Electrocardiographic and Electrophysiological Characteristics in Idiopathic Ventricular Arrhythmias Originating From the Papillary Muscles in the Left Ventricle
Relevance for Catheter Ablation

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Background—Idiopathic ventricular arrhythmias (VAs) can originate from the left ventricular papillary muscles (PAMs). This study investigated the electrophysiological characteristics of these VAs and their relevance for the results of catheter ablation.

Methods and Results—We studied 19 patients who underwent successful catheter ablation of idiopathic VAs originating from the anterior (n=7) and posterior PAMs (n=12). Although an excellent pace map was obtained at the first ablation site in 17 patients, radiofrequency ablation at that site failed to eliminate the VAs, and radiofrequency lesions in a relatively wide area around that site were required to completely eliminate the VAs in all patients. Radiofrequency current with an irrigated or nonirrigated 8-mm-tip ablation catheter was required to achieve a lasting ablation of the PAM VA origins. During 42% of the PAM VAs, a sharp ventricular prepotential was recorded at the successful ablation site. In 9 (47%) patients, PAM VAs exhibited multiple QRS morphologies, with subtle, but distinguishable differences occurring spontaneously and after the ablation. In 7 (78%) of those patients, radiofrequency lesions on both sides of the PAMs where pacing could reproduce an excellent match to the 2 different QRS morphologies of the VAs were required to completely eliminate the VAs.

Conclusions—Radiofrequency catheter ablation of idiopathic PAM VAs is challenging probably because the VA origin is located relatively deep beneath the endocardium of the PAMs. PAM VAs often exhibit multiple QRS morphologies, which may be caused by a single origin with preferential conduction resulting from the complex structure of the PAMs. (Circ Arrhythm Electrophysiol. 2010;3:324-331.)

Key Words: idiopathic ventricular arrhythmias ■ cardiac arrhythmias ■ electrophysiology ■ papillary muscles ■ radiofrequency catheter ablation

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strated no evidence of structural heart disease in any patient. The sites of origin of VA included the aortic root in 47 (29.6%) patients, aortomitril continuity in 12 (7.5%), epicardial surface of the LV in 17 (10.7%), mitral annulus in 24 (15.1%), fascicles of the left bundle branch in 38 (23.9%), anterior papillary muscle (APM) in 7 (4.4%), posterior papillary muscle (PPM) in 12 (7.5%), and other sites in 2 (1.3%). Although the subjects of the present study were the 19 patients who underwent successful catheter ablation of VT or symptomatic PVCs with the ablation site at the APM or PPM, 1 patient with a PVC origin in the APM that was not successfully ablated was also included. The baseline characteristics, including age, sex, nature of the clinical arrhythmia, and 12-lead ECG during the VAs were recorded. Each patient gave written informed consent, and all antiarrhythmic drugs were discontinued for at least 5 half-lives before the study.

Electrophysiological Study
For mapping and pacing, standard multielectrode catheters were placed in the coronary sinus. His bundle region, and right ventricular apex through the right femoral vein. A quadripolar ablation catheter was advanced into the LV through a retrograde aortic approach. Induction of the VT or PVCs was attempted by programmed electrical stimulation from the right ventricular apex and coronary sinus, with 1, 2, and 3 extrastimuli introduced after an 8-beat drive train, if necessary, with the addition of an isoproterenol infusion or IV administration of boluses of epinephrine (0.05 mg). During the procedures in the LV, IV heparin was administered to maintain an activated clotting time of >250 seconds.

Mapping and Radiofrequency Catheter Ablation
Activation and pace mapping was performed in all cases to identify the site of the VA origin. In some patients when VT or PVCs were frequent, electroanatomic mapping was performed as previously reported.14,15 During mapping and ablation around the PAMs, the catheter was advanced into the LV at a pacing cycle length of 500 milliseconds and 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 ECG as previously reported.17

Radiofrequency current was delivered at myocardial sites exhibiting the earliest bipolar activity or a local unipolar QS pattern or at a Purkinje network with an early activity preceding the QRS onset during the VT or PVCs. Irrigated radiofrequency current was delivered in the power control mode, starting at 30 W with an irrigation flow rate of 30 mL/min using a 7.5-F deflectable 3.5-mm-tip external-irrigated ablation catheter. The radiofrequency power was titrated to as high as 50 W with the goal of being able to achieve a decrease in the impedance of 8 to 10 Ω and with care taken to limit the temperature to <40°C. Nonirrigated radiofrequency current was delivered using a bidirectional ablation catheter with a target temperature of 55°C (8-mm tip) or 60°C (4-mm tip) and maximum power output of 70 W (8-mm tip) or 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 radiofrequency delivery was continued for 60 to 120 seconds; otherwise, the radiofrequency delivery was terminated, and the catheter was repositioned. The end-point of the catheter ablation was the elimination and noninducibility of VT or PVCs during an isoproterenol infusion (2 to 8 µg/min), IV boluses of epinephrine (0.05 mg), and burst pacing from the right ventricle (to a cycle length as short as 300 milliseconds).

Postprocedure follow-up included clinic visits and telephone calls to all patients and their referring physicians. All patients who reported symptoms were given an event monitor to document the

The intracardiac echocardiographic images of the successful ablation site. Two-dimensional intracardiac echocardiographic short-axis images at the level of the PAMs demonstrated that the ablation catheter was positioned on the anterior and posterior sides of the APM. The arrowheads indicate the acoustic shadow from the ablation catheter. ABL indicates ablation catheter.

Table 1. Clinical Characteristics of Patients with Ventricular Arrhythmias Originating from the Papillary Muscles in the Left Ventricle

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Origin</th>
<th>Age, yrs</th>
<th>Sex</th>
<th>Type</th>
<th>Duration of Symptoms, yrs</th>
<th>LVEF, %</th>
<th>Ex-Triggered</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>APM</td>
<td>57</td>
<td>M</td>
<td>SVt</td>
<td>0.2</td>
<td>58</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>APM</td>
<td>54</td>
<td>F</td>
<td>PVC</td>
<td>6.0</td>
<td>61</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>APM</td>
<td>75</td>
<td>F</td>
<td>PVC</td>
<td>2.0</td>
<td>66</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>APM</td>
<td>39</td>
<td>M</td>
<td>PVC</td>
<td>1.0</td>
<td>77</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>APM</td>
<td>73</td>
<td>M</td>
<td>PVC</td>
<td>0.1</td>
<td>59</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>APM</td>
<td>78</td>
<td>M</td>
<td>PVC</td>
<td>5.0</td>
<td>57</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>APM</td>
<td>78</td>
<td>F</td>
<td>NSVt</td>
<td>0.3</td>
<td>63</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>PPM</td>
<td>46</td>
<td>M</td>
<td>SVt</td>
<td>0.5</td>
<td>69</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>PPM</td>
<td>42</td>
<td>M</td>
<td>SVt</td>
<td>9.0</td>
<td>72</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>PPM</td>
<td>67</td>
<td>F</td>
<td>NSVt</td>
<td>1.0</td>
<td>58</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>PPM</td>
<td>68</td>
<td>M</td>
<td>PVC</td>
<td>4.0</td>
<td>56</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>PPM</td>
<td>82</td>
<td>M</td>
<td>NSVt</td>
<td>1.0</td>
<td>57</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>PPM</td>
<td>48</td>
<td>F</td>
<td>PVC</td>
<td>0.1</td>
<td>60</td>
<td>-</td>
</tr>
<tr>
<td>14</td>
<td>PPM</td>
<td>47</td>
<td>F</td>
<td>NSVt</td>
<td>4.0</td>
<td>66</td>
<td>+</td>
</tr>
<tr>
<td>15</td>
<td>PPM</td>
<td>51</td>
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<td>SVt</td>
<td>0.1</td>
<td>58</td>
<td>+</td>
</tr>
<tr>
<td>16</td>
<td>PPM</td>
<td>34</td>
<td>M</td>
<td>PVC</td>
<td>7.0</td>
<td>75</td>
<td>-</td>
</tr>
<tr>
<td>17</td>
<td>PPM</td>
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<td>M</td>
<td>NSVt</td>
<td>2.0</td>
<td>59</td>
<td>-</td>
</tr>
<tr>
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<td>PPM</td>
<td>59</td>
<td>F</td>
<td>SVt</td>
<td>3.0</td>
<td>60</td>
<td>+</td>
</tr>
<tr>
<td>19</td>
<td>PPM</td>
<td>57</td>
<td>M</td>
<td>PVC</td>
<td>0.3</td>
<td>64</td>
<td>+</td>
</tr>
</tbody>
</table>

Ex indicates exercise; LVEF, left ventricular ejection fraction; NSVt, nonsustained VT; SVt, sustained VT.
cause of the symptoms. Successful catheter ablation was defined as no recurrence of any VAs during >6 months of follow-up. 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.

ECCG Analysis
Twelve-lead ECCGs during the VAs and pace mapping were recorded digitally at a sweep speed of 100 to 200 mm/s in all patients for offline analysis. The QRS duration, R-wave amplitude, and depth of the S wave in the limb leads were measured with electronic calipers by 2 experienced investigators blinded to the site of the origin. If there were discrepancies between those results, they were adjudicated by a third investigator.

Statistical Analysis
The continuous variables are expressed as the median with the first and third quartiles (Q1 to Q3), as appropriate. The comparisons of the continuous variables between the 2 groups were analyzed with Wilcoxon signed rank test. Statistical significance was selected at a value of $P<0.05$.

Results
Clinical Characteristics
The baseline characteristics of the main study patients are shown in Table 1. In the 13 men and 6 women aged 34 to 82 years (median, 57 years; Q1 to Q3, 46 to 68 years), echocardiography demonstrated normal LV systolic function (median LV ejection fraction, 0.63; Q1 to Q3, 0.60 to 0.69) and no evidence of structural heart disease. The clinically presenting arrhythmia was sustained VT in 5 patients, nonsustained VT in 5 patients, and frequent PVCs without any runs of nonsustained VT in the remaining 9 patients. These 9 patients with frequent PVCs had symptoms of palpitations and light-headedness that progressively increased in frequency. Ten patients exhibited significant worsening of symptoms with exertion. None of the 19 patients suffered from cardiac arrest or syncope. The duration of symptoms before the study ranged from 1 month to 9 years.

Mapping and Catheter Ablation
The results of mapping and catheter ablation of the PAM VAs are shown in Table 2. In all patients, transthoracic (n=10) and intracardiac echocardiography (n=13) and left ventriculography revealed that the successful ablation sites were localized at the base of the PAMs in the LV. The successful ablation sites of PAM VAs were also identified in specific regions on biplane fluoroscopic images (Figures 2 and 3). They were always seen approximately halfway between the His bundle catheter and left lateral cardiac silhouette in the left anterior oblique projection and approximately halfway between the coronary sinus catheter and apical cardiac silhouette in the right anterior oblique projection. The successful ablation sites of the APM VAs were in the lateral region, whereas those of the PPM VAs were in the posterior region in the left anterior oblique projection. The definitive difference in the fluoroscopic images between the successful ablation sites of APM VAs and PPM VAs was the direction of the ablation catheter through a retrospective transaortic approach by deflecting the loop of the ablation catheter upward for APM VAs while keeping the tip of the ablation catheter downward for PPM VAs.
VAs. At the successful ablation sites, a small-amplitude, relatively low-frequency potential was observed in the local ventricular electrogram preceding the QRS onset of VAs in 8 patients (APM, 3; PPM, 5) (Figure 2). Although an excellent pace map with a score of ≥21 was obtained at the first ablation site in 17 patients, radiofrequency ablation at that site failed to eliminate the VAs in all of these patients, although typically with a reduction in PVC frequency and a slight change in the QRS morphology (only notches in the precordial leads). The site of the earliest activation moved to a site adjacent to the first radiofrequency lesion where the local ventricular activation time relative to the QRS onset was later than that at the first ablation site. The same events occurred several times, and additional ablation targeting a relatively wide area around the first radiofrequency lesion was required to completely eliminate the VAs (Figure 4). In the remaining 2 patients without an excellent pace map, the electroanatomic map exhibited a larger area of nearly simultaneous early activation in the PAMs, and further radiofrequency lesions were required to completely eliminate the VAs compared with that in the 17 patients with an excellent pace map. There were no correlations between the pace map score and earliest local ventricular activation time relative to the QRS onset. It was noted that during mapping around the PAMs, mechanical compression by the mapping catheter never suppressed the VAs.

**Follow-Up and Repeated Procedures**

During the follow-up, VAs with a similar QRS morphology to the targeted ones during the ablation procedure recurred in 11 (58%) patients, including all 4 in whom there was initial suppression of the VAs using a 4-mm-tip ablation catheter, and 9 of those patients underwent a second procedure. During the second procedure, an irrigated or conventional 8-mm-tip ablation catheter with the same approach always was used, and the VAs were successfully eliminated in all the patients. The remaining 2 patients declined a second procedure because there had been a significant reduction in the incidence of the PVCs and significant improvement in their symptoms.

**Figure 2.** Cardiac tracings (left) and fluoroscopic images (right) exhibiting the successful ablation site of the PVCs originating from the LV PPM. The first beat is a sinus beat and the second a PVC. Note that at the successful ablation site, no Purkinje potentials were observed during sinus rhythm, and a spiky prepotential (arrowhead) was observed in the local ventricular activity during the PVC. ABL indicates ablation catheter; CS 1 to 5, the first to fifth electrode pair of the coronary sinus catheter; d, distal; HB, His bundle; LAO, left anterior oblique view; p, proximal; RAO, right anterior oblique view; RV, right ventricular catheter; V-QRS, local ventricular activation time relative to the QRS onset.

**Figure 3.** The 12-lead ECGs of the PVCs presumably originating from the LV APM and pace map obtained by pacing from the epicardial surface opposite the APM (left) and fluoroscopic images (right) exhibiting the ablation catheter positioned at the endocardial site of the earliest ventricular activation during the PVCs and at the epicardial site of pace mapping. ABL indicates ablation catheter; CS, coronary sinus; EpiPM, epicardial pace map; HB, His bundle; LAO, left anterior oblique view; RAO, right anterior oblique view; RV, right ventricular catheter.
During a follow-up period (median, 21 months; Q1 to Q3, 14 to 37 months) after the last procedure, 17 patients remained free of the VAs, and the other 2 exhibited no worsening of the VAs without any antiarrhythmic drugs. In the 17 patients with complete suppression of VAs, an irrigated (n=15) or nonirrigated (n=2) 8-mm-tip ablation catheter was required to achieve lasting success. Echocardiograms with color Doppler examination demonstrated no evidence of significant mitral regurgitation in any patient at the follow-up.

Overall Results
The successful ablation sites were located on both sides of the base of the PAMs (Figure 5). For APM VAs, the anterior and posterior sides were equally likely to be the site of successful ablation, whereas the septal side was the predominant site of successful ablation for PPM VAs (Figure 5). In 7 patients, radiofrequency ablation on both sides of the PAM was required to completely eliminate the VAs. A subtle, but distinguishable change in the QRS morphology of the VAs occurred in the limb leads during the first procedure in 3 of these patients (spontaneously in 2 [Figure 6] and after several radiofrequency lesions in 1 [case 15]) and between the original and recurrent VAs in the remaining 4. Figure 5 demonstrates this typical change in ECG morphology. For APM VAs, the R-wave amplitude in the inferior leads was...
significantly larger for those VAs that were ablated on the anterior side than for those ablated on the posterior side of the APMs (lead II median [Q1 to Q3], 0.55 [0.46 to 0.69] versus 0.31 [0.28 to 0.33]; \( P = 0.012 \); lead III, 1.26 [1.17 to 1.40] versus 0.90 [0.76 to 1.02]; \( P = 0.025 \); aVF, 0.87 [0.74 to 1.09] versus 0.50 [0.30 to 0.73]; \( P = 0.012 \)). In addition, the R-wave amplitude in lead aVR was significantly smaller (median [Q1 to Q3], 0.22 [0.09 to 0.37] versus 0.28 [0.16 to 0.56]; \( P = 0.017 \)), the depth of the S-waves in lead aVL was significantly larger (1.03 [0.94 to 1.09] versus 0.91 [0.83 to 0.93]; \( P = 0.012 \)), and the QRS duration was significantly longer (193 [187 to 197] versus 187 [178 to 188]; \( P = 0.012 \)) when the ablation site was on the anterior side than on the posterior side of the APMs. For PPM VAs, the depth of the S-waves in the inferior leads was significantly smaller (lead II median [Q1 to Q3], 0.87 [0.76 to 1.14] versus 1.06 [0.84 to 1.22]; \( P = 0.028 \); lead III, 0.80 [0.54 to 0.93] versus 1.15 [0.88 to 1.35]; \( P = 0.041 \); aVF, 0.80 [0.63 to 0.93] versus 1.15 [0.77 to 1.23]; \( P = 0.022 \)), the R-wave amplitude in lead aVL was significantly smaller (0.43 [0.29 to 0.46] versus 0.70 [0.48 to 0.77]; \( P = 0.001 \)), and the QRS duration was significantly longer (179 [177 to 189] versus 175 [171 to 176]; \( P = 0.001 \)) during the VAs ablated on the lateral side than those ablated on the septal side. In 6 patients (APM, 2; PPM, 4), these changes in the QRS morphologies occurred spontaneously before the ablation, and fusion of these different QRS morphologies was observed (Figures 4 and 6). In 4 of these patients, radiofrequency ablation on both sides of the PAM was required to completely eliminate the VAs. In all 11 patients with these changes in the QRS morphology of the VAs, pacing from either side of the PAM could reproduce an excellent match to the 2 different QRS morphologies of the VAs (Figure 4).

**Mapping in the Unsuccessfully Ablated Case**

In the patient in whom PVCs were not successfully ablated, the PVCs exhibited clinical and ECG characteristics consistent with APM VAs (Figure 3). Endocardial activation mapping during the PVCs revealed that the earliest ventricular activation preceded the QRS onset by 36 milliseconds at the APM. Pacing from all sites along the APM never produced an excellent match to the QRS complex of the PVCs, and multiple applications of irrigated radiofrequency current delivered around the site of the earliest ventricular activation never interrupted the PVCs. Epicardial mapping through a subxiphoid pericardial approach was then performed, and it revealed that the epicardial ventricular activation never preceded the QRS onset during the PVCs. Pacing from the epicardial surface opposite to the APM produced a wider QRS complex with a greater precordial maximal deflection index as calculated by dividing the shortest time to the maximum deflection in any precordial lead by the QRS duration as compared with the PVCs (Figure 3). Consequently, catheter ablation was abandoned.

**Discussion**

The detailed electrophysiological features of idiopathic LV VAs have been increasingly recognized as the techniques and technologies in this area have made remarkable progress. \(^1\)\(^-\)\(^13\) Idiopathic VAs arising from the PAMs in the LV form a distinct subgroup of VAs that occur in patients with structurally normal hearts. These VAs are often exercise induced or require IV isoproterenol or epinephrine for induction. The fact that PAM VAs cannot be transiently entrained, the relatively late diastolic activation time at the site of ablation, the observation that the first beat of the tachycardia has the same activation sequence as subsequent beats, and the lack of fractionated potentials at the site of ablation all suggest a focal mechanism rather than reentry. Transthoracic and intracardiac echocardiography and irrigated ablation catheters usually are required for ablation of these VAs. However, they are not routinely used for the mapping and ablation of the usual idiopathic VAs in most electrophysiology centers, considering their complexity, high cost, and potential for creating large ablation lesions. Hence, making an accurate diagnosis of VAs originating from the PAMs before the procedure is important to clinicians. The algorithms based on ECG characteristics, fluoroscopic location, and electrophysiological features acquired by activation and pace mapping, programmed stimulation, and entrainment pacing may differentiate PAM VAs from other LV VAs and thereby help with making a decision on whether additional complex mapping measures, such as transthoracic and intracardiac echocardiography or a 3D mapping system should be used.

Ablation of most idiopathic LV VAs from the endocardial surface by radiofrequency current usually is successful with a 4-mm-tip nonirrigated catheter. Long-term follow-up of patients after ablation of most idiopathic VAs is limited, but the risk of recurrence generally is low. \(^1\)\(^-\)\(^10\)\(^,\)\(^19\) In this study, however, the recurrence rate for VAs originating from the PAMs was relatively high, and the use of high radiofrequency power settings delivered from an irrigated or nonirrigated 8-mm-tip ablation catheter was required to achieve lasting ablation. The possible explanation for the technical challenges of endocardial radiofrequency catheter ablation of these VAs may be that the origin is located on the epicardial surface opposite the targeted PAMs or deep relative to the endocardial surface of the PAMs. However, as our case with epicardial mapping demonstrated, an epicardial origin is less likely, and a deep intramural focus is more likely.
The difficulty in maintaining stable contact of the catheter tip with the PAMs may be another mechanism for the high power requirement. In fact, achieving a stable catheter location in contact with the PAMs can be challenging despite intensive monitoring with echocardiography because of the vigorous motion associated with normal PAM contraction. Further support for a deep origin of the VAs is provided by the observation that suppression of VAs by mechanical pressure was never observed in this series.

In identifying an origin of idiopathic LV VAs, activation mapping is the most reliable method, although pace mapping usually provides helpful clues. In this study, a discrete radiofrequency lesion at the site with an excellent pace map usually failed to eliminate the PAM VAs, although there was often a change in the QRS morphology. As a result, several further radiofrequency lesions always were required to suppress the PAM VAs. These findings suggest that the site of PAM VA origin might have been located away from the breakout site, which can be recognized as the site with the best pace map. This finding adds further support to the concept that VAs originate from a deep site relative to the surface of the PAMs. The patients without an excellent pace map in this study might have exhibited no discrete breakout sites because of a deeper VA origin, resulting in further radiofrequency lesions required to completely eliminate the VAs.

In this study, approximately 50% of patients with PAM VAs exhibited variable QRS morphologies spontaneously, after the initial ablation lesions, or both. In approximately 80% of these patients, radiofrequency lesions on both sides of the PAMs were required to eliminate all variations in the QRS morphology. These differences in QRS morphologies are compatible with the differences in the direction of the vector of the propagating wavefront from the successful ablation sites on both sides of the PAMs and could be reproduced by pacing from those sites. These findings may suggest 2 possible mechanisms: multiple VA origins or a single VA origin with preferential conduction to multiple exit sites. The latter mechanism may be most likely because a fused form of different QRS morphologies often occurred, and in approximately 20% of those patients, radiofrequency lesions at a single site could eliminate all spontaneous QRS morphologies. Anatomically, the LV PAMs are the thickest myocardial structures in the heart and comprise a complex of myocardial strands with some separations between them on the basal and apical sides, likely resulting in anisotropic conduction (Figure 4). Therefore, activation from a VA origin that is located in the subendocardial or deep region of the PAMs may propagate in different directions, resulting in &gt;1 QRS morphology based on the direction that the activation exits the PAM. In approximately 40% of the study patients with PAM VAs, a low-amplitude ventricular potential preceded the larger near-field ventricular potential at the site of successful ablation. The mechanisms in the PAM VAs addressed previously also could well explain the presence of these prepotentials and the possibility of isolating that prepotential, as was demonstrated in the case study. Although a ventricular prepotential is often recorded at the successful ablation site of VAs arising from the LV ostium, the mechanism of those ventricular prepotentials is likely to differ between PAM and LV ostial VAs. In LV ostial VAs, the first ventricular potential is a near-field electrogram representing activation at the site of VA origin, whereas the second ventricular potential is a far-field electrogram representing activation of the larger myocardial mass around the VA origin. For PAM VAs, on the other hand, the first ventricular potential is more likely a far-field electrogram representing activation of the deep VA origin relative to the surface of the PAMs, whereas the second ventricular potential is a near-field electrogram representing activation of the surface myocardium of the PAM. Although a previous study reported that a Purkinje potential often preceded the QRS onset at the successful ablation site of PAM VAs, this finding was never observed in this study. This discrepancy may be explained by the heterogeneity of the VAs ablated around the PAMs, with some being endocardial (closer to the Purkinje fiber system) and others intramyocardial.

This study may provide several important clinical implications. First, the occurrence of spontaneous variable QRS morphologies during VAs as demonstrated in this study suggests a PAM origin with a focal mechanism. In fact, it may be challenging to differentiate PAM VAs from LV fascicular VAs on the basis of QRS morphology alone. However, previous studies demonstrated that the QRS morphologies are consistent during LV fascicular VAs probably because those VAs are associated with the normal conduction system, with a rapid conduction and mostly a reentrant mechanism. Therefore, those ECG features of PAM VAs may be a useful clue to differentiate them from LV fascicular VAs. Second, the altered QRS morphologies of PAM VAs after the ablation may guide the mapping and catheter ablation. Understanding the relationship between changes in QRS morphology and a shift in the breakout site to the opposite side of the PAM may be helpful for determining the next target of the mapping and ablation.

No complications have been reported in the catheter ablation of VAs arising from the PAMs. However, it should be emphasized that the safety of ablating PAM VAs with an irrigated catheter and with a high power setting is unknown because the number of reported cases is still small.

**Study Limitations**

Although this study provides several findings supporting the concept that VAs originate from a deep site to the surface of the PAMs, there was no definitive proof of the site of origin of these PAM VAs. The ability to map intramural activation may be required to definitively establish the site of origin.

**Conclusions**

Radiofrequency catheter ablation of idiopathic LV PAM VAs is particularly challenging probably because the origins of these VAs may be located relatively deep beneath the endocardium of the PAMs, which have a complex structure. The PAM VAs often exhibit multiple QRS morphologies, with a subtle, but distinguishable difference occurring spontaneously or after the initial ablation lesions that may be caused by a single origin with preferential conduction resulting from the complex structure of the PAMs. These ECG features may be helpful in differentiating PAM VAs from LV fascicular VAs and in guiding mapping and ablation.
Disclosures
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References

CLINICAL PERSPECTIVE
The present study revealed the electrophysiological features of ventricular arrhythmias (VAs) originating from the papillary muscles (PAMs) in the left ventricle and their relevance for the results of catheter ablation. Although an excellent pace map was obtained at the first ablation site in 90% of the patients, radiofrequency ablation at that site failed to eliminate the VAs, and radiofrequency lesions in a relatively wide area around that site were required to completely eliminate the VAs in all patients. Radiofrequency current with an irrigated or nonirrigated 8-mm-tip ablation catheter was required to achieve a lasting ablation of the PAM VA origins. During 40% of the PAM VAs, a sharp ventricular prepotential was recorded at the successful ablation site. In 50% of the patients, PAM VAs often exhibited multiple QRS morphologies, with a subtle, but distinguishable difference occurring spontaneously and after the initial ablation lesions that may be caused by a single origin with preferential conduction resulting from the complex structure of the PAMs. In 80% of those patients, radiofrequency lesions on both sides of the PAMs where pacing could reproduce an excellent match to the 2 different QRS morphologies of the VAs were required to completely eliminate the VAs. Radiofrequency catheter ablation of these VAs is particularly challenging probably because the VA origins may be located relatively deep beneath the endocardium of the PAMs with a complex structure.
Electrocardiographic and Electrophysiological Characteristics in Idiopathic Ventricular Arrhythmias Originating From the Papillary Muscles in the Left Ventricle: Relevance for Catheter Ablation


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