Adenosine-Sensitive Ventricular Tachycardia
Clinical Characteristics and Response to Catheter Ablation

David J. Wilber, MD; Jeffrey Baerman, MD; Brian Olshansky, MD; John Kall, MD; and Douglas Kopp, MD

Background. Sustained ventricular tachycardia in the absence of structural heart disease may have diverse mechanisms. Termination of the tachycardia by adenosine suggests triggered automaticity as the etiology in many of these patients. We examined the clinical characteristics, electrophysiological responses, and results of catheter ablation in this patient subgroup.

Methods and Results. Intravenous adenosine terminated sustained ventricular tachycardia in seven of 14 consecutive patients without evidence of structural heart disease. In each of these patients, the tachycardia had a left bundle branch block, inferior-axis QRS configuration and occurred predominantly during stress or exertion. A morphologically similar sustained tachycardia was induced in six of seven patients during programmed ventricular stimulation, although day-to-day reproducibility was poor. Signal-averaged ECGs were normal in all patients. Imaging with 123I-metaiodobenzylguanidine did not reveal focal abnormalities in any of five patients. A discrete site of origin was identified in the free wall of the pulmonary infundibulum in all patients. Limited application of direct current shocks (two patients) or radiofrequency energy (five patients) resulted in long-term abolition of spontaneous and inducible ventricular tachycardia in all patients.

Conclusions. Adenosine-sensitive ventricular tachycardia appears to arise from relatively discrete sites predominantly located in the free wall of the pulmonary infundibulum. The localized nature of this tachycardia renders it amenable to long-term cure by catheter ablation techniques. (Circulation 1993;87:126-134)

KEY WORDS • pace mapping • activity, triggered

Ventricular tachycardia in patients without structural heart disease is an uncommon clinical entity.1 In a large proportion of such patients, the tachycardia has a uniform left bundle branch block, inferior-axis QRS configuration, suggesting an origin in the right ventricular outflow tract.2-13 However, these latter patients do not constitute a homogeneous group. Clinical presentations range from incessant runs of monomorphic nonsustained ventricular tachycardia to infrequent episodes of sustained ventricular tachycardia. Exercise may either suppress or facilitate tachycardia initiation. Attempts to classify tachycardia mechanism by the response to programmed stimulation and pharmacological manipulation (verapamil, isoproterenol) suggest diverse potential etiologies including reentry, triggered activity, and catecholamine-mediated automaticity.4-8

Lerman et al12 recently described a group of patients with exercise-induced sustained ventricular tachycardia with a left bundle branch block, inferior-axis QRS configuration in whom adenosine reliably terminated the tachycardia. The response to adenosine and to autonomic maneuvers that antagonize catecholamine-associated increases in cAMP provided strong evidence for cAMP-mediated triggered automaticity as the mechanism of tachycardia. These observations also suggested that adenosine may be used as a diagnostic tool to identify a unique subset of patients with idiopathic ventricular tachycardia.

The purpose of this report is to further characterize the clinical presentation and electrophysiological responses of patients with adenosine-sensitive, catecholamine-mediated ventricular tachycardia. Detailed mapping and pacing studies demonstrated that these tachycardias arise from discrete foci in the free wall of the pulmonary infundibulum. Catheter ablation at these sites resulted in long-term abolition of spontaneous ventricular tachycardia.

Methods

Patient Selection

In 14 patients presenting with spontaneous sustained ventricular tachycardia without evidence of structural heart disease, intravenous adenosine was administered during the tachycardia to assess effects on termination (see below). Adenosine failed to terminate ventricular tachycardia in seven patients. The remaining seven patients, all with adenosine-sensitive left bundle branch block, inferior-axis ventricular tachycardia, comprise the study group. In these patients, ambulatory monitoring, formal exercise testing, and signal-averaged ECGs (method of Simpson14) were performed before electrophysiological evaluation.
**Electrophysiological Testing**

Studies were performed in the postabsorpive state in the absence of antiarrhythmic drug therapy after informed consent had been obtained. Quadrupolar catheters were inserted percutaneously and advanced under fluoroscopic guidance to the right atrium, right ventricular apex, and the atrioventricular junction. Bipolar electrograms were filtered at 30–500 Hz and displayed simultaneously with six surface ECG leads on a multichannel oscilloscope (Siemens-Elema, Inc. Solna, Sweden). Data were stored on magnetic tape (Teac XR-510, Montebello, Calif.) and printed on an ink-jet recorder (Siemens-Elema Mingograf) for immediate review. Electrical stimulation was performed with a programmable stimulator and an isolated constant current source (Bloom Associates, Ltd., Narbeth, Pa.) using rectangular pulses of 2-msec duration at twice diastolic threshold. Decremental burst atrial pacing and the introduction of single atrial extrastimuli at decremental intervals during multiple paced atrial cycle lengths were performed in all patients. Ventricular stimulation, which included the introduction of single, double, and triple extrastimuli during multiple paced cycle lengths at two right ventricular sites, was performed as previously described. Burst ventricular pacing at cycle lengths of 200–400 msec was also performed.

If sustained ventricular tachycardia could not be induced during baseline stimulation, the stimulation protocol was repeated during isoproterenol infusion (2–8 g/min). Intravenous adenosine (6–18 mg) was injected via a central vein during sustained ventricular tachycardia to assess the effect on termination. During sustained tachycardia, autonomic maneuvers, including both right and left carotid sinus massage and the Valsalva maneuver, were also performed.

**Mapping and Catheter Ablation**

After completion of electrophysiological studies, activation sequence mapping of the right ventricle was performed during sustained or nonsustained ventricular tachycardia. Electrograms from the distal bipolar pair of the mapping catheter (2-mm bipolar separation) were filtered between 30 and 500 Hz, and activation times were recorded relative to the onset of the surface QRS. Positioning of the catheter tip was confirmed in multiple projections and recorded on film. Since all tachycardias had a left bundle branch block, inferior-axis morphology, mapping efforts were concentrated in the region of the right ventricular outflow tract. Pace mapping was performed during sinus rhythm at a rate similar to the induced tachycardia or during ventricular tachycardia at a rate slightly faster than the tachycardia rate. The QRS morphology in each of 12 leads was compared with the morphology during ventricular tachycardia. Optimal pace maps were defined as those with the closest possible match between QRS morphologies in each of the 12 leads.

After identification of the putative site of tachycardia origin, ablation was performed using either direct current shocks or application of radiofrequency energy. For patients undergoing direct current shocks, the distal tip of a 7F quadripolar catheter (USCI, Billerica, Mass.) was connected to the cathodal output of a standard defibrillator. A 16-cm² cutaneous patch electrode (R-2 Corporation, Morton Grove, Ill.) positioned on the back was used as the anode. During direct current shocks, all patients were anesthetized with intravenous midazolam. Radiofrequency energy applications were made using a programmable lesion generator with 300-kHz output with continuous monitoring of voltage and impedance (Radionics Inc., Burlington, Mass.). The cathodal output was delivered to the distal 4-mm tip electrode of a steerable quadripolar catheter (Mansfield, Boston Scientific Corp., Watertown, Mass.). The anode was a cutaneous patch.

**Results**

**Patient Characteristics**

The clinical characteristics of the study population are summarized in Table 1. All patients had a history of sustained exertional or stress-related palpitations. At least one spontaneous episode of sustained monomorphic ventricular tachycardia with a left bundle branch

---

**Table 1. Clinical Characteristics**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)/sex</th>
<th>Duration (years)</th>
<th>Episodes (n)</th>
<th>CL (msec) SMVT</th>
<th>Prior drug therapy</th>
<th>ETT</th>
<th>Holter</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27/M</td>
<td>2</td>
<td>&gt;10</td>
<td>240–280</td>
<td>βB, V, Q, ME, FL</td>
<td>SMVT</td>
<td>PVCs</td>
</tr>
<tr>
<td>2</td>
<td>51/F</td>
<td>8</td>
<td>&gt;20</td>
<td>220–280</td>
<td>βB, V, Q, PA, N, AM</td>
<td>PVCs</td>
<td>PVCs</td>
</tr>
<tr>
<td>3</td>
<td>53/M</td>
<td>30</td>
<td>&gt;50</td>
<td>390</td>
<td>βB, V, Q</td>
<td>SMVT</td>
<td>PVCs</td>
</tr>
<tr>
<td>4</td>
<td>37/F</td>
<td>15</td>
<td>&gt;20</td>
<td>300–360</td>
<td>βB, V, PP, EM</td>
<td>NSVT</td>
<td>NSVT*</td>
</tr>
<tr>
<td>5</td>
<td>25/M</td>
<td>1</td>
<td>4</td>
<td>270</td>
<td>βB, EM</td>
<td>NSVT</td>
<td>PVCs</td>
</tr>
<tr>
<td>6</td>
<td>49/F</td>
<td>1</td>
<td>2</td>
<td>350–400</td>
<td>βB, V</td>
<td>SMVT</td>
<td>PVCs</td>
</tr>
<tr>
<td>7</td>
<td>23/F</td>
<td>1</td>
<td>3</td>
<td>390</td>
<td>βB, V</td>
<td>NSVT</td>
<td>PVCs</td>
</tr>
</tbody>
</table>

CL, cycle length; SMVT, sustained monomorphic ventricular tachycardia; ETT, exercise tolerance test; βB, β-blockers; V, verapamil; Q, quinidine; M, mexiteline; F, flecainide; PA, procainamide; N, norpace; AM, amiodarone; PP, propafenone; EM, ethmozine; PVCs, premature ventricular complexes; NSVT, nonsustained ventricular tachycardia.

*Parentheses indicate number of nonsustained tachycardia beats.
block, inferior-axis QRS configuration was documented electrocardiographically in each patient. Five patients had syncope or near syncope. In the absence of sustained ventricular tachycardia, spontaneous ventricular premature complexes with a similar QRS configuration were present in all patients and usually increased during periods of stress or exercise. Ambulatory monitoring demonstrated fewer than 200 ventricular premature complexes per 24-hour period in five patients. Only one patient had repetitive paroxysms of monomorphic nonsustained ventricular tachycardia during ambulatory monitoring. In this patient (No. 3), ventricular premature complexes comprised 43% of recorded beats.

Coronary arteriography and left ventriculography were normal in all patients. Two-dimensional echocardiographic assessment of right ventricular size and function was normal in all patients. Myocardial biopsy was performed in three patients and was normal in each. The signal-averaged ECG did not meet any criterion for late potentials (total filtered QRS >120 msec, root mean-squared amplitude of the terminal 40 msec <25 V, duration of low amplitude signals >39 msec<sup>14</sup>) in any patient.

**Electrophysiological Testing**

The results of preablation electrophysiological testing are summarized in Table 2. In the absence of isoproterenol, sustained ventricular tachycardia was initiated reproducibly in only one patient. During isoproterenol infusion, sustained ventricular tachycardia was induced in an additional five patients. In one patient (No. 5), only nonsustained ventricular tachycardia could be induced. All induced tachycardias had a left bundle branch block, inferior-axis QRS configuration.

The relation between the coupling interval of the last paced beat and the return cycle of the first tachycardia beat was examined in four patients. There was no consistent relation between these two variables in any patient. Single and double extrastimuli were introduced during sustained ventricular tachycardia at progressively shorter coupling intervals in four patients. Resetting was not observed in any patient. In all patients with induced sustained ventricular tachycardia, burst ventricular pacing terminated the tachycardia.

Electrophysiological testing was performed in the absence of antiarrhythmic drug therapy on multiple days in four patients. In these patients, the induction of sustained ventricular tachycardia was variable. In patient 4, sustained ventricular tachycardia could not be induced by any technique on two consecutive days. However, sustained ventricular tachycardia was induced during preoperative ventricular stimulation on the preablation study 4 months later. In patients 2 and 7, sustained ventricular tachycardia could be induced only at the second of two studies 1 and 3 months apart, respectively. Patient 3 underwent three studies. Sustained ventricular tachycardia was not induced initially but was reproducibly induced on two different days 6 months later.

**Response to Adenosine and Autonomic Maneuvers**

Adenosine (6–12 mg) was administered during sustained ventricular tachycardia in all seven patients. In each patient, the tachycardia had persisted for at least 3 minutes before injection and was terminated within 15 seconds of injection. At least two successful terminations of induced sustained ventricular tachycardia were documented in six patients. In one patient (No. 5) in whom sustained ventricular tachycardia could not be induced during electrophysiological testing, adenosine had terminated a previous episode of spontaneous sustained ventricular tachycardia. Autonomic maneuvers were performed in three patients (No. 2, No. 3, and No. 6). Carotid sinus massage reproducibly terminated sustained ventricular tachycardia in all three patients. The Valsalva maneuver reproducibly terminated sustained ventricular tachycardia in two patients.

**Mapping and Ablation**

Mapping during sustained or nonsustained ventricular tachycardia revealed endocardial activation before the onset of the surface QRS in the right ventricular outflow tract in all patients, although presystolic activation was recorded over a relatively large area. Activation
TABLE 3. Catheter Ablation Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>SMVT CL (msec)</th>
<th>Frontal axis</th>
<th>EAT (msec)</th>
<th>Ablation site</th>
<th>Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>270</td>
<td>+115</td>
<td>-20</td>
<td>A</td>
<td>DC (200 J, 1 application)</td>
</tr>
<tr>
<td>2</td>
<td>220</td>
<td>+75</td>
<td>-10</td>
<td>C</td>
<td>RF (50 W x 30 seconds, 4 applications)</td>
</tr>
<tr>
<td>3</td>
<td>390</td>
<td>+90</td>
<td>-20</td>
<td>B</td>
<td>RF failed, DC (200 J, 1 application)</td>
</tr>
<tr>
<td>4</td>
<td>290*</td>
<td>+90</td>
<td>-30</td>
<td>B</td>
<td>RF (40 W x 30 seconds, 6 applications)</td>
</tr>
<tr>
<td>5</td>
<td>270*</td>
<td>+75</td>
<td>-10</td>
<td>C</td>
<td>RF (40 W x 30 seconds, 6 applications)</td>
</tr>
<tr>
<td>6</td>
<td>350</td>
<td>+90</td>
<td>-20</td>
<td>B</td>
<td>RF (50 W x 30 seconds, 7 applications)</td>
</tr>
<tr>
<td>7</td>
<td>380</td>
<td>+75</td>
<td>-10</td>
<td>C</td>
<td>RF (50 W x 30 seconds, 4 applications)</td>
</tr>
</tbody>
</table>

SMVT, sustained monomorphic ventricular tachycardia; CL, cycle length; EAT, earliest activation time; DC, direct current shock; RF, radiofrequency energy; A, site near septal attachment of the free wall; B, site on mid free wall of the outflow tract; C, free wall site posterolaterally (closer to tricuspid valve).

*CL of nonsustained ventricular tachycardia at preablation testing.

at the site of successful ablation preceded the onset of the surface QRS by 10–30 msec (Table 3). Low-amplitude or fractionated electrograms were not observed.

The optimal pace map in each patient demonstrated a close concordance of QRS configuration in each of the 12 leads (Figure 1). In each patient, the site of the optimal pace map was discrete and generally reproducible only within a few millimeters. Presystolic activation was frequently recorded at sites with poor pace map matches. For this reason, the optimal pace map was used as the primary guide for selection of the ablation site.

![Figure 1. QRS configuration in each of 12 surface ECG leads during ventricular tachycardia (VT) (left panel) and ventricular pacing from the optimal pace map site (right panel) in patient 2. Horizontal bar indicates 1 second. See text for details.](image)

Optimal pace maps and the subsequent sites of successful ablation were all located on the anterior free wall of the pulmonary infundibulum (Figure 2 and Table 3), ranging from sites near the septal attachment anteromedially to sites closer to the tricuspid valve posterolaterally. The frontal-plane QRS axis was an excellent predictor of the site of the optimal pace map. A predominantly negative QRS in lead I was associated with a site near the septal attachment of the free wall (site A). An isoelectric QRS in lead I was associated with a site on the mid free wall of the outflow tract (site B). A third distinct morphology had a predominantly positive QRS configuration in lead I and was associated with a free wall site posterolaterally (site C), closer to the tricuspid valve (Figure 3).

Direct current shock was the initial energy modality in the first patient only. Radiofrequency energy was the initial treatment in the remaining six patients. Application of radiofrequency energy during the tachycardia was associated with brief acceleration followed by irregular slowing for several seconds before termination (Figure 4). The goal of radiofrequency application was the elimination of all spontaneous ventricular ectopy and inducible tachycardia (sustained or nonsustained) during isoproterenol infusion. If spontaneous ectopy or inducible ventricular tachycardia persisted, minor repositioning of the catheter within a few millimeters was performed, and another application was given. In one patient with unsuccessful radiofrequency ablation, a single direct current shock applied to the same site was successful. In the remaining five patients, spontaneous and inducible ventricular arrhythmias were eliminated with two to five radiofrequency applications.

**Follow-up**

There were no immediate adverse sequelae in any patient. The initial patient, who received a single direct current shock, had several runs of polymorphic nonsustained ventricular tachycardia (three to seven beats) within the first 24 hours after ablation. No treatment was given, and these arrhythmias resolved spontaneously. Two-dimensional echocardiograms were performed 1–4 weeks after ablation in four patients (patients 1–4), and no abnormalities were observed.

All patients were discharged on no antiarrhythmic medications. Follow-up electrophysiological testing, including stimulation during isoproterenol infusion, was performed at 1–4 weeks in all patients. Nonsustained or
sustained ventricular tachycardia could not be induced in any patient. Ambulatory monitoring and formal treadmill testing performed during a similar time frame revealed at most rare premature ventricular complexes (often with a different QRS configuration) in all patients.

During a mean follow-up of 16 months (range, 5–33 months), no patient had recurrent ventricular tachycardia or sustained palpitations. Transtelphoneic event monitors were used in all patients to investigate complaints of palpitations or “cardiac awareness.” In all instances, only isolated premature ventricular complexes were documented.

Five patients underwent radionuclide scanning with the guanethidine analogue 123I-metaiodobenzylguanidine (MIBG) 1–4 months after ablation to assess the integrity of cardiac adrenergic innervation.16 Focal abnormalities were absent in all patients. In four patients, cardiac uptake of the tracer was dense and homogenous throughout, including the region of the pulmonary infundibulum. In one patient, cardiac uptake of the tracer was minimal throughout the heart despite normal uptake in other organs, suggesting diffusely rapid turnover of cardiac neurotransmitters.16 These observations suggest that the mechanism of successful ablation was direct tissue injury rather than focal denervation or local catecholamine depletion.

**Patients With Ventricular Tachycardia Unresponsive to Adenosine**

Patients without structural heart disease in whom adenosine did not terminate ventricular tachycardia were a heterogeneous group (Table 4). The tachycardias had a left bundle branch block, inferior-axis QRS configuration in two patients, and activation/pace mapping identified a site of origin on the septum of the right ventricular outflow tract. Five patients had right bundle branch block, superior-axis tachycardias. Mapping in four of these patients revealed a site of origin in the left ventricular inferior apical septum. Two of these patients had exercise-induced ventricular tachycardia and required isoproterenol for induction of the tachycardia during programmed stimulation. Verapamil suppressed inducible and spontaneous tachycardias in one of these latter patients.

**Discussion**

**Major Findings**

This study demonstrates that adenosine reproducibly terminates ventricular tachycardia in a large proportion of patients with structurally normal hearts, particularly those who present with a left bundle branch block, inferior-axis QRS configuration. These patients share several common features. Patients typically have stress-related or exertion-related sustained ventricular tachycardia; repetitive paroxysms of monomorphic nonsustained tachycardia are rare. The provocation of sustained tachycardia during standard exercise testing and programmed ventricular stimulation is not always reproducible. With the exception of adenosine sensitivity, these characteristics may also be observed in other patients with idiopathic ventricular tachycardia. However, in each
Adenosine-responsive patient, the tachycardia had a left bundle branch block, inferior-axis QRS configuration and arose from a discrete site in the free wall of the pulmonary infundibulum. Focal ablation at this site resulted in long-term abolition of ventricular tachycardia.

**Electrophysiological Mechanisms**

The use of adenosine to identify a common tachycardia mechanism in patients with idiopathic ventricular tachycardia is of particular importance, given the unreliability of other diagnostic maneuvers or clinical findings. The only known effect of adenosine in ventricular myocardium is the antagonism of catecholamine-stimulated elevations in cAMP. The efficacy of adenosine in terminating ventricular tachycardia strongly supports triggered activity as the responsible mechanism in these patients. The response to carotid sinus massage and the Valsalva maneuver in the study patients and those

**FIGURE 3.** Radiograph in the right anterior oblique projection illustrating the positioning of catheters for ablation in patient 2. Catheters are positioned in the right ventricular inflow tract (bottom right), the right atrium (middle left), and at the optimal pace mapping site on the free wall of the pulmonary infundibulum (top middle).

**FIGURE 4.** Onset of radiofrequency application during sustained ventricular tachycardia (indicated by bold line above the right ventricular outflow tract [RVOT] electrogram). There is an initial acceleration of the tachycardia followed by progressive slowing and irregularity. After 14 seconds, ventricular tachycardia terminates and does not recur. Bold vertical line is equal to 200 msec.
TABLE 4. Characteristics of Patients With Ventricular Tachycardia Not Responsive to Adenosine

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)/sex</th>
<th>SMVT morphology</th>
<th>Exercise induced</th>
<th>Verapamil response</th>
<th>Programmed stimulation</th>
<th>Mapping</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>38/M</td>
<td>RBBB-sup</td>
<td>Yes</td>
<td>Yes</td>
<td>SMVT (2 VES)</td>
<td>LV IAS</td>
</tr>
<tr>
<td>2</td>
<td>54/M</td>
<td>RBBB-sup</td>
<td>Yes</td>
<td>No</td>
<td>SMVT (2 VES)</td>
<td>LV IAS</td>
</tr>
<tr>
<td>3</td>
<td>14/F</td>
<td>RBBB-sup</td>
<td>No</td>
<td>No</td>
<td>SMVT (3 VES)</td>
<td>LV IAS</td>
</tr>
<tr>
<td>4</td>
<td>36/M</td>
<td>RBBB-sup</td>
<td>No</td>
<td>No</td>
<td>SMVT (3 VES)</td>
<td>LV IAS</td>
</tr>
<tr>
<td>5</td>
<td>14/M</td>
<td>RBBB-sup</td>
<td>No</td>
<td>No</td>
<td>SMVT (1 VES)</td>
<td>NT</td>
</tr>
<tr>
<td>6</td>
<td>42/M</td>
<td>LBBB-inf</td>
<td>No</td>
<td>No</td>
<td>SMVT (2 VES)</td>
<td>RVOT septum</td>
</tr>
<tr>
<td>7</td>
<td>72/M</td>
<td>LBBB-inf</td>
<td>No</td>
<td>NT</td>
<td>SMVT (3 VES)*</td>
<td>RVOT septum</td>
</tr>
</tbody>
</table>

SMVT, sustained monomorphic ventricular tachycardia; RBBB, right bundle branch block; sup, superior axis; LBBB, left bundle branch block; inf, inferior axis; NT, not tested; VES, ventricular extrastimuli; LV, left ventricle; IAS, inferior apical septum; RVOT, right ventricular outflow tract.

*Two additional morphologies not seen clinically also induced.

characterized by Lerman et al7 are also consistent with this mechanism.18

In contrast, the response to programmed stimulation has important limitations in defining the mechanism of ventricular tachycardia18; both reentrant and triggered ventricular arrhythmias may be initiated by premature extrastimuli and burst ventricular pacing.19 The induction and termination of ventricular tachycardia by programmed ventricular stimulation in the study patients does suggest that normal or abnormal automaticity are unlikely mechanisms.18

In previous reports of electrophysiological testing in patients with catecholamine-associated, idiopathic, left bundle branch block, inferior-axis sustained ventricular tachycardia, the induction of ventricular tachycardia during electrophysiological testing ranged from 0% to 100%.2-10 Lerman and associates7 found that sustained ventricular tachycardia could be induced by programmed ventricular stimulation in three of four patients with adenosine-sensitive ventricular tachycardia and by atrial pacing in the remaining patient. Sung and coworkers5 found that atrial or ventricular pacing could induce ventricular tachycardia in four of four patients with verapamil-sensitive, idiopathic, left bundle branch block, inferior-axis ventricular tachycardia. In this study, the induction of sustained ventricular tachycardia required the use of a relatively aggressive stimulation protocol and repeated stimulation on different days. Thus, failure to induce ventricular tachycardia by programmed stimulation at any single point in time does not exclude triggered automaticity as the underlying mechanism of spontaneous tachycardia.

Neither verapamil sensitivity nor adrenergic responsiveness are specific probes for tachycardia mechanisms. Catecholamines may facilitate arrhythmias caused by reentry, automaticity, or triggered activity,20 and β-blockade occasionally may be effective in abolishing ventricular tachycardias caused by any of these mechanisms.7,8,21 Although verapamil frequently suppresses ventricular tachycardia presumed to be secondary to triggered automaticity,7,8 it has also been shown to suppress arrhythmias consistent with reentry in both ischemic22 and normal hearts.23 One patient in this study had catecholamine-induced, verapamil-sensitive ventricular tachycardia that did not respond to adenosine.

**Localization and Ablation Techniques**

Experience with both direct surgical14-24 or percutaneous catheter ablation25,26 in patients with idiopathic right ventricular tachycardia is limited. Reports of successful catheter ablation are limited to the use of direct current shocks. Stevenson et al25 described a patient with catecholamine-mediated, left bundle branch block, inferior-axis ventricular tachycardia in whom three direct current shocks delivered to the right ventricular outflow tract eliminated subsequent episodes of ventricular tachycardia and markedly reduced the frequency of spontaneous premature complexes. Morady et al26 recently reported successful catheter ablation in eight of 10 patients with idiopathic left bundle branch block, inferior-axis sustained ventricular tachycardia using direct current shocks applied to the right ventricular outflow tract. None of these patients received adenosine, and specific localization of the ablation site within the outflow tract was not reported.

Previous mapping studies of catecholamine-associated right ventricular outflow tract tachycardia indicated that such tachycardias arise predominantly from the intraventricular septum.24 In this study, all patients with adenosine-responsive ventricular tachycardia had effective ablation with localized application of energy in the free wall of the pulmonary infundibulum. In the former studies, pace mapping and ablation were not used to corroborate information from activation sequence mapping and adenosine responsiveness was not assessed.

Pace mapping provides a reliable, practical means to identify appropriate ablation sites in patients with adenosine-sensitive ventricular tachycardia. In contrast to its unreliability in identifying appropriate ablation sites in reentrant ventricular tachycardia associated with myocardial infarction,27,28 pace mapping may more precisely imitate activation patterns during ventricular tachycardia arising from a single anatomically discrete focus in a structurally normal heart.26 The optimal pace map in the study patients invariably had a short stimulus to QRS interval, providing additional evidence that an area of slow conduction did not participate in the genesis of this arrhythmia.29

In this study, the only complication related to catheter ablation was a new but self-limited nonsustained ventricular tachycardia early after direct current shock. Transient myocardial dysfunction and cardiac perforation have also been described after the application of direct current shocks in the right ventricle.30,31 For these reasons, radiofrequency energy remains the modality of first choice. The cause of unsuccessful radiofrequency
ablation in one patient is uncertain because the subsequent successful direct current shock was applied at the same site. It is possible that inadequate electrode contact precluded effective transmission of energy to the endocardium.

The apparent predilection of this tachycardia for specific sites within the free wall of the pulmonary infundibulum is unexplained. Histological abnormalities at the site of tachycardia origin could not be identified in a previously reported patient undergoing direct surgical ablation. In addition, multiple or “latent” foci, which could increase the risk of late recurrences, were not observed in any patient. A limited number of energy applications within a very circumscribed area resulted in long-term abolition of all ventricular tachycardias. Of interest, there appear to be no regional inhomogeneities of sympathetic innervation in patients with adenosine-sensitive ventricular tachycardia, at least as reflected by MIBG scanning. The mechanism of successful ablation appears unrelated to sympathetic denervation.

**Limitations**

Patients with ventricular tachycardia and structurally normal hearts, even those arising exclusively from the right ventricular outflow tract, are most likely a heterogeneous group who may not share a common mechanism. We included only patients in whom sustained tachycardias could be documented so that the response to adenosine could be unequivocally demonstrated. It is possible that many nonsustained ventricular tachycardias in patients without structural heart disease, including those with repetitive monomorphic ventricular tachycardia,12,13 also may be due to triggered automaticity. In this study, we did not observe adenosine sensitivity in idiopathic sustained ventricular tachycardias arising from other right or left ventricular sites. Further experience with the diagnostic use of adenosine may modify this preliminary observation.

**Clinical Implications**

The prognosis of patients with idiopathic right ventricular tachycardia is reported to be excellent,4,32,33 provided that occult right ventricular dysplasia or cardiomyopathy have been excluded. Pharmacological therapy is often effective in these patients4; however, recurrent disabling palpitations or intolerable side effects during medical therapy are common. In patients with adenosine-sensitive ventricular tachycardia, catheter ablation, particularly with the use of radiofrequency energy, appears to provide a safe and highly effective therapeutic alternative.

**Acknowledgments**

The authors gratefully acknowledge the expert assistance of Dr. Robert Henkin and Steven Karesh, who performed and interpreted the MIBG scans, and the technical assistance of Sally Botkin in the performance of electrophysiological studies and catheter ablation.

**References**

Adenosine-sensitive ventricular tachycardia. Clinical characteristics and response to catheter ablation.
D J Wilber, J Baerman, B Olshansky, J Kall and D Kopp

Circulation. 1993;87:126-134
doi: 10.1161/01.CIR.87.1.126
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1993 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/87/1/126

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/