Epicardial Catheter Ablation of Ventricular Tachycardia in No Entry Left Ventricle
Mechanical Aortic and Mitral Valves

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Background—In patients with mechanical aortic and mitral valves and left ventricular tachycardia, catheter ablation may be prevented by limited access to the left ventricle.

Methods and Results—In our series of 6 patients, 2 patients underwent direct surgical ablation and 4 underwent epicardial catheter ablation via a pericardial window. All patients had abnormal low voltage areas with fractionated potentials on the epicardium. Most of the ventricular tachycardias were targeted by pace mapping. Sites with a good pace match or abnormal electrograms were ablated using an irrigated radiofrequency ablation catheter. A microscopic pathological evaluation of the resected tissue from 2 of the open-heart ablation patients revealed dense fibrosis on the epicardium compared with the endocardium, supporting the feasibility of an epicardial ablation for the ventricular tachycardia.

Conclusions—Epicardial catheter ablation of ventricular tachycardia is a potentially useful therapy in patients who have mechanical aortic and mitral valves. (Circ Arrhythm Electrophysiol. 2015;8:381-389. DOI: 10.1161/CIRCEP.114.002517.)

Key Words: aortic valve ▪ catheter ablation ▪ mitral valve ▪ tachycardia, ventricular

The feasibility of catheter ablation of ventricular tachycardia (VT) associated with structural heart disease has improved dramatically with the use of substrate identification using the electroanatomical mapping systems and cardiac MRI. Sustained VT after valve surgery has been reported either early after surgery or years later, and a reentrant mechanism in a region of scar is the most common mechanism. However, in patients with prior mechanical mitral and aortic valves, the access to the left ventricle (LV) is limited, preventing an endocardial approach to ablation via a retrograde aortic or atrial transseptal approach. Here, to the best of our knowledge, we report the largest series of ablation for VT in these patients, demonstrate approaches including epicardial catheter ablation via a surgical pericardial window, surgical epicardial cryoablation, and surgical approach to endocardial catheter mapping, and provide the details of the arrhythmia substrate.

Methods
Patients with drug refractory VTs with aortic and mitral mechanical valve prostheses were included in the study. Ethical approval was obtained from the local institutional review committee. All patients gave their informed consent when retrospective data collection started. Echocardiography was performed, and the LV function was evaluated before the catheter ablation. All patients were anticoagulated with warfarin for the mechanical valves, and the warfarin was discontinued and bridged with a heparin infusion before the ablation or open-heart surgery. Heparin was discontinued 4 hours before the procedure. Multielectrode catheters were inserted and placed at the His recording site, coronary sinus and right ventricular apex. Programmed stimulation was performed to identify the target VT. Bundle branch re-entry or VT originating from the right ventricle was ruled out. If the VT was from the LV, a surgical pericardial window was used as an access to the epicardium with the use of pericardial adhesions. A 3-dimensional electroanatomical mapping system (Carto system, Biosense Webster, or EnSite NavX, St. Jude Medical) was used to identify the substrate and map the VT if allowed. Sites with abnormal potentials, such as fractionated signals,
WHAT IS KNOWN

- In patients with prior mechanical mitral and aortic valves, the access to the left ventricle is limited.
- Direct puncture or transvalvular access to the left ventricle has been reported.

WHAT THE STUDY ADDS

- Apical epicardial low voltage area may serve as a substrate for re-entrant ventricular tachycardia after a double valve replacement.
- Epicardial catheter ablation of ventricular tachycardia is a potentially useful therapy in patients who have mechanical aortic and mitral valves.

delayed isolated potentials, or double potentials, were tagged on the map. Entrainment and pace mapping were used for the identification of the isthmus of the VT circuit. Catheter ablation was performed using an external irrigated catheter (ThermoCool, Biosense Webster, Diamond Bar, USA, or Cool Path, St. Jude Medical, Minneapolis, USA). In one patient, 3-dimensional electroanatomical mapping was used to identify the re-entrant circuit during open-heart surgery. Briefly, before the operation, the location pad was properly placed underneath the operating table. An external reference patch that provided location reference data to the CARTO XP system was placed on the patient’s back, just underneath the heart. The ventricular epicardium was mapped by roving the 4-mm non-irrigated catheter (Navistar, Biosense Webster, Diamond Bar, CA, USA) directly on the epicardium.

Pathology

In patient 4, who underwent a surgical ablation, the resected tissue was fixed with 10% formaldehyde, embedded in paraffin, and sliced into 4-μm-thick sections. The sections were stained with Elastica van Gieson staining, and the images were recorded by a digital microscopy (BZ-X700; Keyence, Osaka, Japan). The longitudinal sections were divided into 3 layers (endocardium, midmyocardium, and epicardium). The red pixel content of the digitized images was detected as collagen fiber and measured relative to the total tissue area in each layer with a digital imaging analyzer (BZ-H3C; Keyence, Osaka, Japan). The longitudinal sections were divided into 3 layers (endocardium, midmyocardium, and epicardium). The red pixel content of the digitized images was detected as collagen fiber and measured relative to the total tissue area in each layer with a digital imaging analyzer (BZ-H3C; Keyence, Osaka, Japan).

In 2 patients (patients 1 and 4) who underwent an open-heart surgical ablation, an aneurysm or dense scar from the area thought to contain the re-entrant circuit was resected macroscopically or by 3-dimensional electroanatomical mapping system. Abnormal low voltage areas were identified in all of these patients, mainly at the apical inferior wall. One of 4 patients had recurrent VT after the catheter ablation and underwent an open-heart surgical ablation. A total of 3 patients underwent an open-heart surgical resection and cryoablation. In all patients, abnormal scar tissue was identified macroscopically or by 3-dimensional electroanatomical mapping during the surgery. Characteristics of the scars are shown in Table 3.

In 2 patients (patients 1 and 4) who underwent an open-heart surgical ablation, an aneurysm or dense scar from the area thought to contain the re-entrant circuit was resected and evaluated microscopically. Figure 1 (patient 4) shows the microscopic view of the myocardium with Elastica–Masson

Results

A total of 6 patients with refractory VT associated with double valve prostheses were included; one patient underwent open-heart surgical mapping and cryoablation with a resection in 2007, and the other 5 patients underwent ablation from May to December, 2012. In all patients, the pathogenesis of the valvular disease was rheumatic, and an implantable cardioverter defibrillator (ICD) was implanted before the ablation. Multiple antiarrhythmic drugs [2.5 (2 and 4.5)] had failed to control the VT before the ablation. The patient characteristics are described in Table 1. No patients had any inducible sustained bundle branch re-entry or VT originating from the right ventricle. VT was documented after 10.5 (8.5 and 18.5) years, and ablation was performed 15.5 (15 and 19.75) years after the surgery.

VT Morphology

The characteristics of the VTs are shown in Table 2. A total of 11 VTs were induced [1.5 (1 and 2.75) VT/patient]. All VTs except for one had right bundle branch morphology, and all but 2 VTs had a superior frontal plane QRS axis. In 3 patients, nonclinically documented VTs were inducible. All VTs were inducible with programmed stimulation and the mechanism was considered to be re-entrant. In 2 patients, the VT was incessant. All VTs were targeted for ablation.

Mapping and Substrate Identification

Epicardial mapping was performed in 4 patients via a surgical pericardial window using a 3-dimensional electroanatomical mapping system. Abnormal low voltage areas were identified in all of these patients, mainly at the apical inferior wall. One of 4 patients had recurrent VT after the catheter ablation and underwent an open-heart surgical ablation. A total of 3 patients underwent an open-heart surgical resection and cryoablation. In all patients, abnormal scar tissue was identified macroscopically or by 3-dimensional electroanatomical mapping during the surgery. Characteristics of the scars are shown in Table 3.

In 2 patients (patients 1 and 4) who underwent an open-heart surgical ablation, an aneurysm or dense scar from the area thought to contain the re-entrant circuit was resected and evaluated microscopically. Figure 1 (patient 4) shows the microscopic view of the myocardium with Elastica–Masson

Statistics

Data are presented as median (25th and 75th percentile).

Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Gender</th>
<th>LVEF (%)</th>
<th>Age at the DVR</th>
<th>Age at the Initial VT</th>
<th>Pathogenesis</th>
<th>Valvular Disease</th>
<th>Failed Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64</td>
<td>M</td>
<td>27</td>
<td>39</td>
<td>61</td>
<td>Rheumatic</td>
<td>AR, MS</td>
<td>Amio</td>
</tr>
<tr>
<td>2</td>
<td>71</td>
<td>M</td>
<td>35</td>
<td>58</td>
<td>69</td>
<td>Rheumatic</td>
<td>AsR, MS</td>
<td>Amio, Sotalol</td>
</tr>
<tr>
<td>3</td>
<td>66</td>
<td>M</td>
<td>40</td>
<td>51</td>
<td>61</td>
<td>Rheumatic</td>
<td>AsR, MS</td>
<td>Sotalol</td>
</tr>
<tr>
<td>4</td>
<td>59</td>
<td>M</td>
<td>25</td>
<td>44</td>
<td>51</td>
<td>Rheumatic</td>
<td>AR, MSr</td>
<td>Amio, Aprindine, Propafenone</td>
</tr>
<tr>
<td>5</td>
<td>75</td>
<td>M</td>
<td>21</td>
<td>59</td>
<td>71</td>
<td>Rheumatic</td>
<td>AR, MS</td>
<td>Amio, Mexiletine</td>
</tr>
<tr>
<td>6</td>
<td>64</td>
<td>M</td>
<td>29</td>
<td>43</td>
<td>63</td>
<td>Rheumatic+IE</td>
<td>AR, MS</td>
<td>Amio</td>
</tr>
</tbody>
</table>

Amio indicates amiodarone; AR, aortic valve regurgitation; AsR, mild aortic valve stenosis and moderate regurgitation; DVR, double valve replacement; IE, infectious endocarditis; LVEF, left ventricular ejection fraction; M, male; MS, mitral valve stenosis; MSr, mitral valve stenosis (moderate) and mild regurgitation; and VT, ventricular tachycardia.
Goldner stain. In this slice, the scar burden (area of scar tissue/total area) of the endocardium, mid myocardium, and epicardium was 34.8%, 43.4%, and 50.6%, respectively. More scar tissue was identified in the epicardial region. In patient 1, under direct visualization, an 8F sheath was inserted into the LV by a direct puncture. Endocardial substrate mapping was also performed during the surgery, which showed a smaller low voltage area compared with the epicardium, but mapping was limited because of the apical insertion of the sheath. From the sheath insertion site, 2 biopsies were performed and the tissue was stained with an Elastica–Masson Goldner stain (Figure 2). The section exhibited dense epicardial scar tissue and surviving myocardium was observed within 1 mm from the endocardium.

Ablation

In all but 2 patients, substrate mapping was conducted and areas with a low voltage and abnormal electrograms were identified, and the isthmus was identified by pace mapping or entrainment mapping. The majority of the VTs were unstable, and entrainment mapping was performed only in 2 VTs. Pace mapping was performed for the rest of the VTs.

In the open-heart surgical ablation patients, one patient had an aneurysmectomy with the earliest activation in the aneurysm recorded with a needle electrode (patient 4), and another patient underwent cryoablation after the electrophysiology study, which identified the exit of the re-entry circuit at the apical portion in the middle cardiac vein (patient 6). The results of ablation are shown in Table 4.

Table 2. Characteristics of Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>VT</th>
<th>Morphology</th>
<th>Axis</th>
<th>CL (ms)</th>
<th>Mapping Methods</th>
<th>Clinical</th>
<th>Access</th>
<th>Inducibility</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>VT1</td>
<td>RBBB</td>
<td>Superior</td>
<td>400</td>
<td>Pacemap</td>
<td>Clinical</td>
<td>Epi</td>
<td>NI</td>
</tr>
<tr>
<td>2</td>
<td>VT1</td>
<td>Inferior</td>
<td>Apex</td>
<td>750</td>
<td>Pacemap</td>
<td>Clinical</td>
<td>Epi</td>
<td>NI</td>
</tr>
<tr>
<td>3</td>
<td>VT1</td>
<td>Superior</td>
<td>Apex</td>
<td>480</td>
<td>Activation (surgery)</td>
<td>Clinical</td>
<td>Epi</td>
<td>NI</td>
</tr>
<tr>
<td>4</td>
<td>VT1</td>
<td>Superior</td>
<td>Apex</td>
<td>245</td>
<td>Pacemap</td>
<td>NC</td>
<td>Open heart</td>
<td>Induced</td>
</tr>
<tr>
<td>5</td>
<td>VT1</td>
<td>Superior</td>
<td>Apex</td>
<td>330</td>
<td>Activation</td>
<td>Clinical</td>
<td>Open heart</td>
<td>Induced</td>
</tr>
<tr>
<td>6</td>
<td>VT1</td>
<td>Superior</td>
<td>Apex</td>
<td>550</td>
<td>Entrainment</td>
<td>Clinical</td>
<td>Epi</td>
<td>Induced</td>
</tr>
</tbody>
</table>

CL indicates cycle length; LBBB, left bundle branch block; MCV, middle cardiac vein; NC, nonclinical; NI, noninducible; RBBB, right bundle branch block; and VT, ventricular tachycardia.

Table 3. Scar Characteristics

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Low Voltage Area (3D or Macroscopic)</th>
<th>Scar Area (&lt;1.5 mV) (cm²)</th>
<th>Dense Scar Area (&lt;0.5 mV) (cm²)</th>
<th>Total Points</th>
<th>Total Area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Apical</td>
<td>91</td>
<td>37.4</td>
<td>299</td>
<td>254.9</td>
</tr>
<tr>
<td>2</td>
<td>Apical</td>
<td>194.2</td>
<td>92.5</td>
<td>295</td>
<td>407.7</td>
</tr>
<tr>
<td>3</td>
<td>Apical</td>
<td>69.8</td>
<td>57.1</td>
<td>662</td>
<td>302.7</td>
</tr>
<tr>
<td>4</td>
<td>Apical, aneurysm</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>5</td>
<td>Anterior, apical, lateral</td>
<td>244.6</td>
<td>62.3</td>
<td>125</td>
<td>260</td>
</tr>
<tr>
<td>6</td>
<td>Apical</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

3D indicates 3-dimensional; and NA, not available.

Cases

Patient 1

A surgical epicardial access was obtained as a postsurgical adhesion was anticipated. After the manual release of a severe adhesion, an irrigated ablation catheter was inserted into the pericardial space. Programmed stimulation induced 3 hemodynamically unstable VTs (Figure 3). Then during a stable rhythm, substrate mapping was performed with special attention paid to the abnormal electrograms, such as fragmentation and delayed isolated potentials. A large low voltage area was identified on the LV apex extending to the lateral wall, and isolated delayed potentials and fragmented potentials were recorded (Figure 4). Sites with abnormal potentials and/or a good pace match were ablated. After the ablation, no VT was inducible with programmed stimulation. However, a VT with a slightly different morphology recurred on day 7. Then another attempt of an epicardial ablation failed because of severe adhesions at the ablation sites, despite the previous infusion of 2 mg/kg of methyl prednisolone into the pericardial space at the end of the first procedure. Although the VT was infrequent, the patient wished to proceed to a surgical ablation, as the patient experienced post-traumatic stress disorder from the previous frequent shocks.

Open-heart surgical ablation was performed using the previously described method. The ventricular epicardium was mapped by roving the 4-mm nonirrigated catheter (Navistar, Biosense Webster, Diamond Bar, CA, USA) directly on the epicardium. In addition to the substrate mapping of the epicardium (Figure 5A), endocardial mapping was performed with a direct puncture of the LV (Figure 5B). Hemodynamic support with cardiopulmonary bypass allowed the mapping of unstable VTs, and the activation map showed a figure of 8 reentrant circuit (Figure 5C). Then it was switched to on-pump surgery. Two myocardial biopsies were performed from the sheath insertion site. At the identified earliest activation sites with abnormal electrograms, cryoablation was performed. No VT was inducible with programmed stimulation from the ICD after the ablation. The patient recovered well from the surgery and during a follow-up of 18 months, and no VT recurrence was observed without any antiarrhythmic agents. The biopsied tissue was stained with Elastica–Masson Goldner.
staining (Figure 5). Dense fibrosis was observed 100% in mid-myocardium and epicardium, and only 1 mm of the endocardium exhibits residual myocardium.

**Patient 2**
Surgical epicardial access was obtained following the program stimulation from the ICD. Nonclinical VT was inducible by the program stimulation from the ICD. A substrate map obtained during sinus rhythm showed multiple sites with isolated potentials and extensive low voltage areas on the LV inferior and apical wall. All sites with delayed potentials or fractionated potentials were targeted for ablation. After the ablation, no VT was inducible with programmed stimulation. During a follow-up of 18 months, no VT recurrence was observed.

**Patient 3**
Surgical epicardial access was obtained, and programmed stimulation induced 3 types of VTs (Figure 6). Substrate mapping during sinus rhythm showed an apical low voltage area. Pace mapping was performed in the low voltage area with a stimulus strength change using the Paso Module, which automatically compares the 12 ECG signals of the pace map with the targeted premature ventricular contraction/VT ECG signals by calculating the correlation values. The correlation threshold for matched and unmatched signals was set at 0.80. If a QRS match >80% in >10/12 leads was observed, the site was tagged as orange (Figure 7). At 1 site (tagged as *), pace mapping with different stimulus strengths produced different QRS morphologies, and each matched 2 induced VTs (Figure 8). Entrainment mapping at this site confirmed that the site was located at the exit of the isthmus. A radiofrequency application at this site terminated the VT. Additional radiofrequency applications were made at all sites with abnormal electrograms. After the ablation, no VT was inducible with programmed stimulation. During a follow-up of 18 months, no VT recurrence was observed.

**Patient 4**
This patient had an LV anterior aneurysm. A preoperative hypokinetic area progressed into an aneurysm over 7 years after the valve replacement. During the surgery, an activation map using the needle electrode showed the earliest activation and low voltage fractionated electrograms in the aneurysm; therefore, an aneurysmectomy and cryoablation at the border were performed. An Elastica van Gieson stain of the resected tissue is shown in Figure 1.

**Patient 5**
A substrate map during sinus rhythm exhibited extensive inferior-lateral abnormal low voltage area. Entrainment mapping from the epicardial lateral wall identified the exit site. Ablation was limited because of the extensive adhesion, and the VT remained inducible. The patient died within a week because of the intractable VT and heart failure.

**Patient 6**
An electrophysiology study was performed and conventional activation mapping and entrainment mapping were performed, which revealed that the exit location was at the distal portion of the middle cardiac vein. A radiofrequency application in the middle cardiac vein was unsuccessful, and open-heart cryoablation was performed. During the surgery, no mapping

![Figure 2.](image-url) The microscopic view of the myocardial biopsy with an Elastica–Masson Goldner stain is shown. Myocardial tissue is stained red, elastic fibers are stained purple, and collagen fibers are stained green. The right side is the endocardial side and 100% fibrosis is observed in the midmyocardium and epicardium. Only 1 mm of the endocardium exhibits residual myocardium.

![Figure 1.](image-url) The microscopic view of the excised left ventricular aneurysm with an Elastica Van Gieson stain is shown. Myocardial tissue is stained yellow, elastic fibers are stained black, and collagen fibers are stained red. An increase in the interstitial fibrosis because of collagen deposition and an irregularity in the diameter of the cardiac muscle fibers are observed.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Type of Ablation</th>
<th>Method of Mapping</th>
<th>Abnormal EGM</th>
<th>Acute Result</th>
<th>Recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RF/open-heart surgery</td>
<td>Pacemap</td>
<td>Dip, Frag</td>
<td>Success</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>RF</td>
<td>Entrainment pacemap</td>
<td>Dip, Frag</td>
<td>Success</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>RF</td>
<td>Entrainment pacemap</td>
<td>Dip, Frag</td>
<td>Success, ATP</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Open-heart surgery</td>
<td>Pacemap</td>
<td>Modified</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>RF</td>
<td>Entrainment pacemap</td>
<td>Dip, Frag</td>
<td>Failure, Death</td>
<td>Death</td>
</tr>
<tr>
<td>6</td>
<td>Open-heart surgery</td>
<td>Entrainment pacemap</td>
<td>Success</td>
<td>No</td>
<td></td>
</tr>
</tbody>
</table>

ATP indicates antitachycardia pacing; Dip, diastolic isolated potential; EGM, electrogram; Frag, fragmented potential; and RF, radiofrequency catheter ablation.
was performed, and cryoablation along the middle cardiac vein was performed.

**Discussion**

This study showed that an apical epicardial abnormal low voltage area served as a substrate for the re-entrant VT. In 2 patients, epicardial ablation abolished all VTs, and in 2 patients an open-heart surgical scar resection was successful in abolishing the VTs. A resected tissue evaluation revealed a more dominant scar burden on the epicardium than the endocardial surface.

**Arrhythmogenic Substrate**

To the best of our knowledge, the characteristics and pathogenesis of the scar in these patients have not been evaluated in the past. Cesario et al\(^5\) reported the value of high density mapping, and one of their patients was postmitral valve replacement patient. That patient had end-stage valvular heart disease and underwent a heart transplant. A pathological specimen demonstrated that the epicardial region had a fibro-fatty infiltrate near the mitral prosthesis. In our patients, combined endocardial and epicardial mapping was performed in only 1 patient. Therefore, the existence of the perivalvular scar was not confirmed. On the epicardium, dense scar was noted in the apical area but lesser in the basal area (Figures 4, 5 and 7). This may be because of the underlying disease or surgery-related scar formation, but remains speculative.

Although the cause of the presumed scar was not clear, several possibilities exist. In severe aortic regurgitation, the coronary reserve is reduced, and myocardial ischemia can occur even in the absence of a coronary obstruction. Other possibilities are myocardial fibrosis because of rheumatic myocarditis, cardioplegia-associated myocardial damage, or myocardial fibrosis and ventricular remodeling as a result of severe aortic regurgitation or an embolic myocardial infarction. One study evaluated the endomyocardial biopsy specimen undergoing aortic valve replacement because of aortic regurgitation.\(^6\) Their results showed that all patients with aortic regurgitation had a high content of fibrosis. It was speculated that the disease process affecting the valve and the adjacent myocardium, and the surgical procedure, might somehow explain the observed VT substrate. Eckart reported a series of 20 patients with post valvular replacements.\(^1\) In 14 patients, scar-related re-entrant...
VT was observed, and 9 of 14 patients had abnormal low voltage areas close to the annulus. The anteroapical scars might have been related to the surgery or remodeling linked to the prosthetic valves, sutures, or cardioplegia. We did not observe any incisions or visible scars, which could have been caused by the LV vent. The previous surgical records could not be obtained as all operations were performed at other hospitals almost 20 years prior.

This apical low voltage area extended to the lateral and basal area, and abnormal local electrograms, such as fractionation and isolated delayed potentials, were recorded in these areas. In 1 patient, endocardial mapping was conducted, which also showed the low voltage area at the apex. If the delayed enhanced cardiac MRI was performed before the aortic and mitral valve replacement, the scar area and distribution could be evaluated.

**How to Approach the VT in a Double Valve Replacement**

In patients who had previous double prostheses, mitral and aortic valves, the usual approaches for endocardial access to the LV are not available. Some VTs can be ablated from the right ventricle. Bundle branch re-entry is not rare and easily ablable without access to the LV. Therefore, recording
the His and right bundle should be performed. The retrograde approach through the prosthetic aortic valve has been reported but could lead to damage of the mechanical valve. Alternative approaches include a puncture via the interventricular septum, direct LV puncture, or an epicardial approach. The transcutaneous epicardial approach has dramatically improved the efficacy of catheter ablation, especially in nonischemic cardiomyopathy. In patients with prior cardiac surgery or pericarditis, a subxyphoid pericardial window is usually chosen as an access to the epicardium because of the severe adhesions. Tschabrunn et al reported a percutaneous epicardial approach in 10 patients with prior noncoronary cardiac surgery or pericarditis, including 3 patients with aortic valve replacements. In their series, dense pericardial adhesions limiting the ability to map the entire epicardial space was experienced; however, appropriate targets could be reached and ablated by disrupting the adhesions with the ablation catheter and deflectable sheath, facilitating an excellent long-term clinical outcome in half of the patients with no major complications. In our series, a percutaneous approach was not attempted.

In all 4 patients in our series who had an epicardial access with a surgical window, it was possible to map and ablate the VT to some extent. In 1 patient, a severe adhesion did not allow the access to the critical portion of the VT circuit. All of our patients already had ICDs implanted before the ablation, and cardiac MRI could not be performed. Preoperative identification of the scar location could potentially help to plan the ablation. If the substrates were dominant on the endocardium, it would be difficult to abolish the VTs from the epicardial access, requiring a surgical based ablation. If the scar is predominant on the epicardium, an epicardial catheter ablation likely could control the VTs. Bogun et al reported the usefulness of delayed enhanced MRI as a guide for VT in patients with nonischemic cardiomyopathies. Detection of the scars using delayed enhanced MRI could have an important significance for planning the ablation.

Limitations

The present study included a small number of patients and was retrospective. However, to our knowledge, the current study included the largest series of patients thus far. The mapping was limited by the postoperative adhesions. In 2 patients,
a 3-dimensional electroanatomical mapping system was not used: one had a macroscopic evaluation of the scar using the previous electrophysiological study findings to identify the circuit and had a scar resection and cryoablation, while the other underwent unipolar needle mapping to identify the earliest activation and low voltage area in the aneurysm. Although previous reports showed a frequent scar distribution in the perivalvular area,1,5 1 patient in our study, who underwent endocardial mapping, did not exhibit any clear perivalvular scar. However, combined endocardial and epicardial mapping was not performed in the rest of the patients, so an endocardial perivalvular substrate cannot be excluded.

It is beyond the scope of our article to generalize the specific findings to all rheumatic disease patients. Despite these limitations, we think that the abnormal scar areas were present in the epicardial apical area in all patients.

Conclusions
Recurrent monomorphic VT after a double valve replacement is often because of re-entry in a scar located at the apex, and epicardial catheter ablation can be effective in controlling the VT.

Acknowledgments
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Disclosures
Dr Soejima has received honoraria for lectures from St. Jude Medical Japan, Boston Scientific Japan, and is a steering committee member for Micra, Medtronic. Dr Nogami has received honoraria from St. Jude Medical and Boston Scientific, and an endowment from Medtronic and Johnson & Johnson. The other authors report no conflicts.

References


Epicardial Catheter Ablation of Ventricular Tachycardia in No Entry Left Ventricle: Mechanical Aortic and Mitral Valves
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