Ventricular Tachycardia Originating From the Posterior Papillary Muscle in the Left Ventricle
A Distinct Clinical Syndrome

Harish Doppalapudi, MD; Takumi Yamada, MD; H. Thomas McElderry, MD; Vance J. Plumb, MD; Andrew E. Epstein, MD; G. Neal Kay, MD

Background—Several distinct forms of focal ventricular tachycardia (VT) from the left ventricle (LV) have been described. We report a new syndrome of VT arising from the base of the posterior papillary muscle in the LV.

Methods and Results—Among 290 consecutive patients who underwent ablation for VT or symptomatic premature ventricular complexes (PVCs) based on a focal mechanism, 7 patients were found to have an ablation site at the base of the posterior papillary muscle in the LV. All patients had normal LV systolic function and a normal baseline electrocardiogram. The electrocardiogram during VT or PVCs demonstrated a right bundle-branch block and superior-axis QRS morphology in all patients. VT was not inducible by programmed atrial or ventricular stimulation. In 2 patients with sustained VT, overdrive pacing neither terminated VT nor demonstrated any criterion for transient entrainment. Activation mapping localized the earliest site of activation to the base of the posterior papillary muscle in all patients. When Purkinje potentials were recorded at the site of successful ablation, these potentials preceded local ventricular muscle potentials during sinus rhythm. During VT or PVCs, however, the ventricular muscle potential always preceded the Purkinje potentials. After recurrence of VT or PVCs with standard radiofrequency ablation, irrigated ablation was successful in eliminating the arrhythmia in all patients. Over a mean follow-up period of 9 months, all patients have been free of PVCs and VT.

Conclusion—We present a distinct syndrome of VT arising from the base of the posterior papillary muscle in the LV by a nonreentrant mechanism. Ablation can be challenging, and irrigated ablation may be necessary for long-term success.

Key Words: tachycardia, ventricular □ papillary muscles, posterior □ catheter ablation

Several distinct forms of idiopathic ventricular tachycardia (VT) with diverse mechanisms have been described. In the left ventricle (LV), idiopathic VT may arise by a focal mechanism in the outflow tract, the aortic cusps, the mitral annulus, or in the epicardium or epicardial venous tissue.1–5 These focal forms of VT are usually based on cyclic-AMP–mediated triggered activity (delayed afterdepolarizations) and may be sensitive to adenosine.6 Idiopathic VT also may involve the fascicles of the specialized conduction system with either focal or reentrant mechanisms.1–2 Fascicular VTs usually involve the posterior fascicle of the left bundle branch but may also involve the anterior fascicle or the upper septum of the LV; these forms of VT are often based on a reentrant mechanism and may be sensitive to verapamil.7–9 We describe a distinct clinical syndrome of VT that is exercise induced and arises from the base of the posterior papillary muscle (PPM) in the LV.

Clinical Perspective p 29

Methods

Between January 2002 and August 2007, a total of 290 patients were referred to our institution for ablation of idiopathic ventricular arrhythmias based on a focal mechanism. The ventricular arrhythmias included sustained monomorphic VT in 52 patients, nonsustained VT in 56, and premature ventricular complexes (PVCs) in 182. The site of ablation was the right ventricle in 217 patients and the LV in 73. The sites of origin in the LV included the left coronary cusp of the aorta in 24 patients, the right coronary cusp in 14, the noncoronary cusp in 1, the aortic annulus below the aortic valve in 5, the epicardial surface of the LV in 8, the mitral annulus in 5, the fascicles of the left bundle branch in 8, the anterior papillary muscle in 1, and the PPM in 7. Patients with a reentrant mechanism of VT were excluded. The subjects of the present study are the 7 patients who underwent ablation of VT or symptomatic PVCs with the ablation site at the base of the PPM in the LV. The baseline characteristics of these patients, including age, sex, LV ejection fraction, presence of structural heart disease, baseline electrocardio-
gram (ECG), nature of the clinical arrhythmia, and ECG during arrhythmia, were recorded.

**Electrophysiological Study**

All patients underwent electrophysiological study and catheter ablation. Standard multielectrode catheters were placed in the coronary sinus, His bundle region, and the right ventricular apex. A quadripolar mapping/ablation catheter was advanced into the LV via a retrograde aortic approach. Programmed stimulation was performed from the right ventricular apex and the coronary sinus, with 1, 2, and 3 extrastimuli introduced after an 8-beat drive train. Isoproterenol infusion (2 to 8 µg/min) and intravenous boluses of epinephrine (0.05 mg) were administered when VT did not occur spontaneously or could not be induced by pacing techniques. When sustained VT was induced, pacing was performed from multiple sites in the left and right ventricles to determine whether VT could be transiently entrained or terminated.

**Mapping and Ablation**

Nonfluoroscopic electroanatomic mapping was performed with a quadripolar deflectable 3.5-mm-tip irrigated (Navistar Thermocool, Carto, Biosense Webster, Diamond Bar, Calif) or a 5-mm-tip mapping/ablation catheter (RPM, Boston Scientific, Natick, Mass) in addition to fluoroscopy. Radiofrequency (RF) current was delivered as high as 50 W, with the goal being to achieve a decrease in impedance of 8 to 10 Ω. RF current was delivered for 120 s in all patients, with no evidence of myocardial infarction scar or intraventricular conduction delay. The VT or PVCs morphology in 5 patients and a right bundle-branch block and left superior-axis QRS morphology in 2 patients (Figure 1). The mean QRS duration during VT or PVCs was 158 ms.

**Electrophysiological Findings**

The findings at electrophysiological study are summarized in Table 2. All patients had normal AH and HV intervals at entry.
baseline. Of the 2 patients with sustained VT as the clinical arrhythmia, VT occurred spontaneously during electrophysiological study in 1 patient and was induced by intravenous epinephrine in the other. Of the 5 patients with PVCs and nonsustained VT, 4 had spontaneous PVCs at electrophysiological study, whereas the other had PVCs and nonsustained VT after intravenous isoproterenol infusion. Programmed ventricular and atrial stimulation did not induce VT in any patient. Pacing from multiple sites in the 2 patients with sustained VT did not terminate the VT and did not demonstrate any criterion for transient entrainment. Sustained VT was hemodynamically well tolerated in both patients.

Mapping and Ablation
The local electrogram at the earliest site of ventricular activation during VT or PVCs was recorded 26 to 32 ms (mean 29 ± 2 ms) before the onset of the surface QRS complex (Figure 2). The surface ECG while pacing from this site showed a very close match to the surface ECG during VT or PVCs (Figure 3). In all patients, the earliest site of activation was localized to the base of the PPM in the LV (Figures 4 through 6). In the first 2 patients, ablation was performed at the site of earliest endocardial activation, with conventional RF energy delivered for 120 seconds from a 5-mm electrode with a maximum power of 50 W and target temperature of 60°C. In both of these cases, the PVCs or VT could be transiently suppressed by conventional RF energy but would recur within 60 minutes. In all patients, successful ablation required irrigated RF ablation (externally irrigated catheter in 5 patients and an internally irrigated catheter in 2 patients). The end point of the ablation procedure was the complete elimination of VT and PVCs, including nondiubility with intravenous epinephrine and isoproterenol. Patients were followed up for a mean of 9 months after ablation. All patients remained free of PVCs and VT. Echocardiograms with color Doppler examination were performed in all patients and demonstrated no evidence of significant mitral regurgitation at follow-up.

Discussion
We present a new syndrome of VT localized to the base of the PPM in the LV characterized by: (1) a normal baseline ECG and intracardiac conduction intervals with normal LV systolic function; (2) right bundle-branch block and superior-axis QRS morphology; (3) lack of inducibility with programmed ventricular and atrial stimulation; (4) absence of criteria for transient entrainment; (5) inducibility of VT or PVCs with intravenous isoproterenol or epinephrine; (6) earliest ventricular activation at the base of the PPM in the LV; and (7) absence of high-frequency potentials at the site of origin.

Table 2. Electrophysiological Findings and Follow-Up of the Study Patients

<table>
<thead>
<tr>
<th></th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
<th>Patient 5</th>
<th>Patient 6</th>
<th>Patient 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrophysiological study</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Induction of arrhythmia</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
<td>Isoproterenol</td>
<td>Spontaneous</td>
<td>Epinephrine</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Induced arrhythmia</td>
<td>PVCs/NSVT</td>
<td>PVCs/NSVT</td>
<td>PVCs/NSVT</td>
<td>Sustained VT</td>
<td>Sustained VT</td>
<td>PVCs/NSVT</td>
<td>PVCs/NSVT</td>
</tr>
<tr>
<td>Response to pacing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Termination</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>No</td>
<td>No</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Entrainment</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>No</td>
<td>No</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Mapping/ablation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Local activation time–QRS onset, ms</td>
<td>26</td>
<td>30</td>
<td>31</td>
<td>32</td>
<td>29</td>
<td>26</td>
<td>30</td>
</tr>
<tr>
<td>Mode of RF energy delivery</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of RF lesions</td>
<td>4</td>
<td>10</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>Follow-up duration, mo</td>
<td>14</td>
<td>14</td>
<td>11.5</td>
<td>10.5</td>
<td>9</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

NSVT indicates nonsustained VT; N/A, not applicable.
which suggests that the Purkinje system is not directly involved. Successful catheter ablation at this site uniformly required the use of cooled RF ablation, which suggests that the site of origin was within the papillary muscle itself, somewhat deep within the myocardium. Although the follow-up was short and the number of patients small, this form of monomorphic VT appears to have a benign prognosis, with no patient experiencing syncope or cardiac arrest despite recurrent arrhythmias for as long as 9 years.

ECG Characteristics
The QRS morphology during VT or PVCs was quite characteristic, with a right bundle-branch block and superior axis. The frontal-plane QRS axis was right superior in 5 patients and left superior in 2. The mean QRS duration during PVCs or VT was 158 ms.

Mapping and Catheter Ablation
In all cases, VT was not inducible with programmed ventricular or atrial stimulation; however, VT or PVCs were induced in all patients with intravenous epinephrine or isoproterenol. This observation, combined with the fact that sustained VT could not be terminated by overdrive pacing and with the absence of criteria for transient entrainment, suggests a nonreentrant mechanism for this arrhythmia: either abnormal automaticity or triggered activity. In addition, the absence of high-frequency potentials preceding the earliest local ventricular electrogram suggests that the Purkinje network is not involved in this arrhythmia. In our experience, local activation mapping appeared to be more useful than pace mapping, perhaps because of difficulty in maintaining stable contact of the catheter tip with the papillary muscle. In addition, it is likely that the site of origin may be somewhat deep relative to the endocardial surface of the PPM, as evidenced by the requirement for cooled ablation to achieve long-term success. Although Purkinje potentials were often recorded during sinus rhythm at the site of ablation, there was

Figure 2. Activation mapping: The 12-lead ECGs and bipolar electrograms from the proximal and distal bipoles of the ablation catheter (ABL-P and ABL-D, respectively) at the site of earliest local activation during VT are shown. The local ventricular electrogram recorded from the distal bipoles of the ablation catheter at this site preceded the onset of QRS by 29 ms. RF ablation at this site successfully terminated the VT.

Figure 3. Pace mapping: Left (from the same patient as in Figure 2) shows the 12-lead ECG during pacing in sinus rhythm from the site of earliest local activation during VT. A very close, but not perfect, match to the 12-lead ECG during VT was observed, as shown on the right.

Figure 4. Fluoroscopic images of the successful ablation site referenced to left ventriculograms in the right anterior oblique (top) and left anterior oblique (bottom) projections: The ablation catheter (ABL) seen in the upper and lower left is located at the base of the PPM in the LV. Left ventriculograms seen in the upper and lower right are produced by power injection of non-ionic contrast through a pigtail catheter (LV) positioned in the LV. The PPM is outlined by arrowheads in the upper and lower right. A decapolar diagnostic catheter is seen in the coronary sinus (CS), and 2 quadripolar diagnostic catheters are seen in the apex of the right ventricle (RV) and in the region of the His-bundle (HB).
typically a reversal of Purkinje–ventricular muscle electrograms during PVCs or VT (Figure 7). This finding may also suggest that the site of origin was deeper than the endocardial surface. Early and late diastolic potentials were not observed either in sinus rhythm or during VT or PVCs.

The site of successful ablation in all cases was at the base of the PPM in the LV. This site is located in the inferior wall slightly lateral to the septum and approximately one third of the distance from the mitral valve annulus to the apex of the LV (Figures 4 through 6). Achieving a stable catheter location at this site was challenging, probably because of the normally contracting papillary muscle. Irrigated RF current was required to achieve lasting ablation of the arrhythmogenic focus, which suggests that the site of origin may be deep beneath the endocardium. The fact that 2 patients had recurrent VT after conventional RF ablation had initially suppressed the arrhythmia is also supportive of this notion. It must be emphasized that steam pops and cardiac perforation are potential causes for concern with irrigated ablation, especially in patients with normal LV systolic function.

Meticulous mapping is essential before ablation to ensure that the delivered energy is focused at the base of the papillary muscle, where the myocardium is quite thick and therefore less prone to perforation. A low starting power (30 W) should be chosen and gradually titrated up (to a maximum of 50 W) to achieve an 8- to 10-Ω fall in impedance. The electrode tip temperature should be carefully monitored and maintained at <40°C. Energy delivery should be promptly terminated at any sudden rise in impedance. Postablation follow-up should include echocardiography or other imaging to assess for mitral regurgitation.

**Differential Diagnosis**

Posterior papillary muscle VT must be differentiated from other VTs originating in the LV. The forms of LV outflow tract VT are usually quite easily recognized, with an inferior QRS axis and earliest activation below the aortic annulus or within the right or left aortic cusps. In addition, idiopathic VT with a focal mechanism may occur on the epicardial surface of the LV and in some cases may be associated with early
activation in the anterior interventricular cardiac vein. Focal mechanisms of VT have also been described as arising from the mitral annulus. However, all of these forms of VT can be differentiated from a site of origin in the PPM by the ECG morphology and by careful mapping.

More importantly, PPM VT must be differentiated from other VTs with similar ECG morphology. VT seen in patients with structural heart disease is typically based on a reentrant mechanism and can occur with any QRS morphology. These patients can be identified by the presence of low-voltage regions on the endocardium or epicardium, isolated early diastolic potentials in sinus rhythm, mid-diastolic potentials in VT, and transient entrainment. None of these features was present in the cases of PPM VT. Left posterior fascicular VT is characterized by a right bundle-branch block and left superior QRS axis in the absence of structural heart disease.

In this arrhythmia, discrete fascicular potentials can be recorded over a significant portion of the interventricular septum at the site of successful ablation. In addition, this arrhythmia demonstrates the classic criteria for transient entrainment with rapid ventricular pacing and has been conclusively demonstrated to be due to a macro-reentrant circuit. Interfascicular reentry and automatic fascicular VTs usually occur in patients with dilated cardiomyopathy, although they may also occur in structurally normal hearts. Conduction system disease is often present in these patients, with an abnormal HV interval and/or prolonged QRS with bundle-branch block at baseline. Interfascicular VT is a reentrant arrhythmia that can be transiently entrained. It can be induced with programmed stimulation and can be terminated by pacing. It can be abolished by ablation of either the anterior or the posterior fascicle.

Automatic fascicular VT is distinguished from PPM VT mainly by the presence of high-frequency potentials that suggest origin from the specialized conduction system. This tachycardia is likely to originate from the Purkinje fibers, with high-frequency potentials preceding local myocardial activation during tachycardia. Mitral annular VT usually has an inferior QRS axis, but posterior mitral annular VT has a superior axis and right bundle-branch block QRS morphology. The site of origin of this VT can be localized to the posterior mitral annulus by careful mapping.

Limitations
A few limitations of this study should be emphasized. First, no attempt was made to identify the sensitivity of PPM VT to pharmacological agents such as adenosine or verapamil. Second, because the papillary muscle contracts with each systole, the requirement of irrigated RF may be more an issue of catheter stability than a matter of the depth of the focus. Finally, the lack of high-frequency potentials preceding the VT may not exclude involvement of Purkinje fibers deep relative to the endocardial surface.

Conclusion
This report describes a distinct clinical syndrome of catecholamine-sensitive VT arising from the base of the PPM of the LV that appears to be based on a focal (nonreentrant) mechanism. This VT is characterized by a right bundle-branch block and superior–QRS axis ECG morphology. The site of successful ablation is at the base of the PPM. Ablation of PPM VT can be quite challenging, and irrigated RF current is usually required to achieve lasting success.

Sources of Funding
None.

Disclosures
None.

References


---

**CLINICAL PERSPECTIVE**

We report a series of 7 patients with a distinct clinical syndrome of catecholamine-sensitive ventricular tachycardia arising from the base of the posterior papillary muscle in the left ventricle. All patients had normal left ventricular systolic function and a normal baseline electrocardiogram. The electrocardiogram during ventricular tachycardia or premature ventricular complexes demonstrated a right bundle-branch block and superior-axis QRS morphology. No patient suffered a cardiac arrest. Ventricular tachycardia originated by a focal mechanism, and activation mapping localized the earliest site of activation to the base of the posterior papillary muscle in the left ventricle in all patients. Ablation can be challenging, and irrigated ablation may be necessary for long-term success.
Ventricular Tachycardia Originating From the Posterior Papillary Muscle in the Left Ventricle: A Distinct Clinical Syndrome
Harish Doppalapudi, Takumi Yamada, H. Thomas McElderry, Vance J. Plumb, Andrew E. Epstein and G. Neal Kay

Circ Arrhythm Electrophysiol. 2008;1:23-29
doi: 10.1161/CIRCEP.107.742940

Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/1/1/23

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology 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: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org//subscriptions/