Nonischemic Ventricular Tachycardia
Clinical Course and Long-term Follow-up in Patients Without Clinically Overt Heart Disease

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This report describes the clinical, laboratory, and electrophysiologic features of 52 patients with ventricular tachycardia (VT) who had no clinical evidence of heart disease. The mean age of patients was 36 years, cardiovascular collapse occurred in 18 patients (35%), and exercise-related symptoms were present in 24 of 49 patients (49%). There were 20 patients with sustained monomorphic VT, 11 with incessant VT, and 21 with nonsustained VT. Abnormalities were present in 14 of 38 patients (37%) during echocardiography and in 21 of 47 patients (45%) who underwent cardiac catheterization. During baseline evaluation while patients were not receiving antiarrhythmic drugs, ambulatory monitoring and exercise testing showed an 88% and 57% incidence, respectively, of nonsustained or sustained monomorphic VT, whereas 31 of 50 patients (62%) had inducible VT (requiring an infusion of isoproterenol in 11 patients) during programmed electrical stimulation. The clinical VT (when a 12-lead electrocardiogram was available for analysis) had a left bundle branch block (LBBB) configuration in 20 of 33 patients (61%) and a right axis deviation in 17 of 33 patients (51%). The VT occurring during exercise testing and programmed electrical stimulation had the same configuration as the clinical VT in 22 of 22 patients. Three patients have received an antitachycardia pacemaker, and one patient underwent endocardial resection. Forty-eight patients (92%) were treated medically. One patient died of cancer; the remaining 47 patients were alive at a mean follow-up of 96 months after initial symptoms and 46 months after programmed electrical stimulation. We conclude that in patients without clinical evidence of heart disease, VT may be incessant, sustained, or nonsustained and that VT originates from the right ventricular outflow tract in more than 50% of patients. Although cardiac abnormalities may be found in more than 30% of patients, the exact significance of these abnormalities is unclear because of the absence of progressive changes and the excellent prognosis of this group of patients. (Circulation 1989;79:990–999)

“What seems to be important is the general myocardial integrity in young subjects who are capable of tolerating the most frequent paroxysms.”

Froment et al

Ventricular tachycardia (VT) occurring in subjects without evidence of clinical heart disease was described more than 60 years ago. Gallavardin’s initial observations, later followed by those of Parkinson and Papp, led Froment et al in 1953 to classify VT into four groups, of which two groups had apparently normal hearts: On one hand, “ventricular extrasystoles with paroxysms of ventricular tachycardia,” and on the other hand “persistent and prolonged ventricular tachycardia developing in sound hearts usually in young subjects.”

Most of these patients have an excellent prognosis, but sudden cardiac death has been reported. The purpose of this study is to present the clinical, laboratory, and electrophysiologic features of 52 patients with VT without apparent clinical heart disease. We also present the long-term follow-up of this group of patients.

Methods

Since 1977, 52 patients with VT, without clinical evidence of heart disease, have been seen at our institution. VT was defined and documented with
standard electrocardiographic criteria of at least three consecutive ventricular beats at a rate greater than 120 beats/min. Sustained monomorphic VT, present in 20 patients, was defined as VT with a well-defined QRS complex with constant axis and configuration that lasted more than 30 seconds or that caused cardiovascular collapse but beats less than 30 seconds. Incessant VT, present in 21 patients, was defined as three or more consecutive ventricular beats but lasting less than 30 seconds. Nonsustained VT, present in 22 patients, was defined as VT with constant configuration that lasted less than 11 seconds. Incessant VT, present in 11 patients, was defined as VT present for at least one half of each of three days during a continuous observation period.

All patients had their history recorded, and they underwent physical examination, 12-lead electrocardiogram, laboratory analysis, and chest radiography. M-mode or two-dimensional echocardiography or both were performed in 37 patients (71%), and cardiac catheterization was performed in 47 patients (90%). However, cardiac catheterization or echocardiography or both were performed in all but one patient, a 15-year-old boy who had a normal 12-lead electrocardiogram and a normal nuclear angiogram. None of the patients had evidence of ischemic heart disease. Only six patients did not undergo coronary angiography: five were 25 years of age or less, and one was a 62-year-old woman with a 30-year history of palpitations, a normal 12-lead electrocardiogram, and absence of chest pain or ischemic changes during a treadmill exercise test. The normal values for mean right atrial and right ventricular end-diastolic pressure were less than 8 mm Hg, and the mean values for left ventricular end-diastolic pressure were less than or equal to 12 mm Hg. Ambulatory monitoring was performed during hospitalization, and patients exercised on a treadmill according to the Bruce protocol.

Programmed electrical stimulation was performed in 50 patients (96%) with one to three tripolar or quadripolar electrode catheters, which were introduced percutaneously from the femoral vein. Thirty-two patients (64%) had a baseline study, after discontinuation of all antiarrhythmic drugs for at least five half-lives before testing. Studies were performed in the postsorptive, nonsedated state, after obtaining informed consent in all.

Intracavitary electrograms with a minimum of five scalar leads (I, II, III, V1, and V6) were recorded on a multichannel recorder at a paper speed of 100 mm/sec; filters of 30–500 Hz were used for the intracavitary electrograms. Programmed electrical stimulation was performed according to our described protocol. Ventricular stimulation was performed from the right ventricular apex; three patients also received left ventricular stimulation. All patients received single, double, or triple ventricular extrastimuli during sinus rhythm or when present pacing at 100, 120, and 140 beats/min. In addition, 11 patients received four premature ventricular extrastimuli, and in 18 patients, programmed electrical stimulation was performed during an infusion of up to 5 μg/min of isoproterenol.

Long-term follow-up studies were obtained in all patients by visit to the outpatient clinic, by re-admission to the hospital, or by contacting the patient or the patient’s physician by telephone. We have assessed recurrence of VT, antiarrhythmic therapy, and length of follow-up from initial history of symptoms, from first documentation of VT, and from the time of programmed electrical stimulation.

Statistical analysis was performed with Fisher’s exact test.

**Results**

**Clinical and Laboratory Features**

The mean age of patients at the time of diagnosing VT was 36 years (range, 10–64 years) (Table 1). Patients with incessant VT were younger (mean age, 23 years). There were 26 male and 26 female patients, and all but one patient with nonsustained VT were symptomatic, having symptoms of palpitations, dizziness, or atypical chest pain related to the occurrence of VT. Exercise-related symptoms were present in 24 of 49 patients (49%). Cardiovascular collapse or syncope occurred in at least once in 18 of 52 patients (35%) and was most frequent in the group of patients with incessant ventricular tachycardia (50%). Symptoms (palpitations and either of the above) had been present for 1 year or less in 67% of the patients. One patient had symptoms that had been present for 30 years, whereas the 16 remaining patients had symptoms that had been present between 1 and 15 years (mean, 4.7 years). All patients were considered to be in New York Heart Association Functional Class I.

A family history of sudden cardiac death was present in two patients. One patient with nonsustained VT, who had a normal two-dimensional echocardiogram and a normal right ventricular and left ventricular nuclear angiogram, was the father of a patient with arrhythmogenic right ventricular dysplasia. A history of alcohol abuse was present in three patients, and two patients had a history of systemic hypertension, which was adequately controlled. A possible remote history of myocarditis was present in four patients. One of these patients had a previous Guillain-Barré illness, and in the other three, VT was noted after an upper respiratory tract illness. Two of these three underwent a right ventricular endomyocardial biopsy, which showed dilated and hypertrophic myocardium in one and showed changes consistent with a myocarditis on an initial biopsy that were no longer present on a second biopsy in the other patient. A right ventricular endomyocardial biopsy was normal in another patient.

A cardiac physical examination was normal in all but nine patients who had a midsystolic click or a late systolic ejection murmur or both. A diagnosis of mitral valve prolapse was confirmed by echocar-
diography or left ventricular angiography. Mitral valve prolapse was not significant in any of these patients, and eight of nine patients had clinical documentation of monomorphic ventricular tachycardia with a left bundle branch block and right axis deviation, suggesting that the site of origin of VT was in the right ventricular outflow tract and not in the left ventricle,7,8,18 This site was confirmed during endocardial mapping in two patients. In one of these patients, endocardial resection of the right ventricular outflow tract completely abolished any clinical or induced VT.

The resting electrocardiogram was normal in 86% of patients; no patient had prolongation of the QT or corrected QT interval. Three patients had complete right bundle branch block, and three patients had an rSr’ pattern in V1, with a normal QRS duration. Echocardiographic findings (Table 1) were normal in 63%; apart from mitral valve prolapse, abnormalities consisted of a slight increase in right ventricular (three patients) or left ventricular (one patient) size.

During cardiac catheterization, abnormalities of intracardiac pressures, cardiac size or function (Table 1) were present in 21 of 47 patients (45%). The most common abnormality was an increase in left ventricular end-diastolic pressure, which was present before cineangiography in six patients and after cineangiography in nine patients. Only two of 36 patients had an abnormal right ventricular angiogram, both showing mild-to-moderate dilatation. In patients who underwent echocardiography and cardiac catheterization, resting values were normal in 50% of patients excluding mitral valve prolapse in six patients.

The left ventricular ejection fraction exceeded 50% in all but one patient, who had an ejection fraction of 44%, secondary to the underlying incessant ventricular tachycardia. In this patient,
left ventricular function returned to normal after surgical ablation of VT.

Noninvasive Testing

Ambulatory monitoring, performed while the patients were not receiving antiarrhythmic drugs, revealed Lown’s grade IV B ventricular arrhythmias in 26 of 42 patients (62%) and sustained VT in 11 of 42 (26%) (Table 2). During exercise testing, while not receiving antiarrhythmic drugs, Lown’s grade IV B ventricular arrhythmias were present in 20 of 42 patients (46%), but a sustained VT occurred in only four patients. During drug treatment, Lown’s grade IV B ventricular arrhythmias occurred in 10 of 33 patients (30%) who underwent ambulatory monitoring compared with 37 of 42 patients (88%) who were not receiving drug therapy (p<0.0002). During exercise testing, Lown’s grade IV B ventricular arrhythmias occurred in 24 of 42 patients (57%) who were not receiving treatment compared with four of 30 patients (13%) who were receiving antiarrhythmic drugs (p<0.0001).

Programmed Electrical Stimulation

VT was induced in 31 of 50 patients (62%) (Table 2). A sustained monomorphic VT was induced primarily in patients who had clinically documented sustained monomorphic VT. An infusion of up to 5 μg/min isoproterenol resulted in sustained monomorphic VT in two patients and nonsustained VT in nine patients, and VT in seven patients remained noninducible. The addition of a fourth premature ventricular stimulus to the stimulation protocol in seven patients resulted in nonsustained VT in two patients, and VT in five patients remained noninducible. Two patients had VT induced during incremental atrial pacing. A total of 10 restudies were performed on patients receiving antiarrhythmic drugs (including two studies on patients receiving amiodarone). Sustained monomorphic VT was induced in three patients, and VT in seven patients was noninducible.

Morphologic Features of Clinical and Induced Ventricular Tachycardia

The clinical VT had a left bundle branch block configuration in 61% of patients (20 of 33) (Table 3). The mean rate of the spontaneous VT was 183 beats/min. The provoked VT, either by exercise testing or programmed electrical stimulation, had a left bundle branch block configuration in 45% of patients, and the mean rate was 137 beats/min. Of the 22 patients in whom analysis of both the clinical and induced VT was possible, the same axis occurred in 54%, but concordance of bundle branch block configuration occurred in 100% of patients. Most patients with incessant VT had a left bundle branch block configuration of VT, whereas patients with sustained VT had an equal distribution of left and right bundle branch block configuration of VT.

Treatment and Follow-up

The mean follow-up from the initial history of symptoms was 96 months (119 months in patients with sustained VT, 64 months in patients with incessant VT, and 91 months in patients with non-sustained VT). The mean follow-up from first documentation of VT was 65 months (68 months in patients with sustained VT, 60 months in patients with incessant VT, and 63 months in patients with non-sustained VT). From the time of programmed electrical stimulation, the mean follow-up was 46

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**Table 2. Results of Noninvasive and Invasive Testing**

<table>
<thead>
<tr>
<th></th>
<th>Ambulatory monitoring (≥IV B Lown class)</th>
<th>Exercise testing (≥IV B Lown class)</th>
<th>Programmed electrical stimulation (all baseline studies)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No antiarrhythmic drugs</td>
<td>Amiodarone</td>
<td>No antiarrhythmic drugs</td>
</tr>
<tr>
<td>Sustained monomorphic VT</td>
<td>20</td>
<td>9/13 (69%)</td>
<td>5/9 (55%)</td>
</tr>
<tr>
<td>Incessant VT</td>
<td>11</td>
<td>11/11 (100%)</td>
<td>3/9 (33%)</td>
</tr>
<tr>
<td>Nonsustained VT</td>
<td>21</td>
<td>2/6 (33%)</td>
<td>0/4</td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>37/42 (88%)</td>
<td>2/11 (18%)</td>
</tr>
</tbody>
</table>

*All numbers are number of patients unless otherwise specified.
Amiodarone was administered at a dosage of 200 mg/day.
VT, ventricular tachycardia; SPVT, sustained polymorphic VT; Iso, isoproterenol infusion, up to 5 μg/min.
When comparing with no antiarrhythmic drugs, * p<0.0002, †p<0.0001.*
months (48 months in patients with sustained VT, 32 months in patients with incessant VT, and 52 months in patients with nonsustained VT).

Three patients received a patient-activated burst antitachycardia pacemaker. Only one of these patients received antiarrhythmic drug therapy (proprafenone). Documented recurrence of VT has occurred in two of these patients after dislodgement of the endocardial lead, but both have had an uneventful course after reposition of the lead. The only patient who has been operated on, by the group of Dr. Josephson in Philadelphia, has had a total cure after an extensive endocardial resection with cryoablation of the septal side of the right ventricular outflow tract. Now, 48 months after surgery, the patient has been free of any symptoms or recurrence of VT.

All remaining 48 patients were initially treated medically: One patient died of cancer. Medical survival is 100% in all others. Most patients were treated with either sotalol or a class IC antiarrhythmic drug (48%), and amiodarone was given to 19% of patients. Two patients were treated with calcium channel blockers. Recurrence of documented and symptomatic VT has occurred in 15 patients (29%) (Table 4). Although the numbers are small, in the group of patients with recurrence of VT who were receiving antiarrhythmic drugs, Lown's grade IV B ventricular arrhythmias during noninvasive testing were significantly more frequent than in the patients who have been free of recurrence ($p<0.002$). When only patients are considered rather than the total results of noninvasive studies, 40% of patients with less than IV B Lown ventricular arrhythmias during ambulatory monitoring or exercise testing or both had recurrence of VT compared with 68% in the group without recurrence (not significant). However, 19% of patients who have not had docu-

### Table 3. Morphologic Features of Ventricular Tachycardia*

<table>
<thead>
<tr>
<th>Type of VT</th>
<th>LBBB</th>
<th>RBBB</th>
<th>Rate† (beats/min)</th>
<th>LBBB</th>
<th>RBBB</th>
<th>Rate† (beats/min)</th>
<th>Concordance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RAD LAD</td>
<td>RAD LAD</td>
<td></td>
<td>RAD LAD</td>
<td>RAD LAD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sustained monomorphic VT</td>
<td>20 7 2 5 4 178 (range, 125–214)</td>
<td></td>
<td>6 2 3 6 184 (range, 120–333)</td>
<td></td>
<td></td>
<td>10/17 17/17 (59%) (100%)</td>
<td></td>
</tr>
<tr>
<td>Incessant VT</td>
<td>11 8 0 3 0 192 (range, 150–222)</td>
<td></td>
<td>2 0 0 1 206 (range, 200–222)</td>
<td></td>
<td></td>
<td>2/3 3/3 (66%) (100%)</td>
<td></td>
</tr>
<tr>
<td>Nonsustained VT</td>
<td>21 2 1 1 0 178 (range, 150–214)</td>
<td></td>
<td>2 0 0 1 209 (range, 167–300)</td>
<td></td>
<td></td>
<td>0/2 2/2 (100%)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>52 17 3 9 4 183 (range, 125–222)</td>
<td></td>
<td>10 2 3 8 188 (range, 120–333)</td>
<td></td>
<td></td>
<td>12/22 22/22 (54%) (100%)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>VT, ventricular tachycardia; LBBB, left bundle branch block; RBBB, right bundle branch block; RAD, right axis deviation; LAD, left axis deviation.</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Includes only patients who had a 12-lead electrocardiogram during VT.</td>
</tr>
<tr>
<td>†Provoked during exercise testing or programmed electrical stimulation.</td>
</tr>
<tr>
<td>‡Rate (beats/min) with or without antiarrhythmic drugs.</td>
</tr>
</tbody>
</table>

### Table 4. Treatment and Clinical Course During Follow-up

<table>
<thead>
<tr>
<th>Type of VT</th>
<th>Documented recurrence of VT</th>
<th>No documented recurrence of VT (excludes palpitations)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patients (n)</td>
<td>Studies with &lt;IV B Lown grade during AAD therapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amb-M</td>
</tr>
<tr>
<td>Sustained monomorphic VT</td>
<td>7 0/2 0/2 1/3</td>
<td>13 6/7 6/7 3/3</td>
</tr>
<tr>
<td>Incessant VT</td>
<td>5 1/2 2/4 1/3</td>
<td>6 4/5 4/5 1/1</td>
</tr>
<tr>
<td>Nonsustained VT</td>
<td>3 1/2 0/2 0/1</td>
<td>18 6/10 7/9 3/4</td>
</tr>
<tr>
<td>Total</td>
<td>15* (29%)</td>
<td>2/6 (33%) 2/8 (25%) 2/7 (28%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Amb-M, ambulatory monitoring; Ex-T, exercise test; PES, programmed electrical stimulation; AAD, antiarrhythmic drugs; PM, permanent pacemaker; A, amiodarone (all dosages ≤200 mg/day).</th>
</tr>
</thead>
<tbody>
<tr>
<td>*At last follow-up: A=two patients; other AAD=seven; no AAD=three; PM=three.</td>
</tr>
<tr>
<td>†At last follow-up: A=six; other AAD=20; no AAD=10; one death due to cancer.</td>
</tr>
<tr>
<td>‡$p&lt;0.002$ includes all Amb-M and Ex-T studies while patients received AAD treatment; $p=NS$, represents patients in whom Amb-M or Ex-T or both showed less than IV B Lown grade.</td>
</tr>
</tbody>
</table>
mented recurrence of VT continue to have palpitations and may continue to have VT. At last follow-up, 25% of patients were not receiving antiarrhythmic drug treatment (four patients with a history of sustained monomorphic VT, eight patients with nonsustained VT, and one patient with incessant VT who had a spontaneous cure). Finally, when comparing patients who had no cardiac abnormalities with those who had various alterations of cardiac function, we found no difference in the clinical course or in the long-term outcome of both groups of patients.

Discussion

This study describes the clinical, laboratory, and electrophysiologic features of a group of children and adults with VT who have no clinical evidence of heart disease. The episodes of documented VT were monomorphic and sustained or incessant in 60% of patients, and cardiovascular collapse occurred in 35%. The repetitive and paroxysmal nature of VT in this group of patients is well known, but the prognosis, though generally excellent, is guarded because of reports of sudden cardiac death.

Significance of Underlying Cardiac Findings

Documentation of subclinical evidence of cardiac disease could indeed be important in relation to the potential morbidity of idiopathic VT. Several workers, from data obtained from echocardiography, cardiac catheterization, endomyocardial biopsy, or autopsy, have found potential contributing factors to explain the ventricular arrhythmias. Evidence of cardiac dysfunction was present at cardiac catheterization in 45% of our patients. Although we believe that these abnormalities may be important, to fully attribute VT to these hemodynamic or angiographic changes is difficult because 1) the abnormalities are most often subtle, and we found no characteristic features between groups of patients with or without cardiac dysfunction, 2) these abnormalities, at least in some patients, could be the result rather than the cause of repeated and paroxysmal episodes of VT, and 3) no patient has developed a clinically apparent cardiomyopathic process during a mean follow-up of 8 years since the initial symptoms (although tachycardia-induced left ventricular dysfunction occurred in one patient before surgical ablation of VT). However, echocardiographic studies were not done systematically in all patients during follow-up because of the excellent outcome in most patients and the absence of any clinical symptoms.

The finding of mitral valve prolapse in association with a right ventricular outflow tract ventricular tachycardia in eight of our patients is of interest. It is important to emphasize that mitral valve prolapse
FIGURE 2. 12-lead electrocardiogram from a patient with paroxysmal (repetitive) ventricular tachycardia. A fusion beat is followed by a six beat run of ventricular tachycardia, which has a left bundle branch block configuration and a vertical axis.

was mild and was not associated with electrocardiographic changes or with polymorphic VT, which have been reported in patients with mitral valve prolapse and ventricular arrhythmias.\textsuperscript{33-36} However, mitral valve prolapse may be associated with abnormal tension on papillary muscles,\textsuperscript{30} autonomic dysfunction with excess catecholamines,\textsuperscript{37,38} or even a cardiomyopathic process.\textsuperscript{33,39,40} These or other abnormalities could be responsible for the development of a right ventricular outflow tract tachycardia in patients with mitral valve prolapse, but we found no evidence to support this hypothesis.

FIGURE 3. Resting 12-lead electrocardiogram from a patient who had sustained monomorphic ventricular tachycardia. The ventricular tachycardia has a left bundle branch block configuration with a normal axis and a rate of 167 beats/min.
Electrophysiologic Features

Our group of patients present several of the characteristics that have been described in idiopathic VT. Repetitive monomorphic VT occurred in our patients with incessant VT and in some patients with sustained or nonsustained VT (Figure 1). Gallavardin and Parkinson and Papp originally described this type of VT. Coumel et al have clearly shown that the spontaneous extrasystoles are determined by the preceding mean heart rate and the length of the RR cycle preceding the initial extrasystole (adrenergic and rate dependence). They have also shown the relative dependence of the repetitive activity on long coupled extrasystoles. These observations prompted those investigators to suggest that the mechanism of VT was due to triggered activity or modulated parasystole or both. Clinically, the presence of exercise-related symptoms in 49% of patients, as well as the 62% incidence of inducible VT, would be compatible with triggered activity.

Sustained idiopathic monomorphic VT usually has a left bundle branch block configuration (Figures 2 and 3). The main differential diagnosis in these patients with VT and a left bundle branch block configuration is arrhythmogenic right ventricular dysplasia (ARVD). In the latter patients, VT may have either a left axis or right axis deviation compared with the predominance of right axis deviation in idiopathic VT. Patients with ARVD are typically young males with T wave changes in the right precordial leads and distinctive right ventricular abnormalities during echocardiography or right ventricular angiography. Whether or not some of our patients or others with mild right ventricular abnormalities represent forms of ARVD is unclear. However, the benign nature of these patients, the good control of VT, and the absence of right ventricular changes does not indicate ARVD.

Sustained monomorphic VT with a right bundle branch block configuration (Figure 4) suggesting a left ventricular site of origin of VT, has been postulated to originate in or close to the left anterior or posterior fascicles. This type of VT frequently responds to calcium channel blockers and therefore may arise from slow channel-dependent mechanisms operating at the level of the left intraventricular conduction system. However, in most of our patients, this type of VT was treated with sotalol or amiodarone and had excellent results.

Treatment and Prognosis

The common feature of VT described above is the generally excellent outcome and the occasional unpredictable course of the patients. Although patients may have had symptoms for years, their condition may suddenly deteriorate, and they may develop incessant VT. Others, who have been symptomatic or incapacitated, may have a spontaneous cure and others may not require antiarrhythmic drugs. However, there have been reports of sudden cardiac death in patients with idiopathic VT especially in patients not receiving antiarrhythmic drug treatment. Indeed, we believe that most, if not all patients, with
symptomatic idiopathic VT require antiarrhythmic drug therapy, especially those with sustained or incessant VT. Only four of 41 of our patients with such VT are not receiving treatment (one patient with incessant VT has had a spontaneous cure, and in three others, no recurrence of VT has occurred during a mean follow-up of 12 months). In our group of patients, class IA agents were practically never used. Most patients were treated with class IC or class III antiarrhythmic agents, and these resulted in control of VT in most patients.

Recurrence of VT occurred in 29% of our patients; noninvasive testing may identify a subset of patients that may be more resistant to treatment and should be used to assess the clinical efficacy of antiarrhythmic drug treatment. Although programmed electrical stimulation may offer greater insight into the pathogenesis and origin of the VT, its usefulness in guiding therapy is questionable because of the low incidence of induced sustained VT (24%) in our patients and the overall good response to antiarrhythmic drug therapy in these patients.

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