Nonclinical Polymorphic Ventricular Tachycardia
Induced by Programmed Cardiac Stimulation:
Incidence, Mechanisms and Clinical Significance

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SUMMARY We analyzed the morphology and rate of ventricular tachycardia induced by programmed cardiac stimulation in 18 patients with recurrent sustained ventricular tachycardia who had only one type of ventricular tachycardia during spontaneous episodes. In nine of 18 patients, we induced only one type of ventricular tachycardia, with a morphology and rate similar to those noted during spontaneous ventricular tachycardia. In the other nine patients, the induced ventricular tachycardia was either different from spontaneous ventricular tachycardia (two patients) or was polymorphic (seven patients). The multiple morphologies were induced in addition to those similar to spontaneous ventricular tachycardia. Seven of these nine patients had ischemic heart disease. Only one patient had a left ventricular aneurysm. The morphology and rate of induced tachycardia could be changed by changing the basic cycle length (two patients), the method of initiation of tachycardia (one patient), and by introducing paced ventricular beats during tachycardia (four patients). Our data suggest that ventricular tachycardia induced by programmed cardiac stimulation is nonclinical in 50% of patients with recurrent ventricular tachycardia. The clinical course and prognosis of patients with polymorphic ventricular tachycardia compared with those without polymorphic tachycardia are not known.

INCREASING RELIANCE is being placed on the information obtained from electrophysiologic studies and acute drug testing in designing the treatment for recurrent sustained ventricular tachycardia.1,14 Recent investigations have suggested that such studies may be useful in identifying the patients with recurrent ventricular tachycardia suitable for specific modes of pharmacologic, pacemaker and surgical treatment.1,10

This approach to the treatment of recurrent ventricular tachycardia is based on the premise that programmed cardiac stimulation in the laboratory reliably reproduces the QRS pattern (morphology) and rate of spontaneous ventricular tachycardia. However, the relation between the characteristics of induced and spontaneous ventricular tachycardia in the same patient is not always consistent.1,4,7,9,11 Fisher et al. reported that programmed cardiac stimulation duplicated the morphology and rate of spontaneous ventricular tachycardia in 95% of patients with ventricular tachycardia.1 Horowitz and co-workers obtained similar results during chronic serial electrophysiologic studies and did not mention any cases in which the ventricular tachycardia initiated by stimulation was different from the spontaneous ventricular tachycardia. In contrast, Mason and Winkle reported that the induced ventricular tachycardia was different from spontaneous ventricular tachycardia in five of their 31 patients (16%) and the tachycardia morphology and rate could be altered by paced ventricular beats introduced during tachycardia in nine other patients (32%).

Josephson et al. reported induction of two or more morphologically distinct ventricular tachycardias in 14 of 26 patients with recurrent sustained ventricular tachycardia, and attributed it to variable exit sites and/or ventricular activation from a single tachycardia circuit. It appeared to us that the induction of ventricular tachycardia with single or multiple (polymorphic) morphologies could be caused by the possible differences in patient selection, type of applied stimulation techniques or both.

To study these problems and determine the incidence, possible mechanisms and clinical significance of polymorphic ventricular tachycardia, we analyzed the morphology and rate of ventricular tachycardia induced by programmed cardiac stimulation in 18 patients with recurrent sustained ventricular tachycardia. In nine of these 18 patients, the morphology and rate of induced tachycardia were similar to those noted during spontaneous ventricular tachycardia. In the other nine patients, the induced tachycardia was either different from spontaneous ventricular tachycardia or had multiple morphologies (in addition to those similar to spontaneous ventricular tachycardia). The observations made in these nine patients in whom the induced ventricular tachycardia was either different from spontaneous ventricular tachycardia or had multiple morphologies are the subject of this report.

Methods

Nine patients with recurrent sustained ventricular tachycardia referred for diagnostic and therapeutic evaluation were studied. Pertinent clinical data are presented in table 1. All patients had spontaneous episodes of ventricular tachycardia that were recurrent and sustained for minutes to hours and required termination by drugs or DC cardioversion. In all patients, at least three spontaneous episodes of ventricular tachycardia were recorded. Six or more
episodes were recorded in four patients and four or more episodes in three patients. In each of nine patients, only one ventricular tachycardia pattern was recorded during spontaneous episodes. However, the rate of tachycardia varied by 5–15 beats/min. Ambulatory electrocardiographic monitoring for one or more 24-hour periods was performed in all patients. In four patients in whom the arrhythmia could be recorded during monitoring, the morphology and the rate of tachycardia were similar to those of previously recorded episodes. The diagnosis of ventricular tachycardia was made using standard electrocardiographic criteria and confirmed in all cases by intracardiac recordings during the arrhythmia. No patient demonstrated Wolff-Parkinson-White pattern. Patients 1, 2, 4, 5, 7, 8 and 9 underwent cardiac catheterization, including coronary arteriography, which revealed occlusive coronary artery disease and low ejection fraction ($\leq 0.4$) and/or left ventricular wall motion abnormalities. Only patient 2 had a ventricular aneurysm. In patients 3 and 6, in whom cardiac catheterization was not performed, echocardiography and radionuclide angiography revealed left ventricular wall motion abnormalities and depressed ejection fraction (0.41 and 0.37, respectively). No discrete ventricular aneurysm was detected.

Electrophysiologic studies were performed in the postabsorptive, nonsedated state after an informed consent was obtained. All patients were in sinus rhythm at the time of study. All cardiac drugs were discontinued at least 48 hours before the study. No premedication was administered. Bundle of His electrograms were recorded as previously described using a #7 tri- or quadripolar electrode catheter, which was introduced percutaneously into the right femoral vein and fluoroscopically positioned in the region of the tricuspid valve. A #6 quadripolar electrode catheter was introduced into an antecubital vein and advanced to the high right atrium for pacing and recording the local electrogram. An additional #6 bi- or quadripolar electrode catheter was percutaneously introduced into a separate antecubital vein and positioned at the right ventricular apex for ventricular stimulation and recording the local electrogram. Atrial and ventricular stimulation were performed using a programmable digital stimulator that delivered rectangular pulses of 1.5 msec in duration at twice diastolic threshold. Intracardiac electrograms and ECG leads I, II and V$_1$ were simultaneously displayed on multichannel oscilloscope and recorded on paper moving at a speed of 100 mm/sec.

**Definition of Terms**

- $S_1S_2$: basic paced cycle length;
- $S_2S_3$: coupling interval from eighth beat of a basic cycle length to the first premature beat;
- $S_3$: second premature beat delivered after $S_2$ during a basic cycle length, $S_2S_3$;
- $S_3S_4$: coupling interval between the first and second premature stimuli;
- $V_1V_2V_3$: ventricular depolarization produced by $S_1$, $S_2$ and $S_3$, respectively;
- **Tachycardia zone**: range of $S_2S_3$ or $S_3S_4$ coupling.

### Table 1. Clinical and Electrophysiologic Data

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Cardiac diagnosis</th>
<th>Resting ECG</th>
<th>Spontaneous VT Morphology</th>
<th>CL (msec)</th>
<th>AH</th>
<th>HV</th>
<th>Induced VT Morphology</th>
<th>CL (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>Cardiomyopathy</td>
<td>Normal</td>
<td>LBBB, LAD</td>
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<td>LBBB, LAD</td>
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<tr>
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<td>58</td>
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<td>Old DMI, IVCD</td>
<td>RBBB, RAD</td>
<td>370</td>
<td>95</td>
<td>69</td>
<td>LBBB, RAD</td>
<td>260</td>
</tr>
<tr>
<td>3</td>
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<td>LVH, IVCD</td>
<td>LBBB, LAD</td>
<td>240</td>
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<td>65</td>
<td>LBBB, LAD</td>
<td>260</td>
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<td>LBBB</td>
<td>RBBB, RAD</td>
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<td>95</td>
<td>65</td>
<td>LBBB, LAD</td>
<td>280</td>
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<tr>
<td>5</td>
<td>57</td>
<td>MV prolapse</td>
<td>LVH, IVCD</td>
<td>LBBB, LAD</td>
<td>280</td>
<td>120</td>
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<td>260</td>
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<td>Old AWMI, RBBB, LAH</td>
<td>LBBB, LAD</td>
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<td>90</td>
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<td>LBBB, LAD</td>
<td>240</td>
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</tbody>
</table>

**Abbreviations:** VT = ventricular tachycardia; ASHD = atherosclerotic cardiovascular disease; Vent An = ventricular aneurysm; S/P = status post; MV = mitral valve; DMI = diaphragmatic myocardial infarction; IVCD = intraventricular conduction defect; LVH = left ventricular hypertrophy; AWMI = anterior wall myocardial infarction; RBBB = right bundle branch block; LBBB = left bundle branch block; LAH = left anterior hemiblock; LAD = left-axis deviation; RAD = right-axis deviation; CL = cycle length.
intervals during which ventricular tachycardia could be initiated.

Effective refractory period (ERP) of ventricle: the longest S3S4 interval at which S4 does not evoke a ventricular response.

Left and right bundle branch block patterns: These are defined using the standard criteria.16

Right- and left-axis deviation: For the purposes of this study, a predominantly negative deflection in lead I is considered suggestive of right-axis deviation and a positive deflection in lead I with a predominantly negative deflection in lead II is considered suggestive of left-axis deviation. The following protocol of programmed stimulation was utilized:

1. Incremental atrial pacing (maximum rates of 130 to 200 beats/min).
2. Premature atrial stimuli during sinus rhythm and/or atrial pacing.
3. Incremental ventricular pacing with abrupt cessation after each increment of 10 beats/min (maximum rates 140–220 beats/min).
4. Ventricular extrastimulus testing during sinus rhythm and ventricular pacing at one or more cycle lengths. If scanning with a single ventricular premature stimulus (S4) from late diastole to ventricular refractoriness failed to induce ventricular tachycardia, the S4 was moved to 50 msec outside the refractory period and scanning with a second premature stimulus (S4) was then done. S4 was introduced starting with an S1S4 interval equal to the S1S2 interval. S2S4 was then progressively shortened and when S4 encountered ventricular muscle refractoriness, the S1S2 interval was decreased until S4 could elicit a ventricular response V3, or ventricular tachycardia. This protocol was continued until both S2 and S4 failed to evoke ventricular responses.
5. After the initiation of ventricular tachycardia, timed single, double or triple ventricular stimuli or short bursts of rapid ventricular pacing were used to terminate the arrhythmia.

Results

The clinical and electrophysiologic data for each patient are presented in table 1.

Initiation of Ventricular Tachycardia

Ventricular tachycardia could be reproducibly induced in all patients by one or two critically timed ventricular premature depolarizations. A reproducible tachycardia zone of 20–110 msec was observed for each induced ventricular tachycardia. For ventricular tachycardias that were similar to the spontaneous tachycardia, the tachycardia zone ranged from 40–110 msec, and for those which were different, the tachycardia zones were narrow and ranged from 20–50 msec. A single ventricular premature depolarization initiated the tachycardia in patients 2, 5, 6, 7 and 8 and two ventricular premature depolarizations were required to initiate the tachycardia in patients 1, 3, 4 and 9. In patients 2 and 8, ventricular tachycardia could be initiated by ventricular premature depolarizations delivered during sinus rhythm and in the remaining patients only by ventricular premature depolarizations delivered during ventricular pacing. In patients 6, 7 and 8, ventricular tachycardia could also be initiated by short bursts of ventricular pacing at rates of 150–180 beats/min.

QRS Morphology and Rate of Ventricular Tachycardia

Eighteen distinct ventricular tachycardia patterns could be induced by stimulation techniques (table 1). Two patterns of ventricular tachycardia were induced in five patients and three patterns in two (patients 4 and 7). In each of these seven patients, one pattern of induced ventricular tachycardia was similar to that of the spontaneous ventricular tachycardia. In patients 8 and 9, one pattern of ventricular tachycardia different from that of the spontaneous ventricular tachycardia could be induced (fig. 1). In patients 1–7, the induced tachycardias had both right and left bundle branch block pattern.

Method of Initiation of Tachycardia and Tachycardia Morphology and Rate

In two of six patients (nos. 4 and 5) in whom ventricular tachycardia could be induced during pacing at different cycle lengths, two morphologically distinct ventricular tachycardias could be induced at shorter cycle lengths (fig. 2). In one of three patients (no. 6) in whom ventricular tachycardia could be induced both by ventricular premature depolarizations and rapid ventricular pacing, the morphology of ventricular tachycardia induced by ventricular premature depolarizations was different from that induced by rapid ventricular pacing (fig. 3A and B). In this patient, the rate of tachycardia induced by ventricular pacing was faster than that induced by ventricular premature depolarizations (fig. 3B). In patient 1, two patterns of ventricular tachycardia were induced, each by two ventricular premature depolarizations delivered at different coupling intervals during the same basic cycle length. In patients 8 and 9, in whom the morphology of induced ventricular tachycardia was different from that of spontaneous tachycardia, changes in the methods of initiation or termination of ventricular tachycardia did not result in changes in morphology. No patient showed spontaneous variations in morphology of induced tachycardia.

Ventricular Stimulation During Tachycardia and Its Effect on Tachycardia Morphology and Rate

Critically timed ventricular premature depolarizations and/or short bursts of ventricular pacing resulting in ventricular capture terminated the tachycardia in all patients. In patients 1–6, 8 and 9, the tachycardia could be consistently terminated by one or two critically timed ventricular premature depolarizations. In patient 7, ventricular pacing at a rate faster than that of tachycardia was required to
Figure 1. Induction of nonclinical ventricular tachycardia (VT) (patient 8). The records are organized in each panel from top to bottom: ECG leads I, II, V1, and electrograms from the high right atrium (HRA) and the His bundle (HBE). The time lines (T) are 50 msec apart. During sinus rhythm, premature ventricular depolarizations (S) delivered at a coupling interval of 250 msec initiate a VT with left bundle branch block and left-axis deviation pattern and a cycle length (CL) of 350 msec. Termination of VT with a single ventricular premature depolarization is shown in the bottom panel. ECG strips from leads I, II, and V1, recorded during one of the spontaneous episodes of VT are shown to the right. The tachycardia has a right bundle branch block and right-axis deviation pattern and a CL of 380 msec.

Figure 2. Basic pacing rate and the ventricular tachycardia (VT) morphology and rate (patient 4). The records are organized as in figure 1. The time lines (T) are 40 msec apart. During a ventricular paced cycle length (CL) (S1S3) of 650 msec (top panel), two ventricular premature depolarizations (VPDs), S2 and S3, delivered at coupling intervals of 270 and 230 msec, respectively, initiate a VT with a right bundle branch block and right-axis deviation pattern and a CL of 450 msec. Note that the first QRS complex of tachycardia is different from that of subsequent beats. In the bottom panel, during a ventricular paced CL of 550 msec, two VPDs (S4 and S5) delivered at coupling intervals of 270 and 240 msec, respectively, initiate a VT with a left bundle branch block and left-axis deviation pattern and a CL of 270 msec.
terminate the tachycardia. Single ventricular premature depolarizations delivered during tachycardia and resulting in apparent ventricular capture but failing to terminate or alter the rate and morphology of the tachycardia were followed by less-than-compensatory pauses in all patients (fig. 4A). However, in patients 2, 3, 4 and 7, in whom two or more paced ventricular beats introduced during tachycardia failed to terminate the tachycardia, the morphology and the rate of tachycardia were grossly altered. The interval between the last ventricular premature depolarization and the first complex of ventricular tachycardia was longer than one tachycardia cycle length but shorter than two cycle lengths (fig. 4B).
The Activation of Proximal His-Purkinje Conduction System During Tachycardia

Bundle branch reentry (V3 phenomenon) occurred in six patients. However, V3 phenomenon was not causally related to the initiation of ventricular tachycardia in any patient because the initiation of tachycardia was independent of the presence of retrograde His-Purkinje conduction delay and the QRS complexes of tachycardia were not consistently preceded by the His bundle deflections. In patient 2, His bundle deflections appeared consistently after the onset of each QRS complex of tachycardia (figs. 4A and B).

Safety of the Procedure

During attempted termination by ventricular pacing or ventricular premature depolarizations, acceleration of ventricular tachycardia occurred in patients 2 and 7. In both patients, acceleration of tachycardia was associated with rapid hemodynamic depolarization requiring DC cardioversion. In patient 4, ventricular tachycardia induced by two ventricular premature depolarizations was rapid enough to cause hemodynamic deterioration necessitating DC cardioversion.

Follow-up

All patients have been followed for periods of 1–29 months. Patient 2, in whom tachycardia was relieved by aneurysmectomy, died 4 months later of cerebral hemorrhage. Patient 7 died suddenly after a follow-up of 1 month. In the remaining seven patients, who were treated with different antiarrhythmic drugs, episodes of ventricular tachycardia recurred. In three of these seven patients, the recurrence was related to non-compliance with drug regimen. Patients 1, 5 and 8 had only one episode and four patients two or more

FIGURE 4. Patient 2. (A) Ventricular stimulation during tachycardia. The records are organized similarly to previous figures. Ventricular tachycardia (VT) with a left bundle branch block (LBBB) and right-axis deviation pattern is present (first four complexes). The tachycardia cycle length (CL) is 515 msec and 1:1 retrograde conduction to the atria is present. A single ventricular premature depolarization (VPD) (S), introduced during tachycardia at a coupling interval of 420 msec, is followed by a less-than-compensatory pause (650 msec), and tachycardia continues unaltered. Note the consistent activation of the His bundle (H) simultaneous with the ventricular activation. (B) Ventricular stimulation during tachycardia. The ECG leads and intracardiac electrograms and time lines are organized as in (A). During VT with a left bundle branch block and right-axis deviation pattern and a CL of 500 msec (first four complexes), two VPDs delivered at coupling intervals of 460 and 390 msec alter the tachycardia morphology and rate. The tachycardia now has right bundle branch block (RBBB) configuration and the CL is 400 msec. The interval between the second VPD and the first QRS complex of RBBB tachycardia is 560 msec. Consistent activation of the His bundle is present during both LBBB and RBBB pattern tachycardias. One-to-one retrograde conduction to the atria is present during LBBB pattern tachycardia. However, atrioventricular dissociation is present during the first three complexes of RBBB pattern tachycardia.
episodes. The morphology and rate of these tachycardias were similar to those of previously recorded spontaneous episodes of ventricular tachycardia.

Discussion

Mason and Winkle introduced the term nonclinical ventricular tachycardia when the morphology of induced tachycardia differed from that of spontaneous ventricular tachycardia. Josephson et al. used the term polymorphic ventricular tachycardia to describe multiple distinct morphologies of ventricular tachycardia. In this report, the term polymorphic tachycardia is used to designate the presence of more than one bundle branch block pattern or different axis deviations with same bundle branch block pattern. Invariably, the changes in morphology were accompanied by changes in tachycardia rate.

Incidence

In this study, ventricular stimulation was performed in all patients only from the right ventricular apex. We do not know whether stimulation at other sites within the right ventricle or left ventricle would have induced ventricular tachycardia with additional morphologies. In our study, programmed cardiac stimulation induced nonclinical ventricular tachycardia in 50% of the patients with recurrent ventricular tachycardia. This incidence is similar to that reported by Mason and Winkle and Josephson et al. However, in contrast to the observations of Josephson and co-workers, none of our patients exhibited morphologic variations during spontaneous tachycardia. We have considered the possibility that in some of our patients spontaneous ventricular tachycardias with different morphologies were not detected clinically. However, this appears unlikely for three reasons: (1) In no patient were these tachycardias self-terminating; (2) in all patients the arrhythmias were symptomatic; and (3) all patients could recognize the arrhythmia.

Mechanisms

The precise mechanisms by which nonclinical ventricular tachycardia was induced in some patients and ventricular tachycardia similar to the spontaneous tachycardia in others are uncertain. However, two possibilities should be considered: (1) effects of stimulation techniques applied for the induction of ventricular tachycardia and (2) differences in patient population. Procedure effect alone does not explain our results because the same stimulation protocol was used in all patients. Furthermore, in patients in whom only one type of ventricular tachycardia was induced, changes in the method of initiation did not alter the morphology or the rate of tachycardia.

Single vs Multiple Reentrant Circuits

If we assume reentry as the mechanisms of arrhythmia and attribute the induction of multiple ventricular tachycardia patterns to differences in patient groups, then we expect such patients to have several concealed or dormant but a single clinically manifest reentry circuit or a single reentrant circuit with a change in the activation sequence of myocardium. The changes in conduction properties of ventricular muscle and/or the His-Purkinje system caused by changes in the method of initiation of tachycardia may change the ventricular activation sequence, resulting in multiple ventricular tachycardia patterns. The ventricular premature depolarizations introduced during tachycardia may capture a certain portion of the ventricles, rendering that portion refractory at a time when it was previously activated by the reentrant impulse. The resulting spatial and temporal differences in ventricular refactoriness may change the sequence of ventricular activation and consequently the tachycardia morphology. The ability of two or more ventricular premature depolarizations introduced during tachycardia to alter the tachycardia morphology when a single ventricular premature depolarization failed may be explained in the following manner: A single ventricular premature depolarization failed because the impulse was unable to reach or penetrate the reentrant circuit, but when two ventricular premature depolarizations were introduced, the first shortened the refractory period of the ventricle, permitting the second ventricular premature depolarization to penetrate the reentrant circuit.

Although the induction of nonclinical polymorphic ventricular tachycardia may be explained by a single reentrant circuit with variable ventricular activation patterns, we cannot exclude the possibility of activation by different stimulation techniques of concealed sites of reentry remote from the site of manifest reentry. The paced ventricular beats introduced during tachycardia may have terminated the tachycardia arising from one site while initiating it at another site. The tachycardia arising from one site of reentry could enter and interrupt potential reentry from another site by colliding with the returning wave front so that reentry at one site may mask reentry at another site. Possibly, more than one site of reentry may exist in diseased ventricular myocardium with large akinetic or dyskinetic areas and distorted conduction system.

Endocardial ventricular mapping studies by Josephson et al. suggested a single reentrant circuit with variable exit sites and ventricular activation patterns as the mechanism of polymorphism. Endocardial ventricular mapping was not performed in any of our patients.

In contrast to the observations of Josephson et al., who found left ventricular aneurysm in 11 of 14 patients with pleomorphic ventricular tachycardia, left ventricular aneurysm was present in only one patient in our study. In the studies of Josephson et al., in two of three patients without left ventricular aneurysm the morphologically different tachycardias appeared to originate in different sites.

Changes in morphology were accompanied by changes in rate, so one could argue that morphologic changes were caused by rate-dependent aberrant con-
duction. However, this in unlikely because comparable changes in rate were noted in ventricular tachycardias with similar morphology in the same patient (table 1).

**Macroseentry vs Microreentry**

Spurrell et al. suggested that gross changes in QRS morphology and the rate of tachycardia are unlikely to occur in a small reentrant circuit and postulated macroreentry involving bundle branches as the mechanism of tachycardia. In contrast, recent studies of Josephson et al. have suggested that ventricular tachycardia is due to microreentry within the ventricles. However, although our observations do not support bundle branch reentry as the mechanism of tachycardia, we cannot rule out the possibility that a long reentrant circuit with widely separated pathways may be required to produce gross changes in morphology of ventricular tachycardia. The induction of both left and right bundle branch block morphology tachycardias suggest that the pathways of conduction of the reentrant impulse are widely separated. If these pathways were narrowly separated, one would find only minor variations in the morphology of ventricular tachycardia.

**Clinical Implications**

The prognosis of patients with polymorphic ventricular tachycardia compared with patients without polymorphic tachycardia is not known. The observations made in this study raise the question whether these two types of patients respond differently to long-term drug therapy. Only long-term follow-up of patients with and without polymorphic ventricular tachycardia may provide the needed answers. We are following our patients to compare the clinical course and prognosis of patients with and without polymorphic ventricular tachycardia.

**Acknowledgment**

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