Post-Infarction Ventricular Arrhythmias Originating in Papillary Muscles

Frank Bogun, MD, Benoit Desjardins, MD, Thomas Crawford, MD, Eric Good, DO, Krit Jongnarangsin, MD, Hakan Oral, MD, Aman Chugh, MD, Frank Pelosi, MD, Fred Morady, MD
Ann Arbor, Michigan

Objectives
The aim of this study was to define the role of papillary muscles (PAPs) in post-infarction ventricular arrhythmias.

Background
Papillary muscles have been implicated in arrhythmogenesis; however, their role in post-infarction ventricular arrhythmias has not been well-defined.

Methods
In a series of 9 patients (age 65 ± 9 years, ejection fraction 0.36 ± 0.1) with post-infarction ventricular arrhythmias, electroanatomic mapping in conjunction with intracardiac echocardiography demonstrated that 1 of the PAPs was involved in the arrhythmia. Magnetic resonance imaging with delayed enhancement (DEMRI) was performed in all patients without contraindications. A consecutive series of 9 patients (age 64 ± 8 years, ejection fraction 0.32 ± 0.14) with ventricular arrhythmias that did not originate from the PAP served as a control group and also underwent DEMRI.

Results
Heterogeneous uptake of gadolinium during magnetic resonance imaging was observed more frequently in arrhythmogenic PAPs than in PAPs that were not involved in ventricular arrhythmias (p = 0.01). The PAPs in the control patients did not take up contrast or show homogeneous contrast uptake. Radiofrequency ablation eliminated all arrhythmias originating from PAPs. Echocardiography after the ablation showed no new or worsened mitral regurgitation.

Conclusions
Papillary muscles that lie within an infarct zone might give rise to ventricular arrhythmias. Heterogeneous uptake of gadolinium in magnetic resonance images might be predictive of arrhythmogenic PAPs. Radiofrequency catheter ablation of ventricular tachycardia and ventricular ectopy arising in a PAP has a high success rate.

From the University of Michigan Medical Center, Ann Arbor, Michigan. Drs. Oral and Morady served as consultants to Biosense Webster.
Manuscript received August 2, 2007; revised manuscript received January 23, 2008, accepted January 27, 2008.

The papillary muscles (PAPs) have been implicated in arrhythmogenesis (1–3). However, few reports have described their role in generating ventricular arrhythmias in humans. The PAPs that lie within a zone of infarction have electrophysiological properties similar to other infarcted myocardium (4). The purpose of this study was to assess the role of the left ventricular PAPs in generating ventricular arrhythmias in a consecutive series of post-infarction patients undergoing left ventricular mapping and ablation for symptomatic ventricular ectopy or ventricular tachycardia (VT).

Methods
Patient characteristics. The subjects of this study were 9 post-infarction patients in whom the arrhythmia originated either from the anterolateral or the posteromedial left ventricular PAP (Table 1). All patients without contraindications for magnetic resonance imaging (MRI) underwent MRI. The patients had been referred for mapping and radiofrequency ablation of symptomatic premature ventricular complexes (PVCs) (n = 5) or VT (n = 4). Two of 9 patients had an implantable cardioverter-defibrillator (ICD); they were referred for frequent ICD discharges. One patient was being treated with sotalol, and the other patients were being treated with a beta-blocker but no other antiarrhythmic medications.

The patients were selected from a consecutive series of 58 patients with post-infarction ventricular arrhythmias. Nine consecutive patients (of the 58 patients) without a contraindication for a cardiac MRI also underwent cardiac MRI and served as a control group. None of these 9 patients had VT or PVCs originating in a PAP.

The 12-lead electrocardiogram recordings of PAP and non-PAP arrhythmias that were targeted were compared using previously described criteria (5,6), including bundle
branch block, axis, morphology in V1, as well as R-wave transition from V1 to V6, and notches in the precordial leads. In the presence of a predominant R-wave in V1, the precordial transition of the R- to S-wave was assessed. We compared early (V1 to V2) versus late (V4 to V6) R- to S-wave transition. Notches were defined as deflections in the QRS complex in addition to a triphasic pattern.

**Electrophysiological study.** After informed consent was obtained, a 6-F electrode catheter was introduced into the right femoral vein and positioned in the right ventricle. Programmed right ventricular stimulation was performed at 2 right ventricular sites with 1 to 4 extrastimuli. Systemic heparinization was carried out throughout the procedure with a target activated clotting time of 300 s.

An electroanatomical mapping system (CARTO, Biosense, Diamond Bar, California) with an 8-F catheter (Navistar 4-mm-tip electrode or Thermocool 3.5-mm-tip electrode, separated by 1 mm from a 2-mm ring electrode) was used in all patients. The Navistar catheter was used in 5 patients (4 with PAP-related arrhythmias), and the Thermocool catheter was used in the remaining 13 patients. Electrograms were filtered at 50 to 500 Hz. The intracardiac electrograms and leads V1, I, II, and III were displayed on an oscilloscope and recorded at a speed of 100 mm/s. Access to the left ventricle was obtained via a retrograde aortic approach.

Electroanatomical voltage mapping was performed during sinus rhythm (Fig. 1), and sites with a voltage <1.0 mV were considered to be scar; a voltage of 1.0 to 1.5 mV was defined as border zone. Bipolar pacing was performed at all sites with electrograms <1.5 mV, to identify critical VT sites (7).

**Table 1** Characteristics of Patients With and Without Arrhythmias Involving PAPs

<table>
<thead>
<tr>
<th>Patient/Age (yrs)/Gender</th>
<th>MI Location</th>
<th>EF</th>
<th>Arrhythmia</th>
<th>No. of Induced VTs/ICD</th>
<th>Arrhythmogenic PAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>#1/54/M</td>
<td>Inferior</td>
<td>40</td>
<td>PVCs</td>
<td>0/no</td>
<td>Posteromedial</td>
</tr>
<tr>
<td>#2/48/M</td>
<td>Inferior</td>
<td>20</td>
<td>PVCs</td>
<td>0/yes</td>
<td>Posteromedial</td>
</tr>
<tr>
<td>#3/72/M</td>
<td>Inferior</td>
<td>45</td>
<td>VT</td>
<td>6/yes</td>
<td>Posteromedial</td>
</tr>
<tr>
<td>#4/63/M</td>
<td>Inferior</td>
<td>50</td>
<td>VT, PVCs</td>
<td>1/yes</td>
<td>Posteromedial</td>
</tr>
<tr>
<td>#5/70/M</td>
<td>Inferior</td>
<td>35</td>
<td>PVCs</td>
<td>0/no</td>
<td>Posteromedial</td>
</tr>
<tr>
<td>#6/64/M</td>
<td>Inferior</td>
<td>30</td>
<td>PVCs</td>
<td>0/yes</td>
<td>Posteromedial</td>
</tr>
<tr>
<td>#7/76/F</td>
<td>Lateral MI</td>
<td>25</td>
<td>VT, PVCs</td>
<td>1/yes</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>#8/70/M</td>
<td>Inferior</td>
<td>30</td>
<td>VT</td>
<td>8/yes</td>
<td>Posteromedial</td>
</tr>
<tr>
<td>#9/69/M</td>
<td>Anterior</td>
<td>47</td>
<td>PVCs</td>
<td>0/no</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>#10/74/F</td>
<td>Anterior</td>
<td>30</td>
<td>VT</td>
<td>3/yes</td>
<td>None</td>
</tr>
<tr>
<td>#11/65/F</td>
<td>Anterior</td>
<td>25</td>
<td>VT</td>
<td>2/yes</td>
<td>None</td>
</tr>
<tr>
<td>#12/59/M</td>
<td>Inferior</td>
<td>15</td>
<td>PVCs, VT</td>
<td>6/yes</td>
<td>None</td>
</tr>
<tr>
<td>#13/62/M</td>
<td>Inferior</td>
<td>15</td>
<td>PVCs, VT</td>
<td>4/yes</td>
<td>None</td>
</tr>
<tr>
<td>#14/51/M</td>
<td>Inferior</td>
<td>40</td>
<td>VT</td>
<td>1/yes</td>
<td>None</td>
</tr>
<tr>
<td>#15/56/M</td>
<td>Inferior</td>
<td>50</td>
<td>VT</td>
<td>1/no</td>
<td>None</td>
</tr>
<tr>
<td>#16/68/M</td>
<td>Anterior</td>
<td>45</td>
<td>PVCs</td>
<td>0/no</td>
<td>None</td>
</tr>
<tr>
<td>#17/74/M</td>
<td>Anterior</td>
<td>19</td>
<td>PVCs</td>
<td>0/0</td>
<td>None</td>
</tr>
<tr>
<td>#18/64/M</td>
<td>Inferior</td>
<td>50</td>
<td>PVCs</td>
<td>0/0</td>
<td>None</td>
</tr>
</tbody>
</table>

**Abbreviations and Acronyms**

DEMRI = magnetic resonance imaging with delayed enhancement

ICD = implantable cardioverter-defibrillator implanted

MRI = magnetic resonance imaging

PAP = papillary muscle

PVC = premature ventricular complex

VT = ventricular tachycardia

Activation mapping was performed during ventricular ectopy or VT. Purkinje potentials were sought during sinus rhythm and during ventricular ectopy/VT at the site of earliest ventricular activation or at the VT exit site. Bundle branch re-entry and fascicular VT were ruled out with established criteria (8–10).

With the clipping plane perpendicular to the long-axis orientation of the heart, the distance between the critical site of a VT or the earliest activation site during a PVC and the closest point on the endocardial shell of the electroanatomic map was determined (Fig. 2). If the site of a critical component of an arrhythmia was not on the surface of the endocardial shell of the electroanatomic map, intracardiac echocardiography (Cypres, Acuson, Mountain View, California) was used to confirm contact of the ablation catheter with the PAP (Fig. 3).

The VT isthmus sites were identified by 1 or more of the following criteria: for hemodynamically tolerated VTs: 1) concealed entrainment with termination of VT during radiofrequency energy delivery; and 2) VT termination by mechanical pressure with the mapping catheter; and for VTs that were not hemodynamically tolerated, an isthmus was defined as a site where there was a match in QRS morphology between the pace-map and the VT. Although no direct proof could be provided that this type of site was indeed a critical isthmus, a prior study demonstrated that these findings are present at effective ablation sites in a...
critical isthmus. The site of origin of ventricular ectopy was defined as the site of earliest endocardial activation where radiofrequency ablation eliminated the ectopy (Fig. 4).

If ventricular ectopy was infrequent or if VT was hemodynamically unstable, pace-mapping was used to identify the exit site of the VT or the site of origin of the PVC (7).

Radiofrequency ablation. Applications of radiofrequency energy were titrated to achieve a 10-Ohm impedance drop (power range 30 to 50 W) and were delivered during ventricular ectopy or VT. The applications were continued for at least 30 s if there was an appropriate impedance drop. If the targeted ventricular arrhythmia stopped within 30 s, the energy application was continued for 60 s and was followed by a second 60-s application. If the ectopy or VT did not terminate within 30 s, the radiofrequency energy application was terminated and another target site was sought. After ablation, programmed right ventricular stimulation was repeated. Successful catheter ablation was defined as termination of ventricular ectopy or VT by an application of radiofrequency energy and the inability to induce VT by programmed stimulation.

The study protocol was approved by the Institutional Review Board of the University of Michigan Health System.

MRI. Before the ablation procedure, 6 of the study patients underwent cardiac magnetic resonance imaging with delayed enhancement (DEMRI). The MRI was not feasible in 2 patients who had an implantable defibrillator. One patient had a recent coronary stenting procedure, and an MRI was not performed. Magnetic resonance images from the study patients were compared with those of a control group of 9 consecutive post-infarction patients with VT or PVCs not arising from a PAP who also underwent DEMRI.

The MRI studies were performed on a 1.5-T MRI scanner (Signa Excite CV/i, General Electric, Milwaukee, Wisconsin) with a 4- or 8-element phased array coil placed over the chest of patients in the supine position. Images were acquired with electrocardiography gating during breath-holds. Dynamic short- and long-axis images of the heart were acquired with a segmented, k-space, steady-state, free-precession pulse sequence (repetition time 4.2 ms, echocardiography time 1.8 ms, 1.4 mm in-plane spatial resolution, 8 mm slice thickness). Fifteen minutes after administration of 0.20 mmol/kg of intravenous gadolinium diethylenetriaminepentaacetic acid (Magnevist, Berlex Pharmaceuticals, Wayne, New Jersey), 2-dimensional delayed enhancement imaging was performed with an inversion-recovery sequence (repetition time 6.7 ms, echocardiography time 3.2 ms, in-plane spatial resolution 1.4 × 2.2 mm, slice thickness 8 mm) in the short-axis and long-axis of the left ventricle at matching cine-image slice locations. The inversion time (250 to 350 ms) was optimized to null the normal myocardium. On the DEMRI, the major heads of the anterolateral and posteromedial PAPs were traced with the Osirix (version 2.7.5) Dicom Viewer software (Geneva, Switzerland) and their volumes were computed. The images were examined for heterogeneous or homogeneous uptake of gadolinium (Figs. 5A and 5B).
Heterogeneous contrast uptake was present if any cut of the PAPs and/or longitudinal axis displayed tissue with heterogeneous signal (Figs. 5 and 6). The number of heterogeneous short-axis images was determined. The DEMRIs were reviewed by 2 observers blinded to the results of the ablation procedure. Discrepancies were resolved by consensus.

On each image with an area of heterogeneous signal within the PAPs, an estimated region of interest was traced.
within the area of high signal, and the maximum intensity 
M in this region was computed. The full area of delayed 
enhancement was then automatically determined by a re-
gion growing algorithm as the area encompassing pixels 
with values ≥M/2, with the traditional method of Full 
Width Half Maximum (12). A heterogeneity index was 
determined for each PAP (Figs. 5A and 5B) on short-axis 
views; this index was equal to \( \frac{1}{\text{ABS}(2R - 1)} \), where R 
is the ratio of the volume of tissue with delayed enhance-
ment to the total volume of the PAP. If there was 
homogeneity in signal (either normal or abnormal), the 
heterogeneity index was 0 (Figs. 5A and 5B). If the PAP 
had equal proportions of normal and abnormal signal, the 
heterogeneity index was maximal and equal to 1. The 
volume of normal (no contrast uptake) and abnormal (with 
contrast uptake) PAP tissue was also traced and measured. 

**Follow-up.** An echocardiogram was performed after the 
ablation procedure to document whether there was mitral 
regurgitation. Patients were seen 3 months after the ablation 
procedure, at which point a 24-h Holter monitor and 
echocardiogram were obtained. The mean duration of 
follow-up was 22 ± 9 months.

**Statistical analysis.** Variables are expressed as mean ± 1 
SD. Continuous variables were compared with the Student
Discrete variables were compared with the Fisher exact test or by chi-square analysis, as appropriate. If a cell size was <5, the Fisher exact test was used. A p value <0.05 was considered statistically significant.

**Results**

**Electrocardiographic findings.** In the study group, a total of 16 VTs with a mean cycle length of 274 ± 53 ms (all right bundle branch block morphology) were induced by programmed stimulation. In the control group, 17 VTs (7 left bundle branch block, 10 right bundle branch block; cycle length: 337 ± 72 ms) were induced. These VTs were induced in 6 of 9 patients in the control group; the remaining 3 patients had PVCs without inducible VT. The PAP arrhythmias all had a right bundle branch block morphology (7 with superior, 2 with inferior axis), and among the 32 non-PAP arrhythmias, 4 had a left bundle branch block with inferior axis, 4 had a left bundle branch block with superior axis, 7 had a right bundle branch block with inferior axis, and 17 had a right bundle branch block with superior axis. The QTc (p = 0.1 for comparison of left vs. right bundle, p = 0.2 for multiple comparisons including axis). There was no difference in QRS duration comparing arrhythmias with PAP involvement with arrhythmias without PAP involvement (175 ± 29 ms vs. 167 ± 23 ms; p = 0.4). The VT cycle length of PAP arrhythmias did not differ from the VT cycle length of VTs not involving the PAP (294 ± 89 ms vs. 308 ± 69 ms; p = 0.7). All arrhythmias originating from the posteromedial PAP had a superior axis, and the arrhythmias originating from the anterolateral PAP had an inferior axis. With respect to the precordial QRS complexes, when comparing early R- to S-wave transition (V₁ to V₃) with late R- to S-wave transition (V₄ to V₆), the presence of a right bundle branch block with late transition was associated with a PAP origin as compared with arrhythmias originating from elsewhere (7 of 9 PAP arrhythmias [78%] had this morphology compared with 10 of 32 arrhythmias [31%] without documented PAP involvement; p = 0.02). With respect to notching, although all 9 PAP-related arrhythmias displayed notching in the precordial leads, this was also frequent in arrhythmias originating from sites other than the PAPs (24 of 32 non–PAP-related arrhythmias; p = 0.16).

**Electrophysiological findings.** Among the 9 patients with arrhythmias involving the PAP, 5 had PVCs only. The site of origin of the PVCs was identified in all 5 of these patients by activation mapping. The site of origin preceded the QRS complex by 58 ± 19 ms and was confined to the posteromedial PAP in 4 and to the anterolateral PAP in 1 patient. The stimulus-QRS interval was 51 ± 12 ms, which matched the electrogram-QRS interval. There was a match of the paced QRS morphology in all 12/12 leads with the targeted PVC when pacing was performed from the site of origin of the PVC.

Four sustained VTs involved the PAP. All of the VTs were inducible by programmed stimulation and could be terminated with overdrive pacing. Entrainment mapping was not feasible, because the VTs were not hemodynamically tolerated. A critical isthmus site was found on the posteromedial PAP for 3 of the VTs (Fig. 7A) and on the anterolateral PAP for the remaining VT (Fig. 7B). The QRS morphology of the pace-maps at the exit site matched with the induced VTs in 12/12 leads.

**Target sites and results of ablation.** The critical area involved the posteromedial PAP in 7 of 9 patients and the anterior PAP in 2 of 9 patients.
Two of the 4 patients with VT involving the PAPs had 14 other inducible VTs that were not confined to PAPs. A critical isthmus was identified for 8 of 14 VTs, and these sites were confined to an inferior wall scar. In the other 2 patients with inducible VT, all of the VTs involved the PAPs.

In 1 of 5 patients with frequent PVCs, 4 different PVC morphologies were present and 1 of them originated from the posteromedial PAP, with the remaining PVCs originating from an inferoseptal scar. In the remaining 4 patients with PVCs, all arrhythmias were confined to the PAPs. The site of origins of the PAP PVCs were located within scar tissue (n = 4) or the border zone (n = 1) and had an amplitude of 0.4 ± 0.4 mV; they were located on the body of the PAP. The PAP VT isthmus sites were all located within scar tissue and had a mean amplitude of 0.2 ± 0.07 mV during sinus rhythm (p = 0.2). The PAP-related isthmus sites were at a greater distance from the endocardial shell compared with other VT isthmus sites (10 ± 6 mm vs. 0.6 ± 2 mm; p < 0.0001). During sinus rhythm there were no Purkinje potentials at sites critical for arrhythmias originating from the PAPs.

Radiofrequency ablation at a PAP was effective in eliminating the target arrhythmia in all 9 patients. In the 5 patients in whom PVCs were targeted, the PVC burden was reduced from a baseline of 27.8 ± 10% to 0.4 ± 0.7% (p = 0.01) at 3 months after ablation.

A mean of 26 ± 13 applications of radiofrequency energy were applied in patients with arrhythmias originating from the PAPs, compared with 24 ± 19 applications for non-PAP arrhythmias in the other patients (p = 0.8). This includes all lesions delivered in patients with PAP arrhythmias compared with all lesions delivered in patients without

Figure 7 12-Lead Electrocardiograms of PAP-VTs
(A) A 12-lead electrocardiogram of a ventricular tachycardia (VT) (cycle length 275 ms) involving a posteromedial papillary muscle (PAP). There was a right bundle branch block morphology with superior axis and Q waves in the inferior leads. (B) A 12-lead electrocardiogram of a VT (CL 420 ms) involving an anterolateral PAP. There was a right bundle branch block morphology with inferior axis.
PAP arrhythmias. Targeting PAP arrhythmias, a total of 17 ± 8 lesions were delivered (of these, 7 ± 3 radiofrequency lesions were delivered on the PAPs; the remaining lesions projected on the endocardial shell). A mean of 10 ± 6 applications of radiofrequency energy were required to ablate arrhythmias not originating from the PAPs (p = 0.04 comparing number of lesions delivered/arrhythmia: PAP arrhythmias vs. arrhythmias not involving the PAPs). The procedure time tended to be longer in patients with PAP arrhythmias than in patients without arrhythmias involving the PAPs (331 ± 77 min vs. 262 ± 62 min; p = 0.06). The fluoroscopy time was similar in the 2 patient groups (48 ± 18 min vs. 55 ± 14 min; p = 0.5).

 Imaging of PAPs. The presence of heterogeneous contrast uptake in a PAP was associated with the PAP being arrhythmogenic (Fisher exact test; p = 0.0001) (Table 2). Heterogeneous contrast uptake was observed in 9 of 30 PAP heads that were available for analysis. Six of 9 PAPs with a heterogeneous contrast pattern were found to be involved in ventricular arrhythmias. All 6 arrhythmogenic PAPs that were imaged by MRI showed heterogeneous contrast uptake. None of the PAPs without contrast uptake or with homogeneous uptake was involved in ventricular arrhythmias.

 Follow-up. Echocardiography after ablation did not show new or worsened mitral insufficiency in any patient.

 Three of the 9 patients with arrhythmias originating from a PAP had recurrent arrhythmias. None of these patients had inducible VT after the ablation procedure. Two patients had recurrent VT terminated by the ICD, and on the basis of comparison of the far-field ICD stored electrograms, this was a new VT that had not been induced during the first ablation procedure. The other patient had recurrent PVCs with a morphology different than the morphology of the ablated arrhythmias. All 6 arrhythmogenic PAPs that were imaged by MRI showed heterogeneous contrast uptake. None of the PAPs without contrast uptake or with homogeneous uptake was involved in ventricular arrhythmias.

 Discussion

 Main findings. This study demonstrates that VT and PVCs might originate from PAPs within zones of prior infarction. Visual confirmation of PAP involvement was provided by intracardiac echocardiography. Heterogeneous uptake of contrast by PAPs on MRIs was associated with involvement of the PAP in the generation of a ventricular arrhythmia.

 DEMRI and post-infarction arrhythmias involving a PAP. In prior studies, heterogeneous contrast uptake in areas with delayed enhancement was associated with an increased risk of mortality in post-infarction patients (13) or an increased susceptibility for inducible arrhythmias in post-infarction patients (14). Yan et al. (13) hypothesized that surviving myocytes in the peri-infarct zone might account for the MRI findings as well as for life-threatening ventricular arrhythmias. In the present study of post-infarction patients, a heterogeneous pattern of contrast uptake by a PAP on MRI was associated with arrhythmogenicity of the PAP. This finding is in concert with the findings of Yan et al (13). It seems likely that heterogeneous contrast uptake in a PAP that lies within an infarct zone reflects the presence of surviving muscle bundles within a region of scar. The surviving muscle bundles in an infarct zone would explain the arrhythmogenicity of a PAP. However, MRIs might not be feasible in all patients, limiting the ability to identify tissue heterogeneity in these patients.

 Experiments using PAPs in Langendorf-perfused human hearts demonstrated that zig-zag activation of surviving muscle bundles embedded within scarred PAPs might occur and give rise to circuitous conduction pathways that constitute part of a VT re-entry circuit (15). However, ventricular arrhythmias arising in a PAP in patients with a history of myocardial infarction have not been described previously. This might be because imaging techniques such as intracardiac echocardiography have not been routinely employed to determine whether effective ablation sites in post-infarction patients with VT or PVCs involved a PAP. The mechanism of VT involving a PAP seems to be re-entry, because the VTs were inducible by programmed ventricular stimulation. Higher resolution mapping is required to determine whether re-entry is also the mechanism of the PVCs mapped to the PAPs.

 The ablation procedure was longer and required more ablation lesions in patients with PAP involvement than in patients without PAP involvement. Additional imaging and difficulties with obtaining good contact with the PAP might account for this.

 Study limitations. In this study, intracardiac echocardiography was not performed in all patients but only when PAP involvement was suspected on the basis of the electroanatomic map during sinus rhythm. Therefore, the prevalence of ventricular arrhythmia involving a PAP might have been underestimated. Furthermore, the number of patients in this study was small, and the actual prevalence of VT or PVCs involving a PAP will need to be clarified in larger studies.

 We did not systematically assess the electrogram morphology when the catheter was in contact with the PAP and

| Table 2 | Comparison of Magnetic Resonance Imaging of Arrhythmogenic and Nonarrhythmogenic PAPs |
| --- | --- | --- | --- |
| Arhythmogenic | Nonarhythmogenic | p Value |
| Number | 6 | 24 | — |
| PAP study patients/ PAP control patients | 6/0 | 6/18 | — |
| Total volume (cm³) | 5 ± 3.8 | 5.4 ± 3.0 | 0.8 |
| Normal volume (cm³) | 3.0 ± 1.9 | 5.2 ± 3.3 | 0.2 |
| Abnormal volume (cm³) | 2.1 ± 1.8 | 0.2 ± 0.8 | 0.001 |
| % abnormal | 50 % 26 | 13 ± 33 | 0.02 |
| Heterogeneity index | 0.53 ± 0.36 | 0.01 ± 0.04 | <0.0001 |
| No. of segments with heterogeneous uptake | 2.5 ± 1.4 | 0.1 ± 0.3 | <0.0001 |
| Heterogeneous uptake | 6 (100%) | 3 (13%) | 0.0001 |

PAP = papillary muscle.
can therefore not comment on the location of the Purkinje-muscular interface. The lack of Purkinje potentials at effective ablation sites might be secondary to the presence of scar tissue.

During follow-up, recurrent VTs were judged to be the same or different than previously ablated VTs by comparing stored ICD far-field electrograms with the far-field electrograms recorded during the ablation procedure. However, the accuracy of far-field ICD electrograms to assess outcome of VT ablation needs further evaluation.

**Clinical implications.** Heterogeneous contrast uptake by a PAP in a DEMRI indicates that a post-infarction arrhythmia might originate from a PAP. An arrhythmia with right bundle branch block superior axis morphology with late R- to S-wave transition suggests involvement of the posteromedial PAP, and a right bundle branch block inferior axis morphology with late R- to S-wave transition suggests involvement of the anterolateral PAP. Radiofrequency catheter ablation of ventricular arrhythmias involving a PAP is safe and does not seem to cause clinically significant PAP dysfunction.

Reprint requests and correspondence: Dr. Frank Bogun, Assistant Professor of Medicine, Division of Cardiology, CVC Cardiovascular Medicine, 1500 East Medical Center Drive SPC 5853, Ann Arbor, Michigan 48109-5853. E-mail: fbogun@med.umich.edu.

**REFERENCES**


