 msec, respectively; p < .0005). Similar observations were noted by Waspe et al.10 in a smaller number of patients studied in an uncontrolled energy protocol ranging from 0.01 to 5.0 J. VT cycle length exceeded 400 msec in 68% of episodes of VT reported by Zipes et al.7 This is substantially longer than the value in the present report (mean VT cycle length, 289 ± 59 msec) and could alone account for different efficacy rates. Similar information is unavailable in the report from Yee et al.9

Our observations indicate that the absolute cardioversion shock energy is another important factor in determination of efficacy. Episodes of VT treated with the high-energy protocol had significantly greater mean energy delivered per shock and consequently greater efficacy. Yee et al.9 used energies ranging from 2.5 to 40 J and noted an efficacy rate of 65% when the energy delivered was below 5 J, quite similar to the rate in this study. However, energies up to 40 J increased the overall efficacy rate to 87%. Since patient tolerance for the cardioversion shock becomes a major factor in determining the clinical application of this technique, increasing efficacy rates at increasing energy levels were analyzed in this study. Figure 4 shows

FIGURE 3. Sustained acceleration of VT caused by transvenous countershock. Abbreviations as in figure 1A.
that at energies up to 0.5 J, up to 70% of all successful cardioversions were obtained. At 2.2 J, 82% of all successful transvenous cardioversions were obtained. Since patient tolerance rapidly declines after 0.5 J shocks, use of this technique in clinical practice should, in our opinion, be restricted to energies below 2.2 J.

The time to initial cardioversion attempts can also influence efficacy of the technique. Earlier delivery of the initial cardioversion shock in this study enhanced efficacy. The mean time in this report to initiation of the successful cardioversion attempts was 24 sec. Early application may be important, particularly in some episodes of VT in which a stable VT circuit may not be immediately established, to permit easier interruption. The role of additional factors such as myocardial ischemia during prolonged VT in patients with significant coronary artery disease may be important in reducing the efficacy of this technique. At variance with our observations is a prior report that did not note a relationship of efficacy to the timing of cardioversion shock. However, the onset of cardioversion attempts in that study was somewhat later (43 sec), comparable to our failed cardioversion attempts. In addition, the ventricular function of the patient population in their study (mean left ventricular ejection fraction 40%) was somewhat better than that in our patients.

The intraventricular conduction pattern in sinus rhythm differed in the successful and failed cardioversion groups, being significantly better in the former. Although this has not been previously analyzed, better intraventricular conduction may enhance efficacy of the cardioversion shock by improved and rapid penetration of critical components of the VT circuit.

Safety. Acceleration of VT during failed transvenous cardioversion remains the major safety problem in the clinical application of this technique. Although the potential of acceleration for an individual shock is low, the use of a series of incremental shocks, as is general clinical practice for transthoracic cardioversion, markedly increases the frequency of this problem. Cardioversion protocols for 31% of all episodes of VT were terminated because of this complication. However, the incidence was higher in the absence of antiarrhythmic drugs (32%) as compared with that observed with drugs (23%). Antiarrhythmic therapy also increased the mean energy at which acceleration of VT was observed. Although the wide variety of antiarrhythmic agents used precludes a useful comparative analysis, the electrophysiologic effects of these agents may provide partial protection against this complication.

Lead complications were infrequent in this study. The incidence of displacement of electrode catheters was low (3%) in this and other studies, but active fixation electrodes may be considered to eliminate this problem. Prolonged use of the catheter also appears to be feasible. The catheter electrode system was easily inserted, and electrode location with the 125 mm spacing was usually appropriate in this study group. Early experience with a similar electrode catheter with tines for long-term implantation appears favorable.

The incidence of postcardioversion arrhythmias is substantial and merits analysis. Transient supraventricular arrhythmias were common and usually hemodynamically insignificant. Rarely, sustained supraventricular arrhythmias resulted and necessitated treatment. Early experience with a prototype implantable unit indicates that these arrhythmias can trigger automatic detection algorithms in an implanted device. However, the occurrence of bradyarrhythmias is more disturbing. The incidence of this complication is significant (23%) and although it is usually transient, asystole was observed after one episode of VT. This implies the need for availability of demand pacing capability whenever this technique is used. Yee et al. observed that catheter countershocks can abruptly increase pacing thresholds and decrease R wave amplitude at the same site as the countershock. Therefore the catheter electrodes used for countershock may not be acceptable for immediate backup demand pacing. However, pacing threshold when measured shortly after transvenous cardioversion returns to baseline as seen in this study.

**Comparison of transvenous cardioversion with alternative modes of electrical termination of VT.** Alternative electrical techniques for termination of VT with electrode catheters have been used. Fisher et al. demonstrated the ability of bursts of rapid ventricular pac-
ing to terminate sustained VT in a retrospective analysis of 23 patients. Although the efficacy rate of this technique was high (89%), acceleration of VT was observed infrequently (4% incidence per burst). Comparison of this technique with transvenous cardioversion merits examination. A comparative trial performed in our laboratory with a prospective randomized crossover study design has demonstrated comparable efficacy of the two techniques (transvenous cardioversion 83%, rapid ventricular pacing 80%; p > .2). The incidence of acceleration of VT was also comparable (transvenous cardioversion 7% per shock, rapid ventricular pacing 2% per burst; p > .2). Patient tolerance was clearly better and the incidence of postcardioversion arrhythmias was significantly lower (incidence 3%) with rapid ventricular pacing. Furthermore, data analysis indicates a high degree of overlap in the efficacy of these techniques, suggesting that both techniques are competitive and not complementary.

Conclusions. The results of this prospective study indicate that transvenous cardioversion has limited efficacy for termination of VT in unselected patients. Both intrinsic factors, related to the patient and the tachycardia, and extrinsic factors, related to the energy shock, influence efficacy. Careful selection of patients and the energy of countershock with respect to these factors will enhance the efficacy of this technique. Although antiarrhythmic drugs may increase the safety of the technique, the potential hazard of acceleration of VT requires availability of defibrillation capabilities whenever the technique is used. The occurrence of postcardioversion bradyarrhythmias may warrant the need for a backup demand pacing system. Current lead systems may be clinically applicable for short-term use. However, comparison with a currently available alternative for electrical termination of VT has not demonstrated clear advantages of this technique and indicates potentially limiting untoward effects for short-and long-term use.

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