VENTRICULAR TACHYCARDIA is uncommon in young patients and is usually associated with cardiac tumors in infancy, with congenital heart disease in the older child, or with the prolonged QT syndrome.1–8 Most large series of young patients with ventricular tachycardia but without structural heart disease include individuals with tachycardias of a wide variety of underlying causes, including myocarditis, electrolyte disturbances, and effects of anesthesia.5, 9–13 Under these circumstances, the ventricular tachycardia occurs transiently, and with treatment of the underlying disorder, usually resolves spontaneously.13, 14

Idiopathic ventricular tachycardia, which is not associated with organic heart disease or identifiable pre-
Sustained ventricular tachycardia was that lasting greater than 30 sec or requiring an intervention for termination due to hemodynamic decompensation. Incessant ventricular tachycardia was either the continuous presence of sustained tachycardia, or runs of nonsustained tachycardia interrupted by one or two sinus beats.

The age at onset of ventricular tachycardia ranged from 6 to 21 years (mean ± SD, 14.9 ± 4.3 years). The mean age of the patients at the time of our evaluation was 17.8 ± 3.8 years. The presenting symptoms were cardiac arrest in two patients, syncope in four, presyncope in five, chest pain in four, and abdominal pain in one patient. Eight patients were asymptomatic. According to medical history, ventricular tachycardia was exercise related in nine patients, four of whom were trained athletes. No patient experienced ventricular tachycardia in the setting of an acute illness or during surgery. No history suggestive of preceding myocarditis or systemic illness was noted. Before referral, the number of episodes of tachycardia experienced was one in two patients, two to five in six patients, and more than five in 16 patients. Eleven patients had required electrical cardioversion for termination of ventricular tachycardia. Antiarrhythmic medications had been prescribed unsuccessfully for 19 patients, with a mean of three drugs per patient.

All but two patients had normal cardiac physical examinations; in the remaining two a click of mitral valve prolapse with no murmur was noted. Chest radiographs and electrocardiograms during sinus rhythm were normal in all patients. No patient had prolongation of the QT or corrected QT interval. No electrolyte abnormalities were present. All patients were considered to be in New York Heart Association functional class I.

Graded treadmill exercise testing by the Bruce protocol and 24 hr Holter monitoring were performed in all patients. Electrocardiograms were obtained in 22 patients. Hemodynamic and angiographic cardiac catheterization studies were performed in 23 patients before electrophysiologic testing. The normal values used for this study were mean right atrial and right ventricular end-diastolic pressures less than 8 mm Hg, and mean pulmonary capillary wedge pressure and left ventricular end-diastolic pressures less than or equal to 12 mm Hg. Right ventricular end-diastolic volumes calculated by Simpson's rule were those reported by Gentzler (mean ± SD, 81 ± 12.3 ml/m²)²⁹; left ventricular end-diastolic volumes (area-length method) were those reported by Kennedy et al.³⁰ (mean ± SD, 70 ± 20 ml/m²). Ejection fractions less than 50% for either ventricle were considered abnormal.

Electrophysiologic studies were performed in all 24 patients according to our previously described protocol. Studies were performed in patients in the postabsorptive, nonsedated state after obtaining informed consent. All antiarrhythmic medications were discontinued for at least five half-lives before testing.

Two to three quadripolar electrode catheters were inserted percutaneously into the femoral or basilic veins and positioned in the high right atrium, across the tricuspid valve at the His bundle site, and at the right ventricular apex.

Ventricular stimulation was performed from the right ventricular apex in all patients with the use of ventricular incremental pacing up to 250 beats/min and single premature extrastimulation during sinus rhythm and/or ventricular pacing. Double extrastimulation (S₃S₄) following ventricular paced cycle lengths of 600 or 500 msec was performed in 11 patients, and triple extrastimulation (S₃S₄S₅) was used in three patients. Additional stimulation from the right ventricular outflow tract was performed in three patients. After extrastimulation, the infusion of up to 5 μg/min isoproterenol was used in 10 patients, and atropine (1 mg) was given to three patients.

Endocardial catheter mapping was performed in 17 patients, with the use of the surface electrocardiogram and right ventricular apical electrogram as reference points. A second roving electrode catheter was used for mapping several sites in both ventricles. The earliest site of activation that was recorded during tachycardia and that preceded the onset of the surface QRS was considered to be the site of origin.

Short-term drug testing in the electrophysiology laboratory was performed according to a protocol previously described from this laboratory.³¹ A drug was considered efficacious if it prevented the induction of sustained ventricular tachycardia. Drugs successful in preventing induction of ventricular tachycardia in the laboratory were also evaluated during Holter monitoring and exercise testing. Drugs tested included procainamide (15 mg/kg infusion), quinidine (200 to 300 mg orally every 6 hr), disopyramide (300 mg orally every 6 hr), propranolol (0.15 mg/kg infusion or 40 to 160 mg orally every 6 hr), verapamil (5 to 10 mg infusion or 80 mg orally every 8 hr), aprindine (200 mg infusion or 100 mg orally every 12 hr), phenytoin (15 mg/kg infusion), and amiodarone (400 mg/day after a loading dose).

Drug levels were monitored to ensure that therapeutic levels were achieved. In patients in whom ventricular tachycardia was not inducible, drug testing was performed based on treadmill testing (for those with exercise-related ventricular tachycardia), or on Holter monitoring (for those with incessant ventricular tachycardia).

Results

Characteristics of ventricular tachycardia. Clinical episodes of ventricular tachycardia were sustained in 18 patients, incessant in four, and nonsustained in two. The rate of ventricular tachycardia ranged from 130 to 300 beats/min, with a mean of 200 ± 44 beats/min. The rate of tachycardia in symptomatic patients was 222 ± 38 beats/min and that in asymptomatic patients was 159 ± 17 beats/min (p < .05). Symptoms were present in 15 of 18 patients with sustained, in one of two patients with nonsustained, and none of four patients with incessant ventricular tachycardia. The QRS morphology during tachycardia was of a left bundle branch block configuration in 18, right bundle branch block in three, bilateral bundle branch block in one, and torsade de pointes in two patients.

Treadmill testing. Of the nine patients with a history of exercise-related ventricular tachycardia, treadmill testing induced sustained tachycardia in five and nonsustained tachycardia in one; nonsustained ventricular tachycardia appeared during the recovery period in two patients. One patient did not experience tachycardia during treadmill testing.

Among the 15 patients with no history of exercise-related ventricular tachycardia, nonsustained tachycardia was induced by exercise in one patient, and appeared during the recovery period in two patients. Ventricular tachycardia was suppressed by peak exercise in three patients, two of whom had incessant tachycardia clinically.

Echocardiogram. Abnormalities in cardiac size or function were noted on M mode and two-dimensional
echocardiograms of six patients. Abnormalities noted were mild right ventricular enlargement in two patients; mild left ventricular enlargement in one 17-year-old patient with a left ventricular end-diastolic dimension of 5.8 cm; slightly decreased left ventricular contractility in two patients, with a shortening fraction of 27%; and moderately decreased left ventricular function and ventricular dilatation in one 20-year-old patient during incessant ventricular tachycardia, with a left ventricular end-diastolic dimension of 6.5 cm.

Cardiac catheterization. Abnormalities in cardiac size or function were diagnosed at catheterization in 16 of 23 patients (70%; table 1). The abnormalities in ventricular volume, pressure, or contractility were limited to the right ventricle in five patients and to the left ventricle in another five. In six patients evidence of biventricular dysfunction was detected. Among patients with decreased ejection fractions, the mean right ventricular ejection fraction was 44%, and the mean left ventricular ejection fraction was 46%. Cardiac abnormalities were present in 11 of 16 (69%) symptomatic patients, and in five of eight (63%) asymptomatic patients. No patient had congenital heart disease or coronary artery abnormalities. Both patients with mitral valve prolapse had otherwise normal study results.

Electrophysiologic studies. AH and HV intervals were normal in all but four patients with HV prolongation in whom the HV interval was 59 ± 4 msec. Sinus node recovery time was normal in all patients.

Spontaneous ventricular tachycardia was present during the study in six patients, who therefore underwent endocardial mapping only. Of the 18 patients in whom programmed stimulation was performed, 11 (61%) had inducible sustained ventricular tachycardia with a QRS morphology that was identical to their clinical tachycardia. In two additional patients, non-sustained tachycardia identical to their clinical exercise-related tachycardia was present during the infusion of isoproterenol; ventricular tachycardia was not inducible by programmed stimulation alone in these patients. Sustained ventricular tachycardia was inducible by atrial incremental pacing in three patients, ventricular incremental pacing in seven patients, and ventricular extrastimulation in six patients, two of whom required triple extrastimulation. In five patients ventricular tachycardia was not inducible. In these patients, in addition to atrial and ventricular incremental pacing, the ventricular stimulation protocol included single extrastimulation and isoproterenol infusion in one patient, double extrastimulation and isoproterenol infusion in two patients, and triple extrastimulation in two patients.

Endocardial mapping. Of the 17 patients who underwent endocardial mapping, the QRS morphology of ventricular tachycardia was left bundle branch block in 14, bilateral bundle branch block in one, and torsade de pointes in two. The site of origin of tachycardia was the right ventricle in all patients with a left bundle branch block morphology. In these patients, the tachycardia originated in the outflow tract in 11, the apex in two, and the inflow area in one. In both patients with mitral valve prolapse, the ventricular tachycardia originated in the right ventricular outflow tract. The site of origin was the ventricular septum in the patient with bilateral bundle branch block, and it was indeterminate in the other two patients with torsade de pointes.

Drug testing. Short-term drug testing by programmed stimulation was performed in 12 patients with the use of a mean of 3.7 drugs per patient (table 2). At least one drug successful in preventing induction of sustained ventricular tachycardia was identified in all but one patient tested. Five additional patients underwent drug testing based on treadmill testing and/or Holter monitoring with a mean of 2.6 drugs per patient; an effective drug was identified for each patient. Overall, the success rate in preventing induction of sustained ventricular tachycardia by programmed stimulation and treadmill testing with type I antiarrhythmic medications was 44% (15/34), that for propranolol was 56% (5/9), for phenytoin 33% (1/3), for amiodarone 100% (2/2), and for verapamil 67% (2/3).

Treatment and follow-up. The follow-up period from the initial diagnosis of ventricular tachycardia in our patients ranged from 1 to 20 years (mean, 7.5 years). Figures 1 and 2 summarize treatment and outcome. Of the eight asymptomatic patients, spontaneous ventricular tachycardia was present during electrophysiologic testing in five. Two of these patients, who

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**TABLE 1**

<table>
<thead>
<tr>
<th>Hemodynamic data</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal catheterization</td>
<td>7</td>
</tr>
<tr>
<td>Abnormal catheterization</td>
<td>16</td>
</tr>
<tr>
<td>Elevated RVEDP</td>
<td>2</td>
</tr>
<tr>
<td>Increased RVEDV</td>
<td>5</td>
</tr>
<tr>
<td>Decreased RVEF</td>
<td>5</td>
</tr>
<tr>
<td>Elevated LVEDP</td>
<td>3</td>
</tr>
<tr>
<td>Increased LVEDV</td>
<td>8</td>
</tr>
<tr>
<td>Decreased LVEF</td>
<td>6</td>
</tr>
<tr>
<td>Regional wall motion abnormality</td>
<td>9</td>
</tr>
</tbody>
</table>

EDP = end-diastolic pressure; EDV = end-diastolic volume; EF = ejection fraction; LV = left ventricular; RV = right ventricular.
TABLE 2
Short-term drug testing with programmed stimulation

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Drug tested</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Diso/−; apr/−</td>
<td>Recurrent VT on diso; no VT on apr for 4 yr</td>
</tr>
<tr>
<td>2</td>
<td>Proc/+; diso/+; prop/+; phen/+; ver/+; mex/+; mex and diso/+</td>
<td>No VT on mex and diso for 10 mo</td>
</tr>
<tr>
<td>3</td>
<td>Proc/+; quin/+; diso/+</td>
<td>No VT on apr by Holter and treadmill testing; apr discontinued; sudden death</td>
</tr>
<tr>
<td>4</td>
<td>Proc/±; quin/+; diso/+; prop/−; bre/+; amio/−; apr−</td>
<td>Recurrent VT on proc, quin, diso, amio, amio and prop, amio and ver, amio and quin; no VT on amio and toc for 3 mo</td>
</tr>
<tr>
<td>5</td>
<td>Proc/+; quin/+; diso/+; prop/−; bre/+; amio/−; apr−</td>
<td>Prop discontinued due to side effects; VT on apr; no VT on amio; patient discontinued amio; no VT for 3 yr</td>
</tr>
<tr>
<td>6</td>
<td>Proc/−; quin/+; diso/+; prop/−; apr/+</td>
<td>Seizure on apr; on β-blockers, five episodes of VT over 4 yr</td>
</tr>
<tr>
<td>7</td>
<td>Proc/+; quin/−; diso/+; phen/+</td>
<td>No VT on quin for 1 yr</td>
</tr>
<tr>
<td>8</td>
<td>Proc/−; quin/−; diso/−; prop/+</td>
<td>Lupus on proc; on diso, two episodes of VT over 4 yr</td>
</tr>
<tr>
<td>9</td>
<td>Diso/±</td>
<td>During treadmill test, VT on proc: no VT on prop; patient discontinued prop; sudden death</td>
</tr>
<tr>
<td>10</td>
<td>Proc/+; diso/−; quin/+</td>
<td>No VT on quin for 3 yr</td>
</tr>
<tr>
<td>11</td>
<td>Ver−</td>
<td>VT on ver; no VT on quin for 14 mo</td>
</tr>
<tr>
<td>12</td>
<td>Proc/−</td>
<td>No VT on proc; patient discontinued proc; no VT for 2 yr</td>
</tr>
</tbody>
</table>

Amio = amiodarone; apr = aprindine; bre = bretylium; diso = disopyramide; mex = mexiletine; phen = phenytoin; prop = procainamide; = prop = propranolol; quin = quinidine; ver = verapamil; VT = ventricular tachycardia; ± = inducible nonsustained VT; + = inducible sustained VT; − = no inducible VT.

Spontaneous ventricular tachycardia was present in one of the 16 symptomatic patients during electrophysiologic study. This patient was treated. Tachycardia was not induced by programmed stimulation in three patients, two of whom were treated. The third patient presented with syncope and a single 4 beat run of nonsustained ventricular tachycardia was present during treadmill testing. She received no treatment and has been asymptomatic and without recurrent tachycardia for 10.5 years since her presentation with syncope. Of 12 patients with inducible ventricular tachycardia during electrophysiologic testing, one was not treated. This patient was a 13-year-old boy referred in 1976 with multiple episodes of tachycardia associated with abdominal pain. Because his cardiac angiogram was normal and the tachycardia originated in the right ventricular outflow tract, was resistant to five drugs,

had decreased left ventricular function and incessant tachycardia, were treated to assess the effect of sinus rhythm on ventricular function. The other three patients, two with incessant tachycardia clinically, were not treated. Ventricular tachycardia was induced in the laboratory with the use of isoproterenol in one patient with exercise-related tachycardia; he has been free of tachycardia on β-blocker therapy for 13 months. Tachycardia could not be induced in two patients, who were not treated; one has subsequently required treatment for symptomatic ventricular tachycardia. All patients in this group were alive and well over a mean follow-up period of 7.3 years.

FIGURE 1. Treatment and outcome of asymptomatic patients. All patients were alive and well during a mean follow-up period of 7.3 years. One patient later treated for symptomatic ventricular tachycardia. EPS = electrophysiologic study; Rx = antiarrhythmic therapy; VT = ventricular tachycardia.

Asymptomatic

Symptomatic

Spontaneous VT at EPS

VT inducible at EPS

VT not inducible at EPS

N=16

N=12

N=3

2 deaths

1 death

FIGURE 2. Treatment and outcome of symptomatic patients. Three patients died suddenly during a mean follow-up period of 7.6 years. Medication discontinued. EPS = electrophysiologic study; Rx = antiarrhythmic therapy; VT = ventricular tachycardia.
and was not related to exercise, the arrhythmia was thought to be benign and no treatment was recommended. He died shortly thereafter; postmortem examination revealed biventricular enlargement with fatty infiltration of the right ventricle and diffuse abnormalities of the conduction system.\(^3\) Of the 11 patients with inducible ventricular tachycardia who were treated, all have had recurrent tachycardia requiring adjustments in their medications. Three patients continued to have tachycardia despite treatment, necessitating implantation of an automatic implantable defibrillator in one, and combination therapy to slow the rate of the ventricular tachycardia in two patients. Medications were discontinued in five patients, either by the patient or the referring doctor; two of these patients died suddenly. Both patients had exercise-related tachycardia that had been well-controlled by drug therapy (apridine or propranolol). The mean duration of follow-up for this group was 7.6 years.

**Prognostic factors.** Overall mortality in this population was 13\% (3/24 patients). Because of the small number of patients involved, no statistically significant factor could be identified that predicted a poor prognosis. The rate of ventricular tachycardia, morphology, age at onset, and left ventricular ejection fraction were not different between survivors and nonsurvivors. Two of the nine patients with exercise-related tachycardia died during follow-up.

All patients who died had multiple recurrent episodes of symptomatic ventricular tachycardia and had required cardioversion at least once. Symptoms during tachycardia in these patients were syncope, presyncope, and abdominal pain. Heart disease was diagnosed at catheterization in two patients and at postmortem examination in the third patient. The left ventricular ejection fractions in these three patients were 40\%, 63\%, and 66\%. Sustained ventricular tachycardia was easily induced by use of programmed stimulation in all three patients. No patient was receiving antiarrhythmic medication at the time of death.

**Discussion**

Ventricular arrhythmias in children, although uncommon, have received increasing attention in recent years.\(^5\)\(^-\)\(^8\)\(^,\)\(^11\)\(^-\)\(^13\)\(^,\)\(^18\)\(^,\)\(^27\)\(^,\)\(^33\)\(^,\)\(^34\) In the absence of congenital heart disease or other predisposing factors, the diagnosis of ventricular tachycardia in children is commonly an incidental finding in asymptomatic patients and is often limited to a single episode of nonsustained type.\(^5\)\(^,\)\(^13\)\(^,\)\(^25\)\(^,\)\(^27\)\(^,\)\(^35\) Although reports of deaths in children due to primary ventricular tachycardia have appeared,\(^23\)\(^,\)\(^32\)\(^,\)\(^35\)\(^-\)\(^40\) most authors stress that in the absence of underlying heart disease, tachycardia in the young population is an arrhythmia with a good prognosis that may not need treatment, and may resolve spontaneously.\(^8\)\(^,\)\(^13\)\(^,\)\(^18\)\(^,\)\(^20\)

**Patient selection.** The present population differs markedly from previous series in several respects. All of our patients were otherwise healthy young persons referred for the management of ventricular tachycardia. Most of our patients (67\%) were symptomatic, and almost half (46\%) had required electrical cardioversion before referral. The episodes of ventricular tachycardia were either sustained or incessant in 92\% of patients. With the exception of a click of mitral valve prolapse in two patients, no patient in this series had clinically evident heart disease. In both patients with mitral prolapse the tachycardia originated in the right ventricular outflow tract, making it difficult to assign a role to the prolapse in the cause of their arrhythmia.

**Clinical features.** The high incidence of cardiac dysfunction (70\%) diagnosed at cardiac catheterization in our young patients deserves some comment. An additional patient in whom results of cardiac catheterization were normal was found to have an abnormal heart at postmortem examination, suggesting that either our catheterization values were not entirely sensitive, or that there had been progression of cardiac disease. Echocardiography did not prove to be sensitive in detecting these abnormalities, since only six patients (26\%) had abnormal echocardiograms. The diagnosis of cardiomyopathy was suggested by subtle findings at cardiac catheterization. These findings included elevated end-diastolic pressures, increased ventricular volumes, or regional wall motion abnormalities. Ventricular tachycardia was the first and, to date, the only clinical manifestation of cardiomyopathy in our patients. Since most children without clinical evidence of heart disease in previous studies did not undergo cardiac catheterization, it is possible that subtle evidence of cardiomyopathy was not uncovered.

The existence of subclinical evidence of cardiac disease in patients presenting with ventricular arrhythmias and apparently normal hearts has been well documented by autopsy, cardiac catheterization, and endomyocardial biopsy studies.\(^5\)\(^,\)\(^32\)\(^,\)\(^38\)\(^-\)\(^48\) Diffuse abnormalities of the conduction system were described at autopsy in four young patients with ventricular arrhythmias who died suddenly.\(^32\)\(^,\)\(^39\)\(^,\)\(^40\) Pietras et al.\(^44\) reported abnormal right ventricular volumes and function in 45\% of patients with right ventricular tachycardia, while Kennedy et al.\(^45\) described occult left ventricular dysfunction in 61\% of their patients with

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complex ventricular ectopy. Recently, endomyocardial biopsies in patients with ventricular tachycardia and normal cardiac catheterization data have revealed histologic abnormalities, including fibrosis and myocellular hypertrophy, in 50% to 90% of patients.\textsuperscript{45, 46} Arrhythmogenic right ventricular dysplasia, characterized by hypokinetic areas of the right ventricle, repolarization abnormalities in the electrocardiogram, and fatty deposits in the right ventricular wall, has been described in children and adults with right ventricular tachycardia.\textsuperscript{49, 50} Although several of our patients had right ventricular regional wall motion abnormalities, no characteristic electrocardiographic changes were present; however, the postmortem examination in the boy with normal right ventricular contractility at cardiac catheterization was consistent with the diagnosis of arrhythmogenic right ventricular dysplasia. Attempts to further characterize the anatomic and histologic substrates in these patients may depend on findings obtained at endomyocardial biopsy.

**Electrophysiologic features.** Limited information concerning the electrophysiologic characteristics of primary ventricular tachycardia in children is available.\textsuperscript{5, 8, 12, 51-54} We were able to induce tachycardia in 72% of our patients during electrophysiologic testing. Because of the evolving stimulation protocol used since 1974 in our laboratory, ventricular tachycardia was most often induced by rapid ventricular pacing, which may be consistent with either a reentrant or triggered automatic mechanism. The use of a more rigorous and standardized stimulation protocol (double and triple extrastimulation) in all patients in the future would both increase the number of patients in whom ventricular tachycardia could be induced, and allow postulations regarding the mechanism.\textsuperscript{55-58}

**Treatment.** Once the decision to treat was made, a drug effective in preventing or controlling ventricular tachycardia could be identified in 14 of 17 patients (82%) with a combined approach of programmed stimulation, treadmill testing, and Holter monitoring. Although we were able, using programmed stimulation, to identify drugs that were effective in preventing tachycardia over the short-term, we observed that these drugs were only partially successful in preventing recurrent tachycardia during follow-up. This finding may be due to poor compliance with therapy, progression of the underlying disease, or the inherent limitations of drug testing with programmed stimulation. To date, none of our patients has died while receiving antiarrhythmic medication; three patients died suddenly shortly after discontinuing treatment.

**Prognosis.** The mortality among our patients was 13%, which is higher than previously reported.\textsuperscript{13} This mortality rate reflects both the nature of our population of patients with predominantly sustained, symptomatic ventricular tachycardia without predisposing causes, and the long duration of follow-up. The deaths occurred in patients who had symptomatic tachycardia that could be reproducibly induced by programmed stimulation and who were not receiving treatment. Patients with exercise-related ventricular tachycardia may be at an especially high risk of sudden death. No asymptomatic patient in our series died, but three patients received treatment. However, there have been reported deaths in asymptomatic patients,\textsuperscript{55, 57, 59} suggesting that these patients are also at risk for sudden death. Because ventricular tachycardia may be the clinical manifestation of underlying cardiac pathology, long-term follow-up is imperative to reach conclusions regarding the prognosis of these patients.

**Conclusions and recommendations.** (1) Ventricular tachycardia in a young population without clinical evidence of heart disease may be the first manifestation of cardiomyopathy. By cardiac catheterization at least two-thirds of these patients have subtle evidence of cardiac dysfunction. (2) During a mean follow-up period of 7.5 years, the mortality in our population was as high as 13%, and occurred in patients not receiving antiarrhythmic therapy. (3) In any young patient with ventricular tachycardia with no identifiable predisposing cause, we recommend cardiac catheterization, angiography, and electrophysiologic study to define subclinical evidence of heart disease and to evaluate the characteristics of the tachycardia. (4) Presently we recommend treatment of ventricular tachycardia in any asymptomatic patient, with therapy guided by electrophysiologic and treadmill testing. In addition, we recommend treatment for any asymptomatic patient with exercise-related tachycardia, since this group appears to be at increased risk for sudden death. Finally, we would seriously consider treating any asymptomatic patient in whom sustained monomorphic ventricular tachycardia is inducible by standard programmed stimulation, although the long-term prognosis in this group is uncertain.

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