The implantable transvenous cardioverter: long-term efficacy and reproducible induction of ventricular tachycardia

WILLIAM M. MILES, M.D., ERIC N. PRYSTOWSKY, M.D., JAMES J. HEGER, M.D., AND DOUGLAS P. ZIPES, M.D.

ABSTRACT We followed 11 patients for 5 to 27 months (mean 14.9) after implantation of a permanent transvenous low-energy synchronized cardioverter to evaluate both long-term reproducibility of ventricular tachycardia (VT) induction via noninvasive programmed electrical stimulation with the cardioverter and efficacy of cardioversion. Induction and termination of VT were attempted at implantation and approximately every 3 months thereafter. All patients had coronary artery disease and were receiving antiarrhythmic drug therapy (amiodarone in eight). VT cycle length, morphology, and mode of induction were reproducible on multiple occasions in nine patients; clinical VT was induced inconsistently in two patients. Multiple VT episodes in five patients had one morphology, whereas two morphologies occurred in six patients. Synchronization of the shock within the QRS complex and right ventricular effective refractory period of the cardioverter remained constant over the follow-up period. VT was terminated on every occasion in nine patients and on eight of nine occasions in one patient. Tachycardia was accelerated on three of five occasions in one patient. Consistently effective cardioversion energy (0.2 to 2.0 J) increased modestly in four patients. We conclude that (1) patients with inducible monomorphic VT usually have sustained VT with similar characteristics inducible over a period of time and (2) cardioversion and sensing functions of the cardioverter remain relatively stable over time.


STUDIES in dogs and in human subjects have shown that ventricular tachycardia (VT) can be terminated safely and effectively with low-energy shocks synchronized to the QRS complex and delivered over a catheter electrode. We subsequently developed a system for transvenous cardioversion that employed permanent implantation of the device under local anesthesia without thoracotomy. An important feature of the device is that programmed electrical stimulation and electrophysiologic measurements can be repeatedly performed noninvasively via the implanted cardioverter lead.

Repeated induction of VT on multiple occasions over long periods has never been reported, and therefore the stability of induction characteristics and other electrophysiologic variables is unknown. The purpose of this report is to describe the long-term reproducibility of induction characteristics of VT, transvenous cardioversion of induced VT, and ventricular refractoriness as measured via the implanted cardioverter.

Methods

Patient selection. Patients were considered candidates for the implantable transvenous cardioverter if they had recurrent episodes of sustained monomorphic VT and no history of spontaneous ventricular fibrillation except that associated with an acute myocardial infarction. All patients were able to recognize the onset of VT, and in all except one patient who had syncope, tachycardia was tolerated for at least several hours without hemodynamic deterioration, allowing sufficient time to receive medical help. Each patient had required repeated transthoracic cardioversions despite conventional and investigational drug therapy. Patients either refused or were not considered candidates for surgical treatment of VT, usually because of generalized poor left ventricular function. Each patient had a successful preoperative response to catheter cardioversion during electrophysiologic study (see below). We did not systematically compare efficacy of VT termination by competitive-pacing techniques with that of transvenous cardioversion. Each patient gave oral and written informed consent for both the preoperative studies and the implantation of the permanent unit.
Implantable cardioverter. The implantable cardioverter (Model 7210, Medtronic) has been described in detail previously. In brief, the device is encased in a hermetically sealed titanium case measuring 57 x 73 x 19 mm, weighing 95 g, and powered by two independent lithium battery sources. The device can be programmed to deliver truncated exponential waveforms of 0.06 to 2.0 J synchronized to the QRS complex over a single 10F lead (Medtronic 6882) with electrodes at the right ventricular apex (cathode) and right atrial–superior vena cava junction (anode). The unit can be discharged by the external programmer or automatically by means of a rate-detection algorithm. The unit also functions as a programmable ventricular demand or asynchronous pacemaker and can be programmed to deliver a drive train of basic stimuli at a fixed cycle length, followed by one or more (up to 99) premature ventricular extrastimuli at varying intervals. The unit has bidirectional telemetry with printout of marker pulses, electrograms, device settings, and end of battery life indicators.

Cardioverter testing and follow-up. All patients underwent preoperative electrophysiologic study to test the efficacy of transvenous cardioversion with a temporary lead (Medtronic 6880, very similar to the permanent lead) while receiving the antiarrhythmic regimen that best controlled their VT and that they would most likely receive long term. This testing has been described in detail previously and involves use of an external cardioverter unit (Medtronic 5350) that functions in a manner similar to the implantable unit. If energy levels above 1.7 J were required for termination of VT or if a synchronized shock accelerated the VT by more than 15 beats/min or resulted in more than three to five repetitive ventricular complexes after the shock, the patient was not considered a candidate for cardioverter implantation. At least three episodes of VT per patient were terminated before implantation of the permanent device. At the time of implantation of the permanent device, the cardioversion threshold of the implanted lead was demonstrated to be similar to that at the initial study before the cardioverter was implanted. By means of the external programmer, VT was then induced, if possible, and terminated with the implanted device while the patient was still in the operating room.

Programmed ventricular stimulation was performed via the cardioverter at the time of implantation and approximately 1 week, 1 month, and every 3 months thereafter, unless spontaneous VT had occurred in the interim and was successfully terminated with the device. Six patients had follow-up programmed stimulation performed by the authors, while five patients were also tested by their referring physicians. The VT induction protocol included introducing one ventricular extrastimulus (S2) throughout diastole at 8 msec decrements to ventricular refractoriness after drive trains (S1) of eight complexes at cycle lengths of 600, 504, and 400 msec. If no VT was induced, S2 was introduced at an S1S2 interval 8 to 16 msec longer than ventricular refractoriness, and two (S2) and subsequently three (S3) extrastimuli were introduced. Patient 7 consistently required burst ventricular pacing at a cycle length of approximately 300 msec for induction of VT. Ventricular effective refractory period was defined as the longest S1S2 interval at which S2 failed to capture the ventricle after a pacing drive train of eight complexes. Pacing stimuli were rectangular pulses of 0.5 msec duration at an intensity of 6.0 V.

Whenever possible, multiple electrocardiographic leads were obtained during VT to determine its morphology. Tachycardia morphology was described as resembling that of a left or right bundle branch block with a normal, left, right, or superior frontal axis. Sensing of VT by the cardioverter was described by the interval (in msec) between the onset of the QRS during tachycardia and the delivery of the cardioversion impulse or the ventricular endocardial sensing marker. Cardioversion testing was performed with the device programmed into the manual mode. The cardioversion energy delivered at each follow-up visit was that previously demonstrated to be effective; there was no attempt to reestablish cardioversion thresholds.

Results

Patients. Eleven of 13 patients were followed for a mean of 14.9 months (range 5 to 27) after implantation of the permanent transvenous cardioverter (table 1). Two patients refused follow-up induction of VT. Ten of the 11 patients were men, ages 44 to 67 years (mean 61.9). All patients had coronary artery disease and at least one myocardial infarction. Five patients had left ventricular aneurysms, and five had undergone previous cardiac surgery. Patients had had myocardial infarction or cardiac surgery at least 6 months before implantation of the cardioverter, and no patient had myocardial infarction or cardiac surgery during the follow-up period. All patients received pharmacologic antiarrhythmic therapy (amiodarone in eight); drug therapy was changed during follow-up only in patients 5 and 8. The mean cycle length of VT induced immediately after implantation of the device was 453 msec (range 400 to 510). Five patients (2, 4, 8, 10, and 11) were known to have had two distinct monomorphic VTs inducible before implantation of the cardioverter.

VT cycle length. Eleven patients had spontaneous and/or induced VT during the follow-up period. VT cycle length data are illustrated in figure 1. Filled circles represent induced and unfilled circles spontaneous VT. Where applicable, tachycardias of different morphologies have been separated. Patient 1 was followed for 27 months, and despite reproducible VT induction before implantation of the cardioverter, sustained VT was induced at only three of 12 attempts after implantation. The last two episodes of tachycardia had cycle lengths much shorter than had ever occurred clinically and were thought not to represent the clinical arrhythmia. An attempt to induce VT in this patient after cardioverter implantation with routine intracardiac electrode catheters at two additional right ventricular sites was also unsuccessful. Despite multiple episodes of spontaneous sustained VT before cardioverter implantation, he has had no spontaneous VT after implantation. Patient 8 also demonstrated inconsistent VT induction in the peri-implant period and a more rapid tachycardia of different morphology was induced at 15 months follow-up; this tachycardia may have been nonclinical or related to a change in antiarrhythmic drug therapy. The remaining nine patients demonstrated less variation in VT cycle length, and the cycle length of the induced VT was comparable to that oc-
TABLE 1

Patient characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Myocardial infarction (yr before implant)</th>
<th>Sex</th>
<th>Aneurysm</th>
<th>Myocardial infarction (yr before implant)</th>
<th>Aneurysm</th>
<th>Surgery (yr before implant)</th>
<th>VT cycle length (msec)</th>
<th>VT mode of induction</th>
<th>VT cycle length (msec)</th>
<th>VT mode of induction</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55/M</td>
<td>A (9)</td>
<td>+</td>
<td>An (14)</td>
<td>+</td>
<td>An (14)</td>
<td>+</td>
<td>250</td>
<td>S₄</td>
<td>510</td>
<td>Drive train</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>2</td>
<td>67/M</td>
<td>A (9)</td>
<td>+</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>320</td>
<td>S₂</td>
<td>430</td>
<td>S₃</td>
<td>ProcainamideC</td>
</tr>
<tr>
<td>3</td>
<td>63/M</td>
<td>I (13)</td>
<td>+</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>360</td>
<td>S₃</td>
<td>400</td>
<td>S₃</td>
<td>Propafenone</td>
</tr>
<tr>
<td>4</td>
<td>63/M</td>
<td>I (4)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>260</td>
<td>S₂</td>
<td>420</td>
<td>S₂</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>5</td>
<td>67/M</td>
<td>A (12)</td>
<td>+</td>
<td>CABG (4)</td>
<td>An (4)</td>
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<td>—</td>
<td>400</td>
<td>S₃</td>
<td>480</td>
<td>S₃</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>6</td>
<td>66/M</td>
<td>I, A (12)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>300</td>
<td>S₄</td>
<td>460</td>
<td>S₂</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>7</td>
<td>44/M</td>
<td>I (9)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>300</td>
<td>S₃</td>
<td>420</td>
<td>S₃</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>8</td>
<td>65/M</td>
<td>I, A (10)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>None</td>
<td>None</td>
<td>490</td>
<td>S₃</td>
<td>MexiletineE</td>
</tr>
<tr>
<td>9</td>
<td>66/M</td>
<td>L (1)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>275</td>
<td>S₃</td>
<td>470</td>
<td>S₃</td>
<td>Amiodarone</td>
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<tr>
<td>10</td>
<td>61/F</td>
<td>I (1/2)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>330</td>
<td>S₂</td>
<td>450</td>
<td>S₂</td>
<td>MexiletineF</td>
</tr>
<tr>
<td>11</td>
<td>64/M</td>
<td>I (23)</td>
<td>+</td>
<td>CABG (8)</td>
<td>An (14)</td>
<td>—</td>
<td>—</td>
<td>None</td>
<td>None</td>
<td>450</td>
<td>S₂</td>
<td>Amiodarone</td>
</tr>
</tbody>
</table>

A = anterior; I = inferior; L = lateral; EPS = electrophysiologic study; CABG = coronary artery bypass graft; MVR = mitral valve replacement; An = Aneurysmectomy; S₂ = one ventricular extrastimulus; S₃ = two ventricular extrastimuli; S₄ = three ventricular extrastimuli; VT-NS = nonsustained VT; None = no drug-free EPS performed.

A. Most recent infarction.
B. First VT induced after implant.
C. Amiodarone discontinued before implant.
D. Amiodarone alone months 0–7; tocainide alone month 17; no antiarrhythmic drug months 11 and 18.
E. Amiodarone discontinued for 1 month, receiving mexiletine at implant; disopyramide alone months 2–15; disopyramide plus tocainide at VT induction month 15.

Currington spontaneously. Patients 2, 10, and 11 demonstrated different tachycardia cycle lengths depending on tachycardia morphology. Antiarrhythmic medication was altered over the follow-up period in patients 5 and 8 only (table 1); the remainder of the patients were receiving their “best” antiarrhythmic drug regimen at the time of device implantation and drug therapy was not subsequently altered. Amiodarone had been discontinued just before implantation of the device in patients 2 and 8.

VT induction techniques. Figure 2 illustrates the mode of VT induction for the 11 patients at each follow-up visit. VT was induced with the drive train, one ventricular extrastimulus, two extrastimuli, burst pacing, or three extrastimuli. The height of each bar illustrates the number of follow-up visits at which VT was induced by that mode of induction. As discussed above, sustained VT was not reproducibly induced in patient 1.

Induction of VT was also inconsistent in patient 8 at implantation and during follow-up, although several spontaneous episodes of VT subsequently occurred in this patient (figure 1). In the remaining patients, the mode of VT induction tended to remain stable over time, although slight variation is evident. Most patients had VT induced consistently with one or two ventricular extrastimuli. Upon comparison of the first induction of VT after device implantation (table 1) with subsequent modes of induction, only patient 7 demonstrated a tendency toward requiring a more aggressive pacing technique to induce the clinical arrhythmia.

VT morphology and sensing. The QRS morphology of VT could be analyzed for most episodes in all 11 patients (table 2). Five patients (3, 5, 6, 7, and 9) had one monomorphic VT identified; in one of these five patients (patient 5), spontaneous VT occurred during
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monomorphic but intracardiac phanologies, tachycardia morphologies.

Four patients (2, 4, 10, and 11) had two distinct but monomorphic VTs. In the remaining two patients (Nos. 1 and 8), the more rapid VTs were thought to be nonclinical. In patient 2, the spontaneous VT morphology and cycle length were different from those of the induced VT. In all patients with two tachycardia morphologies, intracardiac cardioversion was successful for both.

Cardioverter sensing characteristics for monomorphic VTs are illustrated in figure 3. Values for two tachycardia morphologies are plotted separately for patients 2, 4, 10, and 11; three of these four patients demonstrated different sensing values for different tachycardia morphologies. The tachycardias in patient 1 were not comparable and are not illustrated. The sensing values for any VT morphology showed no change during follow-up.

Patient 4 demonstrated transient (≤43 sec) loss of VT sensing after unsuccessful shocks on two occasions. Sensitivity thresholds were not obtained, but the only other instance of failure-to-sense was occasional lack of recognition of premature ventricular complexes during ambulatory recording. The ventricular pacing threshold remained low (≤0.1 msec pulse duration). Time to tachycardia QRS sensing was unchanged once sensing returned. Neither patient 4 nor any other patient demonstrated loss of ventricular pacing capture when pacing occurred immediately after a shock.

Ventricular refractoriness. Figure 4 illustrates right ventricular effective refractory period determinations available in seven patients who had not undergone change in antiarrhythmic medications. The most complete evaluation of refractory periods was available for 27 months of follow-up in patient 1. This patient had

FIGURE 1. VT cycle length in each of the 11 patients over the follow-up period. The filled circles represent induced VT and the unfilled circles spontaneous VT. Where applicable, tachycardia morphology is indicated. LB = left bundle branch block morphology; RB = right bundle branch block morphology; Right = right axis deviation; Sup = superior axis deviation.

follow-up and had the same morphology as the induced VT. Four patients (2, 4, 10, and 11) had two distinct but monomorphic VTs. In the remaining two patients (Nos. 1 and 8), the more rapid VTs were thought to be nonclinical. In patient 2, the spontaneous VT morphology and cycle length were different from those of the induced VT. In all patients with two tachycardia morphologies, intracardiac cardioversion was successful for both.

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FIGURE 2. Mode of VT induction immediately after implantation and at each of the follow-up visits for each of the 11 patients (numbered at the right). Modes of VT induction included drive train, one ventricular extrastimulus (S2), two ventricular extrastimuli (S3), burst ventricular pacing, and three ventricular extrastimuli (S4).
TABLE 2
VT morphology

<table>
<thead>
<tr>
<th>Patient</th>
<th>VT morphology</th>
<th>No. of episodes(^a)</th>
<th>Follow-up (mo)</th>
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<tr>
<td></td>
<td></td>
<td>Induced</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>1</td>
<td>RBBB right axis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>RBBB superior axis</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>RBBB right</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>LBBB superior</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>RBBB superior</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>LBBB superior</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>RBBB superior</td>
<td>18</td>
<td>0</td>
</tr>
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<td>5</td>
<td>RBBB superior</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>6</td>
<td>RBBB right</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>RBBB superior</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>RBBB right</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>RBBB superior</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>9</td>
<td>RBBB right</td>
<td>6</td>
<td>0</td>
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<tr>
<td>10</td>
<td>RBBB superior</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>LBBB left</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>11</td>
<td>RBBB superior</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>RBBB right</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

RBBB = right bundle branch block; LBBB = left bundle branch block.

\(^a\)Only VT episodes with known morphology are included.

been receiving amiodarone for 7 months before cardioverter implantation and received no other antiarrhythmic drug during follow-up, although the amiodarone dosage was decreased gradually from 600 to 200 mg daily over the follow-up period. After a small initial rise, the right ventricular effective refractory periods at pacing cycle lengths 600 and 500 msec were stable over the remainder of the follow-up period. Reproducibility of refractory period determinations was confirmed in the remaining six patients.

VT cardioversion. Table 3 illustrates efficacy of cardioversion over the follow-up period. Six of the 11 patients had spontaneous as well as induced VT terminated by the device. The energy required for successful cardioversion of VT during follow-up was unchanged from initial study in six patients (Nos. 1, 2, 4, 6, 7, and 10). Four patients required an increase in energy over the follow-up period but successful cardioversion was still achieved. In patients 5, 9, and 11 cardioversion energy was increased by 0.3 to 0.5 J to the next highest setting programmable on the implanted unit; thus, this may represent only a minimal increase in cardioversion threshold. Patient 8 had one VT episode not terminated with 2.0 J, but six subsequent episodes were cardioverted successfully with 2.0 J. VT was accelerated on three occasions in patient 3. This patient subsequently also experienced VT acceleration from a synchronized transthoracic shock. VT acceleration with appropriately timed shocks (patients 3, 8, and 11) was a contraindication to placing the cardioverter in the automatic mode.

The ventricular pacing threshold of the device was known for five patients (Nos. 1, 4, 6, 8, and 11) at the end of the follow-up period and was less than 0.1 msec pulse duration in each case. Despite good pacing thresholds, two of these patients (Nos. 8 and 11) required an increase in cardioversion energy over the follow-up period.

Tachycardia was not always terminated with the first shock, and a mean of 1.3 shocks were required to terminate each episode of tachycardia. Although charge time varied with energy delivered and time from last discharge, the mean charge time was 12.9 sec (range 2.5 to 40.3) for the first shock delivered on any

FIGURE 3. Cardioverter sensing is illustrated over the follow-up period for patients in whom monomorphic VTs were available for comparison. Patient 1 is excluded because two of the three induced tachycardias were thought to be nonclinical. In patients 2, 4, 10, and 11 the sensing for two tachycardia morphologies is illustrated. The time (msec) from the earliest QRS deflection during tachycardia to the ventricular endocardial sensing marker or cardioversion impulse is plotted for each patient on the ordinates. Abbreviations as in figure 1.
given day and 3.5 sec (range 1.1 to 7.8) for the second shock.

In all, VT was terminated on every occasion in nine patients and on eight of nine occasions in one patient, and VT was accelerated on three of five occasions in one patient. Atrial fibrillation was induced by the shock on three occasions in patient 5 and once in patient 2.

Discussion

In this study we demonstrated that low-energy synchronous intracardiac catheter cardioversion is able to terminate VT over a long period of time, requiring little change in cardioversion energy. We also noted that the mode of VT induction, VT morphology, time to cardioverter sensing of ventricular depolarization, and ventricular effective refractory periods remained stable.

Few data exist regarding stability of these electrophysiologic variables, and we are aware of no study that employed a permanently placed right ventricular endocardial lead such as ours. Schoenfeld et al. addressed long-term reproducibility of VT by performing two electrophysiologic studies in 17 patients at a mean of 18 months apart (range 2 to 42 months) in the drug-free state. They demonstrated that induced VT morphology and mode of VT induction in the 11 patients who had coronary artery disease was reproducible, but reproducibility was poor in patients without coronary artery disease. This study was limited by the fact that reproducibility was defined by only two studies and catheter position was not identical in the two studies. Without the ability to perform noninvasive programmed electrical stimulation, evaluation of VT reproducibility on multiple occasions over time is impractical.

The presence of more than one monomorphic VT in the same patient is common in this population and may add to the complexity of preimplant evaluation and subsequent follow-up. We have previously reported that changes in VT activation sequence can alter the energy required for internal cardioversion, but no distinct VT morphology or sensing time (interval from the beginning of the QRS to the onset of shock) predicted lower cardioversion thresholds. In our six patients with two VT morphologies, both tachycardias could be terminated with intracardiac cardioversion. One variable that may predict cardioversion threshold is the degree of shortening of the first VT cycle ("reset") after a subthreshold (<0.15 J) shock; greater reset

TABLE 3

<table>
<thead>
<tr>
<th>Patient</th>
<th>At implantation</th>
<th>At last follow-up</th>
<th>Spon. VT</th>
<th>Comments</th>
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<tbody>
<tr>
<td>1</td>
<td>0.2</td>
<td>0.2</td>
<td>3</td>
<td>AF once; improperly timed shock during rapid ventricular response to AF precipitated VF on one occasion</td>
</tr>
<tr>
<td>2</td>
<td>0.5</td>
<td>0.5</td>
<td>&gt;20</td>
<td>&gt;13</td>
</tr>
<tr>
<td>3</td>
<td>0.2</td>
<td>0.2-0.5</td>
<td>2</td>
<td>0 VT acceleration three times</td>
</tr>
<tr>
<td>4</td>
<td>1.75</td>
<td>1.75</td>
<td>20</td>
<td>0 AF three times</td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>1.0</td>
<td>30</td>
<td>21</td>
</tr>
<tr>
<td>6</td>
<td>0.2</td>
<td>0.2</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>0.5</td>
<td>0.5</td>
<td>12</td>
<td>5</td>
</tr>
<tr>
<td>8</td>
<td>0.5</td>
<td>2.0</td>
<td>8</td>
<td>5</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; VF = ventricular fibrillation.
predicts a greater likelihood of a cardioversion threshold less than 1 J.8

Our study group represents a selected population of patients with VT initially responsive to catheter cardioversion. All patients had stable coronary artery disease and old myocardial infarction and were receiving antiarrhythmic medications; no patient had a myocardial infarction or underwent cardiac surgery during follow-up. All patients had a relatively slow (400 to 510 msec) stable sustained VT. Thus the reproducibility of VT characteristics demonstrated in this study population must be applied with caution to patients without coronary artery disease, with more rapid ventricular tachyarrhythmias, or with more recent infarctions.

Potential complications of low-energy synchronized intracardiac cardioversion should be emphasized. A ventricular-synchronous cardioversion impulse occurring during the atrial vulnerable period can precipitate atrial fibrillation. The rapid ventricular response, if mistaken for VT, could lead to inappropriate shocks. The occurrence of VT acceleration in some patients emphasizes the need for backup defibrillation capabilities. Transient loss of sensing capabilities is a possibility when the same electrode is used for cardioversion and QRS sensing,9 as illustrated in patient 4.

In conclusion, in patients with inducible, monomorphic, relatively slow VT, the VT usually remains inducible over a long period of time, demonstrating similar VT cycle length morphology and mode of induction. QRS sensing time and ventricular effective refractory periods are also stable. The efficacy of low-energy synchronized cardioversion demonstrated at implantation usually remains stable over the follow-up period, although VT acceleration occasionally occurs. Long-term reproducibility of VT induction by means of noninvasive programmed electrical stimulation is useful for assessing long-term efficacy of implanted antitachycardia devices.

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