Idiopathic Focal Ventricular Arrhythmias Originating from the Anterior Papillary Muscle in the Left Ventricle

TAKUMI YAMADA, M.D.,* H. THOMAS MCELDERRY, M.D.,† YOSHIMASA MURAKAMI, M.D.,‡ YASUYA INDEN, M.D., ‡ HARI SH DOPPALAPUDI, M.D.,* NAOKI YOSHIDA, M.D.,‡ PAUL B. TABEREAUX, M.D.,* JAMES D. ALLRED, M.D.,* TOYOAKI MUROHARA, M.D.,‡ and G. NEAL KAY, M.D.*

*Division of Cardiovascular Disease, University of Alabama at Birmingham, Birmingham, Alabama, USA; †Division of Cardiology, Aichi Prefectural Cardiovascular and Respiratory Center, Ichinomiya; and ‡Department of Cardiology, Nagoya University Graduate School of Medicine, Nagoya, Japan

IVTs Arising from the LV Anterior Papillary Muscle. Introduction: Focal ventricular arrhythmias (VAs) have been reported to arise from the posterior papillary muscle in the left ventricle (LV). We report a distinct subgroup of idiopathic VAs arising from the anterior papillary muscle (APM) in the LV.

Methods and Results: We studied 432 consecutive patients undergoing catheter ablation for VAs based on a focal mechanism. Six patients were identified with ventricular tachycardia (VT, n = 1) or premature ventricular contractions (PVCs, n = 5) with the earliest site of ventricular activation localized to the base (n = 3) or middle portion (n = 3) of the LV APM. No Purkinje potentials were recorded at the ablation site during sinus rhythm or the VAs. All patients had a normal baseline electrocardiogram and normal LV systolic function. The VAs exhibited a right bundle branch block (RBBB) and right inferior axis (RIA) QRS morphology in all patients. Oral verapamil and/or Na+ channel blockers failed to control the VAs in 4 patients. VT was not inducible by programmed electrical stimulation in any of the patients. In 4 patients, radiofrequency current with an irrigated or conventional 8-mm-tip ablation catheter was required to achieve a lasting success. Two patients had recurrent PVCs after a conventional radiofrequency ablation with a 4-mm-tip ablation catheter had initially suppressed the arrhythmia.

Conclusions: VAs may arise from the base or middle portion of the APM and are characterized by an RBBB and RIA QRS morphology and focal mechanism. Catheter ablation of APM VAs is typically challenging, and creation of a deep radiofrequency lesion may be necessary for long-term success.

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ventricular tachycardia, ventricular ectopy, anterior papillary muscle, radiofrequency catheter ablation, normal heart

Introduction

The papillary muscles (PAMs) have been suggested to be potential sites of reentry that may contribute to the maintenance of ventricular fibrillation in animal models.1-3 Recently, the PAMs in the left ventricle (LV) have been reported to be arrhythmogenic in human hearts after myocardial infarction.4,5 In addition to these arrhythmias complicating structural heart disease, idiopathic focal ventricular arrhythmias (VAs) have been reported to originate from the posterior papillary muscle (PPM) in the LV.6 These VAs may be differentiated from other idiopathic VAs with a focal or reentrant mechanism.7-12 In this report, we describe a distinct subgroup of idiopathic VAs that arise from the anterior papillary muscle (APM) in the LV.

Methods

Between January 2002 and July 2008, a total of 432 patients were referred to our institutions for catheter ablation of idiopathic VAs based on a focal mechanism. The VAs included sustained monomorphic ventricular tachycardia (VT) in 77 patients, nonsustained VT in 82, and premature ventricular contractions (PVCs) in 273. The site of successful ablation was the right ventricle in 321 patients and the LV in 111. The sites of the VA origin in the LV included the left coronary cusp of the aorta in 26 patients, right coronary cusp in 17, noncoronary cusp in 1, aortic annulus below the aortic valve in 10, epicardial surface of the LV in 12, mitral annulus in 19, fascicles of the left bundle branch in 10, APM in 6, and PPM in 10. Patients with a reentrant mechanism of the VT were excluded. The subjects of the present study were the 6 patients who underwent catheter ablation of VT or symptomatic PVCs with the ablation site at the APM in the LV. The baseline characteristics of those patients, including the age, sex, LV ejection fraction, presence of structural heart disease, nature of the clinical arrhythmia, and electrocardiograms during the VAs, were recorded. Each
in addition to fluoroscopy, as previously reported.13 External-irrigated (Navistar ThermoCoolTM, Biosense Webster) or 7-French quadripolar deflectable 4- or 8-mm-tip non-irrigated (NavistarTM, Biosense Webster) ablation catheter with a 10-French deflectable intracardiac echocardiography probe (ACUSON AcuNavTM, 64-element, 5.5–10.0 MHz, Siemens). If a CARTO-based 3-dimensional ultrasound imaging system (CARTO SOUNDTM, Biosense Webster Inc.) was available, electroanatomic maps were created by adding the activation data during the VT or PVCs onto the 3-dimensional LV anatomical shells that were reconstructed with real-time integration of the intracardiac echocardiography. The upper figure on the right panel exhibits the image seen through the LV septum and the lower figure the image seen from the mitral annulus (MA). ABL(HB, RV) d(p) = the distal (proximal) electrode pair of the ablation (His bundle, right ventricular) catheter; APM = anterior papillary muscle; CS 1–5 = the first to fifth electrode pair of the coronary sinus catheter; PPM = posterior papillary muscle; V-QRS = the local ventricular activation time relative to the QRS onset.

Electrophysiological Study

For mapping and pacing, standard multielectrode catheters were introduced from the right femoral vein and placed in the coronary sinus, His bundle region, and right ventricular apex. A quadripolar mapping/ablation catheter was advanced into the LV via a retrograde aortic approach. Programmed electrical stimulation was performed from the right ventricular apex and coronary sinus, with 1, 2, and 3 extrastimuli introduced after an 8-beat drive train. During the procedures in the LV, intravenous heparin was administered to maintain an activated clotting time >250 seconds.

Mapping and Ablation

Nonfluoroscopic electroanatomic mapping was performed with a 7.5-French quadripolar deflectable 3.5-mm-tip external-irrigated (Navistar ThermoCoolTM, Biosense Webster) or 7-French quadripolar deflectable 4- or 8-mm-tip non-irrigated (NavistarTM, Biosense Webster) ablation catheter in addition to fluoroscopy, as previously reported.13,14 During mapping and ablation, the site and stability of the ablation catheter were assessed continuously and systematically by using transthoracic or intracardiac echocardiography with a 10-French deflectable intracardiac echocardiography probe (ACUSON AcuNavTM, 64-element, 5.5–10.0 MHz, Siemens). If a CARTO-based 3-dimensional ultrasound imaging system (CARTO SOUNDTM, Biosense Webster Inc.) was available, electroanatomic maps were created by adding the activation data during the VT or PVCs onto the 3-dimensional LV anatomical shells that were reconstructed with real-time integration of the intracardiac echocardiography, as previously reported13 (Fig. 1). Pace mapping was also performed at a pacing cycle length of 500 ms and a stimulus amplitude of 1 mA greater than the late diastolic threshold. The score for the pace mapping was determined from the R/S ratio and notch of the R wave in the 12-lead electrocardiogram, as previously reported (perfect pace mapping equal to 24 points).16 Radiofrequency (RF) current was used as the energy source for ablation. Irrigated RF current was delivered in the power-control mode starting at 30 W with an irrigation flow rate of 30 mL/min. The RF power was titrated to as high as 50 W, with the goal being to achieve a decrease in the impedance of 8–10 Ω and with care taken to limit the temperature to <40°C. Nonirrigated RF current was delivered with a target temperature of 55°C (8-mm tip) or 60°C (4-mm tip) and a maximum power output of 70 W (8-mm tip) to 50 W (4-mm tip). When an acceleration or reduction in the incidence of VT or PVCs was observed during the first 10 seconds of the application, the RF delivery was continued for 60–120 seconds. Otherwise, the RF delivery was terminated, and the catheter was repositioned. The endpoint of the catheter ablation was the elimination and noninducibility of VT or PVCs during an isoproterenol infusion (2–8 μg/min), intravenous boluses of epinephrine (0.05 mg), and burst pacing from the right ventricle (to a cycle length as short as 300 ms).

Postprocedure follow-up included clinic visits and telephone calls to all patients and their referring physicians. All patients underwent echocardiography with color Doppler after the ablation to evaluate the mitral valve, especially the degree of mitral regurgitation. The authors had full access to and take responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Clinical Characteristics

The baseline characteristics of the 6 patients are shown in Table 1. There were 3 men and 3 women between the ages of 39 and 78 years. Echocardiography demonstrated a normal LV systolic function and no evidence of structural heart disease in any patient. The clinically presenting arrhythmia was sustained VT in one patient and frequent PVCs without any runs of nonsustained VT in the other
## TABLE 1
Baseline and Arrhythmia Characteristics of the Study Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
<th>Case 6</th>
</tr>
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<td>54</td>
<td>75</td>
<td>39</td>
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<td>Normal</td>
<td>Normal</td>
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<td>Normal</td>
</tr>
<tr>
<td>Arrhythmia Pattern</td>
<td>Sustained VT</td>
<td>PVCs</td>
<td>PVCs</td>
<td>PVCs</td>
<td>PVCs</td>
</tr>
<tr>
<td>Duration of symptoms</td>
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<td>6 y</td>
<td>2 y</td>
<td>1 y</td>
<td>1 M</td>
</tr>
<tr>
<td>Electrocardiogram QRS morphology</td>
<td>RBBB/RIA</td>
<td>RBBB/RIA</td>
<td>RBBB/RIA</td>
<td>RBBB/RIA</td>
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</tr>
<tr>
<td>QRS duration, ms</td>
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<td>194</td>
<td>148</td>
<td>152</td>
<td>160</td>
</tr>
<tr>
<td>Ineffective AADs</td>
<td>Verapamil</td>
<td>Disopyramide, flecainide</td>
<td>Mexiletine</td>
<td>N/A</td>
<td>Mexiletine</td>
</tr>
</tbody>
</table>

AADs = antiarrhythmic drugs; LV = left ventricular; m = month; N/A = not applicable; PVCs = premature ventricular contractions; RBBB = right bundle branch block; RIA = right inferior axis; VT = ventricular tachycardia; y = year.

The 5 patients with frequent PVCs had symptoms of palpitations and light headiness that progressively increased in frequency. Four patients exhibited significant episodes of symptoms that worsened with exertion. None of the 6 patients suffered from cardiac arrest or syncope. The duration of symptoms prior to the study ranged between 1 month and 6 years.

**Electrocardiographic Findings**

At baseline, the 12-lead electrocardiograms exhibited sinus rhythm with a QRS duration of <120 ms in all patients, with no evidence of any myocardial infarction scar or intraventricular conduction delays. One patient (case 4) exhibited a Brugada type II electrocardiogram. In all 6 patients, the VT or PVCs exhibited a right bundle branch block (RBBB) and right inferior axis (RIA) QRS morphology with an early precordial transition of < lead V1, qR or qr pattern in lead aVR, and rS pattern in lead V6 (Fig. 2). The mean QRS duration during the VT or PVCs was 168 ± 19 ms. In all patients except for case 4 with a Brugada electrocardiogram, the administration of at least one antiarrhythmic drug including verapamil and/or a Na⁺ channel blocker failed to control the VAs.

![Figure 2. The 12-lead electrocardiograms of the QRS complexes during ventricular tachycardia or premature ventricular contractions in each patient.](image-url)
Electrophysiological Findings

The findings from the electrophysiological study are summarized in Table 2. At baseline, the AH and HV intervals were normal, and PVCs with a QRS morphology identical to the clinical VT or PVCs were spontaneous and frequent in all patients. Programmed ventricular or atrial stimulation did not induce VT in any of the patients.

Mapping and Ablation

In all cases, at the earliest site of ventricular activation during the PVCs, the local ventricular electrogram preceded the onset of the surface QRS complex by 18–35 ms (mean 27 ± 7 ms), and no atrial activity, high-frequency potentials, or early or late diastolic potentials were recorded during either sinus rhythm or the PVCs (Fig. 1). The surface electrocardiogram while pacing from that site exhibited a very close match to the surface electrocardiogram during the VT or PVCs in 5 (Fig. 3) and a poor match in 1 (case 2). In all patients, transthoracic (n = 2) or intracardiac echocardiography (n = 4) and left ventriculography revealed that the earliest site of ventricular activation was localized at the base (n = 3) or middle portion (n = 3) of the APM in the LV (Figs. 4 and 5). Catheter ablation was performed at the site of the earliest endocardial ventricular activation. In cases 1, 2, 5, and 6, RF current was delivered using an irrigated ablation catheter. In cases 1, 5, and 6, the PVCs could be successfully eliminated, whereas in case 2, the PVCs could be suppressed only transiently. In case 2, a unidirectional irrigated catheter was then replaced by a bidirectional nonirrigated 8-mm-tip catheter for better manipulation, and conventional high power RF current finally eliminated the PVCs. In cases 3 and 4, RF current delivered using a nonirrigated 4-mm-tip ablation catheter eliminated the PVCs. Acceleration of the PVCs was observed during the RF applications in all cases. During the short duration (<3 weeks) of follow-up, PVCs with the same QRS morphology as the targeted ones during the ablation procedure recurred in cases 2, 3, 4, and 6. Only cases 2 and 6 underwent a second procedure. In the second procedure, the same approach was used and the PVCs

![Figure 3. An excellent pace map obtained during pacing at the successful ablation site. PVC = premature ventricular contraction.](image-url)
were successfully eliminated. Cases 3 and 4 refused a second procedure because there had been a significant reduction in the incidence of the PVCs (15,000 to <2,000 beats/day and 12,000 to <1,000 beats/day on the Holter monitoring, respectively) and a significant improvement in their symptoms. During the follow-up period (7 ± 4 months) after the last procedure, 4 patients (cases 1, 2, 5, and 6) have remained free of the VAs and the other two patients (cases 3 and 4) have exhibited no worsening of the VAs without any antiarrhythmic drugs. Echocardiograms with color Doppler examination were performed in all patients and demonstrated no evidence of significant mitral regurgitation at the follow-up.

Discussion

Several distinct forms of idiopathic LV VAs with diverse mechanisms have been described. Idiopathic VAs may arise by a focal mechanism in the ventricular outflow tract, aortic cusps, mitral annulus, or in the epicardium or epicardial venous tissue.7-12 Those VAs are usually based on cyclic-AMP-mediated triggered activity (delayed after depolarizations) and may be sensitive to adenosine.12 Idiopathic VTs also may involve the fascicles with either focal or reentrant mechanism.7,8 Fascicular VTs are often based on a reentrant mechanism and may be sensitive to verapamil.17-19 This study presents a distinct subgroup of idiopathic VAs localized to the LV APM characterized by (1) an RBBB and RIA QRS morphology; (2) refractoriness to verapamil and Na+ channel blockers, which suggests that the conduction system or myocytes sensitive to these antiarrhythmic drugs are not directly involved; (3) a tendency for PVCs rather than VT; (4) inducibility with exertion; (5) lack of inducibility with programmed ventricular or atrial stimulation; (6) the earliest ventricular activation at the base or middle portion of the LV APM; (7) the absence of high-frequency potentials at the site of origin; and (8) the requirement for high RF power to achieve a long-term successful ablation. Other than the QRS axis, these features are similar to those described with idiopathic PPM VAs.6 Therefore, it seems as though idiopathic PAM VAs have similar characteristics regardless of which muscle is the site of origin.

In all cases, VT was not inducible with programmed ventricular or atrial stimulation. That observation, combined
with the fact that VT was less common than PVCs, suggests a nonreentrant mechanism for this arrhythmia: either abnormal automaticity or triggered activity. Abnormal automaticity may be suggested as the mechanism because acceleration of the PVCs occurred during the RF applications in all the patients in this study. In addition, the absence of high-frequency potentials preceding the earliest local ventricular electrogram suggests that the Purkinje network was not involved in this arrhythmia. RF current with an irrigated or conventional 8-mm-tip ablation catheter was required to achieve a lasting ablation of the arrhythmogenic focus, which suggests that the site of origin might have been within the APM itself and deep beneath the endocardium of the APMs. The fact that 2 patients had recurrent PVCs after conventional RF ablation with a 4-mm-tip ablation catheter had initially suppressed the arrhythmia is also supportive of this notion. In our experience, the local activation mapping appeared to always be the most useful. Pace mapping was helpful, but sometimes unreliable. That was probably because of the difficulty in maintaining the stable contact of the catheter tip with the APMs and/or a deep site of origin relative to the endocardial surface of the APMs. Achieving a stable catheter location at this site was challenging, probably because of the vigorous motion associated with normal APM contraction. It must be emphasized that steam pops and cardiac perforation are potential causes for concern with irrigated or conventional high power ablation, especially in patients with normal LV systolic function. Meticulous mapping is essential before the ablation to ensure that the delivered energy is focused at the base or middle portion of the APMs, where the myocardium is quite thick and therefore less prone to perforation. A low starting power output (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 less than 40°C. Energy delivery should be promptly terminated with any sudden rise in impedance. Postablation follow-up should include echocardiography or other imaging methods to assess for mitral regurgitation.

Differential Diagnosis

APM VAs must be differentiated from other LV VAs. The forms of LV outflow tract VAs are usually quite easily recognized, with a later precordial transition, QS pattern in lead aVR, and an R or Rs pattern in lead V6, and the earliest ventricular activation around the aortic root. Idiopathic focal VAs arising from the epicardial surface of the LV may be associated with early activation in the anterior interventricular cardiac vein. Focal VAs have also been described as arising from the mitral annulus. Especially important, a site of origin in the anterolateral to lateral aspects of the mitral annulus may be relatively close to the APM. However, all of those forms of VAs can be differentiated from APM VAs by the electrocardiographic and electrophysiological characteristics such as the absence of an rS pattern in lead V6 and the presence of an atrial activity at the earliest ventricular activation site and by careful mapping. Left posterior fascicular VT and idiopathic PPM VAs can be easily distinguished from APM VAs by a superior QRS axis. More importantly, APM VAs must be differentiated from other VTs with a similar electrocardiographic morphology. Similar to APM VAs, idiopathic left anterior fascicular VT is characterized by an RBBB and RIA QRS morphology. However, discrete fascicular potentials can be recorded over a significant portion of the anterolateral LV wall at the site of the successful ablation for these reentrant VTs. In addition, anterior fascicular VT is based on a macroreentrant circuit, which contains some tissues sensitive to verapamil and Na+ channel blockers. 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 those 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 induced and terminated by pacing. It can be abolished by ablation of either the anterior or posterior fascicle. Automatic fascicular VT is distinguished from APM VT mainly by the presence of
high-frequency potentials that suggest an origin from the Purkinje fibers.

### Study Limitations

Several limitations of this study should be addressed. First, although the diagnosis of APM VAs mainly depended on the echocardiographic demonstration of the anatomic location of the successful ablation sites, this technique may lead to some imprecision for determining the exact catheter tip position particularly when transthoracic echo was used. Second, no attempt was made during the electrophysiological study to identify the sensitivity of the APM VAs to pharmacological agents difficult. Third, the lack of high-frequency potentials preceding the local ventricular electromagnets may not exclude involvement of Purkinje fibers deep to the endocardial surface.

### Conclusions

This report describes a distinct subgroup of idiopathic VAs arising from the APM in the LV characterized by an RBBB and RIA QRS morphology, a focal (nonreentrant) mechanism and an origin at the base or middle portion of the APM. Catheter ablation of APM VAs can be quite challenging, and RF current with an irrigated or conventional 8-mm-tip ablation catheter is usually required to achieve lasting success.

### References