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

Running Title: Yamada et al.; Catheter ablation of idiopathic LV PAM VT

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ABSTRACT

**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 non-irrigated 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 pre-potential 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 origins 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.

**Key words**: idiopathic; ventricular arrhythmia; papillary muscle; electrophysiological characteristics; radiofrequency catheter ablation.
Introduction

Idiopathic ventricular tachycardia (VT) or premature ventricular contractions (PVCs) may arise from several sites in the left ventricle (LV) and radiofrequency catheter ablation is often an effective technique for curing these ventricular arrhythmias (VAs).\textsuperscript{1-13} Recent studies have reported that idiopathic focal VAs can originate from the papillary muscles (PAMs) in the LV and it is consistent that catheter ablation of these VAs is challenging as compared with the other VAs in the LV.\textsuperscript{11-13} The PAMs of the heart are anatomically complex structure which may cause some difficulties in catheter manipulation during mapping and ablation and exhibit specific electrophysiological features. However, the exact mechanism to explain those findings remains unclear. This study was undertaken to reveal the electrophysiological characteristics of these VAs and their relevance for the results of catheter ablation.

Methods

Patient characteristics

The study population was drawn from 159 consecutive patients (97 men, median age 57 years (range 14 to 82)) with symptomatic idiopathic sustained VT (n=63), non-sustained VT (n=26) or PVCs (n=70) originating from the LV. Echocardiography and
exercise stress testing or coronary angiography demonstrated no evidence of structural heart disease in any patient. The sites of origin of VA included the aortic root in 47 (29.6%) patients, aorto-mitral 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, one patient with a PVC origin in the APM that was not successfully ablated was also included. The baseline characteristics including age, gender, nature of the clinical arrhythmia, and 12-lead electrocardiogram during the VAs were recorded. Each patient gave written informed consent, and all antiarrhythmic drugs were discontinued for at least five half-lives prior to the study.

**Electrophysiological study**

For mapping and pacing, standard multielectrode catheters were placed in the coronary sinus, His bundle region, and right ventricular apex via the right femoral vein. A quadripolar ablation catheter was advanced into the LV via 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 extra-stimuli introduced
after an 8-beat drive train with the addition of an isoproterenol infusion if it was necessary or by the intravenous administration of boluses of epinephrine (0.05 mg). During the procedures in the LV, intravenous 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 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 AcuNav\(^\text{TM}\), 64-element, 5.5 to 10.0 MHz, Siemens) (Figure 1). If a CARTO-based three-dimensional ultrasound imaging system (CARTO SOUND\(^\text{TM}\), Biosense Webster Inc) was available, electroanatomic maps were created by adding the activation data during the VT or PVCs onto the three-dimensional LV anatomical shells which were reconstructed with real-time integration of the intracardiac echocardiography as previously reported\(^{16}\). Pace mapping was also performed at a pacing cycle length of 500 ms 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 electrocardiogram as previously reported (perfect pace mapping equal to 24 points).17

Radiofrequency current was delivered at myocardial sites exhibiting the earliest bipolar activity and/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-French uni-directional deflectable 3.5-mm-tip external-irrigated ablation catheter (Navistar ThermoCool™, Biosense Webster, Diamond Bar, CA, USA). The radiofrequency power was titrated to as high as 50 W, with the goal being able to achieve a decrease in the impedance of 8 to 10 ȍ and with care taken to limit the temperature to < 40°C. Non-irrigated radiofrequency current was delivered using a bi-directional 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) 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 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 non-inducibility of VT or PVCs during an isoproterenol infusion (2 to 8 µg/min),
intravenous boluses of epinephrine (0.05 mg) and burst pacing from the right ventricle (to a cycle length as short as 300ms).

Post-procedure 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 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.

Electrocardiographic analysis

Twelve-lead electrocardiograms 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 (M) with the first and third
quartiles (Q1 and Q3), as appropriate. The comparisons of the continuous variables between the 2 groups were analyzed with the use of the 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. There were 13 men and 6 women between the ages of 34 and 82 years (M=57, Q1=46 and Q3=68 years). Echocardiography demonstrated normal LV systolic function (LV ejection fraction; M=0.63, Q1=0.60 and Q3=0.69) and no evidence of structural heart disease in any patient. The clinically presenting arrhythmia was sustained VT in 5 patients, non-sustained VT in 5 patients, and frequent PVCs without any runs of non-sustained 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 prior to the study ranged between 1 month and 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/or 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, while 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 via 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. At the successful ablation sites, a small-amplitude, relatively low frequency potential was observed in the local ventricular electrogram preceding the QRS onset of the VAs in 8 patients (APM; 3 and PPM; 5) (Figure 2). Although an excellent pace map with a score of $\geq 21$ 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, though 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 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 as 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 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 patients (58%) including all 4 patients in which
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 was always 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. During a follow-up period (M=21, Q1=14 and Q3=37 months) after the last procedure, 17 patients have remained free of the VAs and the other 2 patients have exhibited no worsening of the VAs without any antiarrhythmic drugs. In the 17 patients with complete suppression of VAs, an irrigated (n=15) or non-irrigated 8-mm (n=2) 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 patients. Figure 4 demonstrates this typical change in electrocardiographic 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 as compared with those ablated on the posterior side of the APMs (lead II; \( M=0.55, Q_1=0.46 \) and \( Q_3=0.69 \) versus \( M=0.31, Q_1=0.28 \) and \( Q_3=0.33, p=0.012 \), III; \( M=1.26, Q_1=1.17 \) and \( Q_3=1.40 \) versus \( M=0.90, Q_1=0.76 \) and \( Q_3=1.02, p=0.025 \), aVF; \( M=0.87, Q_1=0.74 \) and \( Q_3=0.1.09 \) versus \( M=0.50, Q_1=0.30 \) and \( Q_3=0.73, p=0.012 \)), respectively. In addition, the R-wave amplitude in lead aVR was significantly smaller (\( M=0.22, Q_1=0.09 \) and \( Q_3=0.37 \) versus \( M=0.28, Q_1=0.16 \) and \( Q_3=0.56, p=0.017 \)), the depth of the S-waves in lead aVL was significantly larger (\( M=1.03, Q_1=0.94 \) and \( Q_3=1.09 \) versus \( M=0.91, Q_1=0.83 \) and \( Q_3=0.93, p=0.012 \)), and the QRS duration was significantly longer (\( M=193, Q_1=187 \) and \( Q_3=197 \) versus \( M=187, Q_1=178 \) and \( Q_3=188, p=0.012 \)), 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; \( M=0.87, Q_1=0.76 \) and \( Q_3=1.14 \) versus \( M=1.06, Q_1=0.84 \) and \( Q_3=1.22, p=0.028 \), III; \( M=0.80, Q_1=0.54 \) and...
Q3=0.93 versus M=1.15, Q1=0.88 and Q3=1.35, p=0.041, aVF; M=0.80, Q1=0.63 and Q3=0.93 versus M=1.15, Q1=0.77 and Q3=1.23, p=0.022), the R-wave amplitude in lead aVL was significantly smaller (M=0.43, Q1=0.29 and Q3=0.46 versus M=0.70, Q1=0.48 and Q3=0.77, p=0.001), and the QRS duration was significantly longer (M=179, Q1=177 and Q3=189 versus M=175, Q1=171 and Q3=176, p=0.001) during the VAs ablated on the lateral side as compared with those ablated on the septal side, respectively. In 6 patients (APM; 2 and PPM; 4), these changes in the QRS morphologies occurred spontaneously before the ablation and fusion of these different QRS morphologies was also 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 electrocardiographic 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 ms 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 via 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\(^{18}\) 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 a 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 intravenous 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 are usually 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, higher cost, and potential for creating larger ablation lesions. Hence, making an accurate diagnosis of VAs originating from the PAMs prior to the procedure is important to clinicians. The algorithms based on electrocardiographic 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 to make a decision on whether additional complex mapping measures such as transthoracic and intracardiac echocardiography or a three-dimensional mapping system should be used or not.

Ablation of most idiopathic LV VAs from the endocardial surface by radiofrequency current is usually successful with a 4 mm-tip non-irrigated catheter. Long-term follow-up of patients after ablation of most idiopathic VAs is limited, but the risk of recurrence is generally low. However, in this study 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 non-irrigated 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 though 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, though there was often a change in the QRS morphology. As a result, several further radiofrequency lesions were always required to suppress the PAM VAs. These findings suggested 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 site deep 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 as compared with that in the patients with an excellent pace map.

In this study, about 50% of patients with PAM VAs exhibited variable QRS morphologies spontaneously and/or after the initial ablation lesions. In about 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: either multiple VA origins or a single VA origin with preferential conduction to multiple exit sites. However, the latter mechanism may be most likely because a fused form of different QRS morphologies often occurred and in about 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 are
composed of 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 which is located in the sub-endocardial or deep region of the PAMs may propagate in different directions, resulting in more than one QRS morphology based on the direction that the activation exits the PAM. In this study, in about 40% of the 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 above also could well explain the presence of these pre-potentials and the possibility of isolating that pre-potential as was demonstrated in the case study20. Although a ventricular pre-potential is also often recorded at the successful ablation site of VAs arising from the LV ostium,19 the mechanism of those ventricular pre-potentials 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 while the second ventricular potential is a far-field electrogram representing activation of the larger myocardial mass around the VA origin. On the other hand, for PAM VAs, the first ventricular potential is more likely a far-field electrogram representing activation of the VA origin deep to the surface of the PAMs while 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, some being endocardial (closer to the Purkinje fiber system) and others intra-myocardial.

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 with a reentrant mechanism. Therefore, those electrocardiographic 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 following 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 as the number of reported cases is still small.

Study limitations

Although this study provides several findings supporting the concept that VAs originate from a site deep 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. This may be caused by a single origin with preferential conduction resulting from the complex structure of the PAMs. These electrocardiographic features may be helpful for differentiating PAM VAs
from LV fascicular VAs and guiding mapping and ablation.

Conflict of Interest Disclosures

Dr. Yamada is supported by a research grant from Boston Scientific and St. Jude Medical. Drs. Epstein, Kay, Plumb, and McElderry have participated in catheter research funded by Biosense-Webster and Irvine Biomedical. Dr. Kay has received honoraria from Medtronic, Boston Scientific, and St. Jude Medical. Dr. Epstein has received honoraria from and served on events committees for Boston Scientific and St. Jude Medical. Dr. McElderry has received consulting fees from Boston Scientific, St. Jude Medical, and Biosense-Webster. The electrophysiology fellowship program at the University of Alabama at Birmingham receives funding support from Boston Scientific and Medtronic. The other authors report no conflicts.
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### Table 1. Clinical Characteristics of Patients with Ventricular Arrhythmias

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Origin</th>
<th>Age (yrs)</th>
<th>Gender</th>
<th>Type</th>
<th>Duration of symptoms (yrs)</th>
<th>LVEF (%)</th>
<th>Ex-triggered</th>
</tr>
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<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>
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<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>
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<tr>
<td>4.</td>
<td>APM</td>
<td>59</td>
<td>M</td>
<td>PVC</td>
<td>1.0</td>
<td>77</td>
<td>–</td>
</tr>
<tr>
<td>5.</td>
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<td>M</td>
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<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>
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<td>+</td>
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<td>+</td>
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<td>10.</td>
<td>PPM</td>
<td>67</td>
<td>M</td>
<td>NSVT</td>
<td>1.0</td>
<td>58</td>
<td>–</td>
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<td>11.</td>
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<td>68</td>
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<td>PVC</td>
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<td>82</td>
<td>M</td>
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<td>1.0</td>
<td>57</td>
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<tr>
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<td>PVC</td>
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<td>60</td>
<td>–</td>
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<tr>
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<td>+</td>
</tr>
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<td>M</td>
<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>
<td>66</td>
<td>M</td>
<td>NSVT</td>
<td>2.0</td>
<td>59</td>
<td>–</td>
</tr>
<tr>
<td>18.</td>
<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>

APM=anterior papillary muscle; Ex=exercise; F=female; LVEF=left ventricular ejection fraction; M=male; NSVT=non-sustained ventricular tachycardia (VT); PPM=posterior papillary muscle; Pt.=patient; PVC=premature ventricular contraction; SVT=sustained VT; yrs=years.
Table 2. Electrophysiological Characteristics of Patients with Ventricular Arrhythmias

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>ABL site</th>
<th>V-QRS (ms)</th>
<th>Pre-P</th>
<th>PM score</th>
<th>ABL catheter</th>
<th>RF lesions (No.)</th>
<th>Recurrence</th>
<th>Change of QRS morph.</th>
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<tbody>
<tr>
<td>1.</td>
<td>Post.</td>
<td>-26</td>
<td>+</td>
<td>24</td>
<td>Ext.-I</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Both</td>
<td>-35</td>
<td></td>
<td>14</td>
<td>8mm-N, Ext.-I</td>
<td>16+6</td>
<td></td>
<td>Recurrent</td>
</tr>
<tr>
<td>3.</td>
<td>Post.</td>
<td>-18</td>
<td>+</td>
<td>24</td>
<td>4mm-N</td>
<td>11</td>
<td>+</td>
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</tr>
<tr>
<td>4.</td>
<td>Ant.</td>
<td>-30</td>
<td></td>
<td>21</td>
<td>4mm-N</td>
<td>5</td>
<td>+</td>
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</tr>
<tr>
<td>5.</td>
<td>Both</td>
<td>-32</td>
<td></td>
<td>24</td>
<td>Ext.-I</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Both</td>
<td>-20</td>
<td>+</td>
<td>24</td>
<td>4mm-N, 8mm-N</td>
<td>2+12</td>
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<td>Recurrent</td>
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<tr>
<td>7.</td>
<td>Ant.</td>
<td>-22</td>
<td></td>
<td>22</td>
<td>Ext.-I</td>
<td>5+2</td>
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<tr>
<td>9.</td>
<td>Both</td>
<td>-23</td>
<td></td>
<td>22</td>
<td>Ext.-I</td>
<td>8+6</td>
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<td></td>
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<tr>
<td>10.</td>
<td>Sept.</td>
<td>-25</td>
<td></td>
<td>23</td>
<td>Int.-I, Ext.-I</td>
<td>4+4</td>
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<td>Recurrent</td>
</tr>
<tr>
<td>11.</td>
<td>Lat.</td>
<td>-31</td>
<td></td>
<td>23</td>
<td>Ext.-I</td>
<td>16+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>Lat.</td>
<td>-24</td>
<td>+</td>
<td>22</td>
<td>Int.-I, Ext.-I</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>Both</td>
<td>-33</td>
<td>+</td>
<td>21</td>
<td>Ext.-I</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>Both</td>
<td>-28</td>
<td>+</td>
<td>24</td>
<td>Ext.-I</td>
<td>8</td>
<td></td>
<td>After ABL</td>
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<tr>
<td>16.</td>
<td>Sept.</td>
<td>-20</td>
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<td>22</td>
<td>4mm-N, 8mm-N</td>
<td>16+5</td>
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<td></td>
</tr>
<tr>
<td>17.</td>
<td>Sept.</td>
<td>-29</td>
<td></td>
<td>24</td>
<td>Int.-I, 8mm-N</td>
<td>10</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ABL=ablation; Ant.=anterior; Ext.=external; I=irrigation; Int.=Internal; Lat.=lateral; morph.=morphology; N=non-irrigation; PM=pace map; Post.=posterior; Pre-P=pre-potential; RF=radiofrequency; Sept.=septal; Spont.=spontaneous. X and Y in RF lesions indicate the number of RF lesions given in the first and second procedures, respectively.
Figure Legends:

**Figure 1:** The intracardiac echocardiographic images of the successful ablation site.

Two-dimensional intracardiac echocardiographic short-axis images at the level of the papillary muscles demonstrated that the ablation catheter (ABL) was positioned on the anterior and posterior sides of the anterior papillary muscle (APM). The arrowheads indicate the acoustic shadow from the ablation catheter.

LV=left ventricle; PPM=posterior papillary muscle.

**Figure 2:** Cardiac tracings (left panel) and fluoroscopic images (right panels) exhibiting the successful ablation site of the premature ventricular contractions (PVCs) originating from the LV PPM.

The first beat is a sinus beat and the second one is a PVC. Note that at the successful ablation site, no Purkinje potentials were observed during sinus rhythm and a spiky pre-potential (arrowhead) was observed in the local ventricular activity during the PVC.

ABL (HB, RV) d(p)=the distal (proximal) electrode pair of the ablation (His bundle, right ventricular) catheter; CS 1 to 5=the first to fifth electrode pair of the coronary sinus catheter; LAO=the left anterior oblique view; RAO=the right anterior oblique view; V-QRS=the local ventricular activation time relative to the QRS onset.

**Figure 3:** The 12-lead electrocardiograms of the PVCs presumably originating from the LV APM and pace map (PM) obtained by pacing from the epicardial (Epi) surface opposite the APM (left panels) and fluoroscopic images (right panels) 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.
The other abbreviations are as in Figure 2.

Figure 4: The 12-lead electrocardiograms of the PVCs originating from the LV PPM and excellent pace maps (PMs) and an electroanatomic map exhibiting the radiofrequency ablation sites.

At baseline, there were 2 main PVCs with differences in the QRS morphology (PVC1 and PVC2) and a fused form of them. The depth of the S waves in the inferior leads was smaller, the R wave amplitude in lead aVL was smaller, and the QRS duration was longer during PVC 1 than during PVC 2. Excellent pace maps of PVC 1 and PVC 2 were reproduced by pacing from the lateral and septal sides of the PPM, respectively, before the ablation. Radiofrequency ablation at the lateral side of the PPM never interrupted the PVCs. Because the QRS morphology of PVC 1 slightly changed and the earliest activation site gradually moved to the septal side of the PPM after every radiofrequency application, radiofrequency lesions targeting the earliest ventricular activation sites were created on approximately half the circumference of the PPM. Finally, radiofrequency ablation at the septal side completely eliminated all the PVCs.

PM 1 and PM 2 correspond to the pace maps for PVC 1 and PVC 2, respectively. The arrow on the electroanatomic map indicates the direction of the creation of the radiofrequency lesions.

INF=inferior.

Figure 5: The human autopsy heart exhibiting the distribution of the successful ablation sites (left panel) and representative 12-lead electrocardiograms exhibiting the differences in the QRS morphology of the ventricular arrhythmias successfully ablated between both sides of the papillary muscles in the same patients (right panels).
The number in the left panel indicates the number of the patients and the number in the parentheses the number of the patients with radiofrequency lesions on both sides of the papillary muscle.

Ant.=anterior side; Lat.=lateral side; Post.=posterior side; Sept.=septal side. The other abbreviations are as in Figure 1.

**Figure 6:** The 12-lead electrocardiograms exhibiting a spontaneous change in the QRS morphology during the ventricular tachycardia originating from the LV PPM.
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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