Percutaneous Pericardial Instrumentation for Endo-Epicardial Mapping of Previously Failed Ablations

Robert A. Schweikert, MD; Walid I. Saliba, MD; Gery Tomassoni, MD; Nassir F. Marrouche, MD; Christopher R. Cole, MD; Thomas J. Dresing, MD; Patrick J. Tchou, MD; Dianna Bash, RN; Salwa Beheiry, RN; Cathy Lam, MD; Logan Kanagaratnam, MD; Andrea Natale, MD

Background—The epicardial location of an arrhythmia could be responsible for unsuccessful endocardial catheter ablation.

Methods and Results—In 48 patients referred after prior unsuccessful endocardial ablation, we considered percutaneous, subxiphoid instrumentation of the pericardial space for mapping and ablation. Thirty patients had ventricular tachycardia (VT), 6 patients had a right- and 4 had a left-sided accessory pathway (AP), 4 patients had inappropriate sinus tachycardia, and 4 patients had atrial arrhythmias. Of the 30 VTs, 24 (6 with ischemic cardiomyopathy, 3 with idiopathic cardiomyopathy, and 15 with normal hearts) appeared to originate from the epicardium. Seventeen (71%) of these 24 VTs were successfully ablated with epicardial lesions. The other 7 VTs had early epicardial sites that were inaccessible, predominantly because of interference from the left atrial appendage. Six of these were successfully ablated from the left coronary cusp. In 5 of the 10 patients with an AP, the earliest activation was recorded epicardially. Three of these were right atrial appendage–to–right ventricle APs, and epicardial ablation was successful. No significant complications were observed.

Conclusions—Failure of endocardial ablation could reflect the presence of an epicardial arrhythmia substrate. Epicardial instrumentation and ablation appeared feasible and safe and provided an alternative strategy for the treatment of patients with a variety of arrhythmias. This was particularly true for VT, including patients without structural heart disease. (Circulation. 2003;108:1329-1335.)

Key Words: tachyarrhythmias ■ electrophysiology ■ catheter ablation ■ mapping ■ pericardium

Percutaneous, transvenous, endocardial catheter ablation has become the preferred approach for the treatment of several types of arrhythmias. However, this approach has limitations, including the inability to access intramural or epicardial portions of arrhythmia circuits. Technological improvements, such as cooled-tip or larger-tip ablation catheters and different energy sources for tissue ablation, have not completely solved the problem.

Before the advent of percutaneous catheter–based endocardial ablation methods, surgical epicardial approaches were used for treatment of refractory arrhythmias, particularly ventricular tachycardia (VT) and supraventricular tachycardia (SVT), by using an accessory pathway (AP). Epicardial ablation has been achieved through various methods, including open-chest surgery,1–2 thoracoscopy,3–4 and by way of epicardial vessels, such as the coronary sinus.5 However, despite the development of catheter-based, endocardial ablation techniques, some arrhythmia substrates might not be accessible from the endocardium. For example, VT from ischemic cardiomyopathy might have significant portions of the arrhythmia circuit in the epicardium.6–10 More recently, the percutaneous approach for epicardial mapping and ablation of VT has been shown to be feasible, primarily in patients with Chagasic heart disease11–13 and ischemic cardiomyopathy.14,15

In light of the experience of others with the percutaneous, epicardial approach and the evidence that an epicardial arrhythmia substrate might be responsible for ineffective endocardial ablation, we offered the percutaneous approach in the electrophysiology laboratory for epicardial mapping and ablation as a potential feasible and effective treatment for such refractory arrhythmias.

Methods

Patient Population

Consecutive patients referred after unsuccessful endocardial ablation were considered for percutaneous, epicardial mapping and ablation. Patients were referred from a broad range of facilities, including the community as well as tertiary settings. No patients were excluded from consideration. Informed consent for the procedure was obtained from each patient. Data from these procedures were entered into an established Electrophysiology Laboratory Database approved by the Cleveland Clinic Foundation Institutional Review Board.
Electrophysiology Study

Patients were studied in the Electrophysiology Laboratory in the postabsorptive state under conscious sedation. Antiarrhythmic drugs were discontinued several days before the procedure, with the exception of amiodarone. Transvenous, multipolar catheters for endocardial recording and stimulation were positioned into the cardiac chambers appropriate for the target arrhythmia. Programmed stimulation for arrhythmia induction was performed from endocardial catheters according to standard protocols. Induction of VT was attempted with standard techniques of burst ventricular pacing and programmed stimulation, including up to 3 ventricular extrastimuli at 2 right ventricular sites. Provocation of the VT and/or premature ventricular contractions (PVCs) for patients with normal hearts included the use of intravenous isoproterenol and/or phenylephrine when necessary.

Percutaneous Subxiphoid Instrumentation of the Pericardial Space

Pericardial puncture was performed with a 17-gauge, epidural needle (Arrow International Inc) in a manner similar to that previously described,13 which is a modification of the traditional pericardiocentesis technique.16

Endocardial and Epicardial Mapping and Ablation

A 7F 4-mm-tip deflectable ablation catheter (Navistar, Cordis Webster) was used for endocardial and epicardial mapping and ablation. All patients underwent both endocardial and epicardial mapping, and ablation was first attempted endocardially when a suitable arrhythmia substrate was found at that site. Analysis of suitable sites for ablation depended on the target arrhythmia and also consisted of standard criteria commonly used for endocardial approaches, including activation and pace mapping, as well as entrainment methods. Mapping was performed with a nonfluoroscopic electroanatomic system (Biosense Webster CARTO). When indicated and not previously attempted, endocardial ablation was performed with a cooled-tip ablation catheter (Cardiac Pathways CHILLI or Biosense Webster Navistar ThermoCool). Epicardial ablation lesions were delivered with a standard radiofrequency generator (EP Technologies). Power for epicardial lesions was automatically adjusted to achieve a temperature of ~50°C. The

### TABLE 1. Results of Percutaneous, Epicardial Mapping and Ablation of VTs

<table>
<thead>
<tr>
<th>Location of VT</th>
<th>Heart Disease</th>
<th>Earliest/Best Site</th>
<th>Successful RFCA Site</th>
<th>Complications</th>
<th>Recurrence</th>
</tr>
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<tbody>
<tr>
<td>AL LVOT</td>
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<tr>
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<tr>
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<tr>
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<tr>
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RFCA indicates radiofrequency catheter ablation; AL, anterolateral; LVOT, left ventricular outflow tract; LV, left ventricle; Post, posterior; PL, posterolateral; RVT, right ventricular outflow tract; RV, right ventricle; PS, posteroseptal; AS, anteroseptal; Lat, lateral; IA, inferoapical; NCC, noncoronary cusp; LCC, left coronary cusp; DCM, dilated cardiomyopathy; and ICM, ischemic cardiomyopathy.
definition of successful ablation depended on the target arrhythmia and generally consisted of no spontaneous or inducible occurrence of the target arrhythmia or ectopic beat after ablation.

**Follow-Up**

After the procedure, patients were treated with an oral nonsteroidal antiinflammatory medication for 2 weeks. Each patient returned for evaluation in the outpatient clinic at 1, 3, 6, and 12 months, at minimum, after the procedure. An echocardiogram was performed the day after the procedure and at the 3-month follow up visit. A cardiac event monitor was provided for the first month after the procedure, and an ambulatory Holter monitor record was obtained at 3 and 6 months after the procedure. Patients with an implantable cardioverter defibrillator (ICD) underwent device interrogation at each follow-up evaluation to assess for arrhythmia recurrence. Cardiac stress testing was performed for patients who had undergone ablation near epicardial coronary vessels. Long-term success was defined as the lack of recurrence of the target arrhythmia.

**Results**

**Patient Characteristics**

Forty-eight patients were included in this series (Tables 1 and 2). The mean ± SD age was 44.6 ± 2.8 years (range, 14 to 81 years). Thirty-four (71%) of the patients were male. The mean number of previous ablation procedures for the entire series was 1.4 ± 0.6 (range, 1 to 3). None of the patients had had prior cardiac surgery, although such patients were not excluded. Percutaneous pericardial access was achieved in all patients in whom it was attempted. The mean ± SD number of epicardial lesions delivered per patient was 2.1 ± 0.3 (range, 0 to 6).

**Ventricular Arrhythmias**

Table 1 summarizes the results of 30 patients with ventricular arrhythmias, consisting of sustained VT, nonsustained VT, and/or frequent, symptomatic PVCs. Twenty patients had normal hearts, 7 had ischemic cardiomyopathy, and 3 had dilated cardiomyopathy. The patients with cardiomyopathy had sustained VT clinically and VT induced during the electrophysiology study. Of the 20 patients with normal hearts, 2 had incessant VT, and the remaining 18 patients had frequent, daily episodes of symptomatic ventricular ectopy, including nonsustained VT and/or PVCs. The mean ± SD number of daily episodes of nonsustained VT and PVCs documented by ambulatory Holter monitoring before the catheter ablation procedure was 22 ± 7 and 5600 ± 1100, respectively. For the 12 patients with ICDs, the mean ± SD number of ICD shocks for VT in the month before the catheter ablation procedure was 12 ± 4. Endocardial ablation with a cooled-tip ablation catheter had been unsuccessful in all patients with ventricular arrhythmias. Ten patients with VT were taking amiodarone, and this medication was discontinued when the ablation procedure appeared to be successful in the short term.

Twenty-four (80%) of the 30 VTs appeared to originate from the epicardium, as demonstrated by activation and/or pace mapping (Table 1). Six of these 24 VTs could be ablated only from the left coronary cusp, because epicardial lesions could not be delivered owing to interference from the left atrial appendage. Of the 18 remaining patients with normal hearts, 2 had incessant VT, and the remaining 18 patients had frequent, daily episodes of symptomatic ventricular ectopy, including nonsustained VT and/or PVCs. The mean ± SD number of daily episodes of nonsustained VT and PVCs documented by ambulatory Holter monitoring before the catheter ablation procedure was 22 ± 7 and 5600 ± 1100, respectively. For the 12 patients with ICDs, the mean ± SD number of ICD shocks for VT in the month before the catheter ablation procedure was 12 ± 4. Endocardial ablation with a cooled-tip ablation catheter had been unsuccessful in all patients with ventricular arrhythmias. Ten patients with VT were taking amiodarone, and this medication was discontinued when the ablation procedure appeared to be successful in the short term.

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of the 24 VTs with an early epicardial site, epicardial pacing could not be achieved because of inability to capture, even with a high stimulation output. For the 15 patients in whom epicardial pace mapping was possible, nearly identical QRS morphology was demonstrated that was never seen with endocardial pace mapping (Figure 1).

For the group of VTs ablated successfully from the epicardial approach, Table 3 summarizes the activation mapping of endocardial versus epicardial sites according to the various types of VT. Epicardial sites were earlier. Six of the 7 ischemic VTs were ablated epicardially, and in 5 of these 6 VTs, the epicardial ablation sites showed middiastolic potentials never seen endocardially. Overall, 28 (93%) of the 30 VTs were successfully ablated with radiofrequency catheter ablation, by endocardial, epicardial, or coronary cusp approaches. Of the 2 unsuccessful VT ablations, one did not have epicardial early sites and could not be ablated endocardially, and the other was the epicardial VT that arose from the anterolateral left ventricle, as described earlier.

SVTs: APs, Inappropriate Sinus Tachycardia, and Atrial Fibrillation
Table 2 summarizes the experience with percutaneous, epicardial mapping and ablation of various forms of SVT. Successful epicardial ablation required a mean ± SD of 3 ± 1 (range, 1 to 4) lesions for this group. Ten patients with APs and SVT underwent percutaneous, epicardial mapping. All except 2 of the patients with APs had failed previous ablation with a cooled-tip ablation catheter. The 8 manifest APs were mapped in sinus rhythm, and in 5, the earliest ventricular activation was recorded epicardially. Three of these 5 manifest APs were right atrial appendage–to–right ventricle APs that showed earliest activation epicardially, where successful ablation was finally achieved. In the other 2 manifest APs, epicardial lesions were only transiently successful (right posterolateral AP) or undeliverable because of excessive impedance (left posteroseptal AP). Both were ultimately ablated from an endocardial approach. The left posteroseptal AP was associated with a large coronary sinus diverticulum and was ablated from that site. Three other manifest APs (left posterolateral, left posteroseptal, and right posterolateral) were not found to have early epicardial sites. The 2 concealed APs were mapped in tachycardia and/or ventricular pacing and were not found to have epicardial early sites.

The percutaneous epicardial approach was generally ineffective in our limited experience with inappropriate sinus

Figure 1. A, Surface 12-lead ECG of normal heart VT. B, Epicardial pace map at successful ablation site on posterolateral left ventricle. This demonstrates a nearly perfect pace map that was better than any endocardial pace map.

Figure 2. CARTO activation maps of VT from patient described in legend to Figure 1. Earliest ventricular activation is denoted in red, and latest ventricular activation, in purple. A, Endocardial (ENDO) activation map (posteroanterior view) alone; B, endocardial (ENDO) and overlying epicardial (EPI) activation maps (right posterior oblique cranial view). Note that earliest site of activation has shifted to epicardium in B. MV indicates mitral valve.
In addition, the epicardial approach for IST might be less desirable because of the number of ablation lesions typically required for sinus node modification.

Overall Results
All 48 patients underwent successful instrumentation of the pericardial space via a percutaneous, subxiphoid approach. Thirty-one (65%) patients were found to have an epicardial arrhythmia substrate (24 with VT, 2 with IST, and 5 with AP). Seven of the 24 VT patients and 2 of the 5 patients with AP had inaccessible epicardial arrhythmia sites, generally because of interference from the left atrial appendage or the posterior fat pad, respectively. Of the 22 patients with an accessible epicardial site, epicardial ablation was successful in 21 (95%) patients. There were no complications that required intervention or treatment during or after the procedure.

Follow-Up
The mean ± SD follow-up was 25.4 ± 12.9 months. No patients were lost to follow-up. Of the 21 patients who were found to have an epicardial arrhythmia substrate and underwent successful epicardial ablation, 2 patients experienced recurrence of the original target arrhythmia. One patient had atrioventricular reentrant tachycardia with a right atrial appendage-to-right ventricle AP. A second epicardial ablation procedure was successful. Another patient with dilated cardiomyopathy had an epicardial anterolateral left ventricular VT. The site of earliest activation and nearly perfect pace map were not suitable for ablation because of phrenic nerve stimulation with pacing. At a more lateral location, there was no phrenic nerve stimulation, and ablation was successful in the short term. However, the VT subsequently recurred.

For the patients with VT, repeated ambulatory Holter monitoring (and device interrogation in those patients with an ICD) demonstrated no recurrence of the target VT. After catheter ablation, the mean ± SD number of daily PVCs decreased to 35 ± 24 and had a different morphology from that of the target PVCs. Three patients had symptoms of pericarditis that persisted >24 hours after ablation but that resolved within 1 week. No patient had evidence of new abnormalities by ECG or echocardiogram, and for those patients who underwent ablation near an epicardial vessel, cardiac stress imaging at follow-up did not reveal new abnormalities in the distribution of that vessel.

Table 3. Endocardial vs Epicardial Activation Times for the 17 VTs Successfully Ablated by the Percutaneous, Epicardial Approach

<table>
<thead>
<tr>
<th>VT Substrate</th>
<th>Endocardial Activation Pre-QRS (Mean ± SD), ms</th>
<th>Epicardial Activation Pre-QRS (Mean ± SD), ms</th>
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<tr>
<td>Normal heart</td>
<td>11 ± 6</td>
<td>36 ± 5</td>
</tr>
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<td>DCM (n=2)</td>
<td>15 ± 5</td>
<td>43 ± 10</td>
</tr>
<tr>
<td>ICM (n=6)</td>
<td>42 ± 10</td>
<td>106 ± 30</td>
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DCM indicates dilated cardiomyopathy and ICM, ischemic cardiomyopathy.
One patient with severe, end-stage ischemic cardiomyopathy and refractory VT who underwent successful epicardial ablation expired from decompensated congestive heart failure several weeks after the ablation procedure. There was no evidence of acute myocardial infarction or pericardial effusion by echocardiography. An autopsy was not performed.

Discussion

Our series demonstrates that percutaneous, pericardial instrumentation for epicardial mapping and ablation of a variety of supraventricular and ventricular arrhythmias is feasible and safe. Other investigators have demonstrated that this approach is safe and effective for VT, primarily in cases of Chagasic heart disease. However, use of this approach for other types of arrhythmias in a series of patients has not been reported.

We report our experience in patients with a variety of tachyarrhythmias that were refractory to endocardial catheter ablation. A high proportion of the patients with VT in our series had an epicardial arrhythmia substrate that was successfully ablated with percutaneous, epicardial lesions. In this regard, this is the first report of a series that included normal-heart VTs that originated and were successfully ablated from the epicardial surface of the ventricle.

Our results indicate that the yield of this approach is highest for VT and SVT with a right atrial appendage–to–right ventricle AP. Right atrial appendage–to–ventricle APs have been described and might be ablated with endocardial lesions in the right atrial appendage. Therefore, this approach should be considered before a percutaneous, epicardial approach is attempted. Indeed, in our experience (personal communication), nearly half of such pathways did not require epicardial ablation. However, endocardial ablation failures have been reported. In some instances, such patients have been referred for surgery. The demonstration that a percutaneous, epicardial approach for right atrial appendage–to–right ventricle APs is safe and effective is therefore important, and such patients should be considered for this approach before resorting to more invasive measures.

In our series, other types of APs were not successfully ablated with this technique, despite recording the earliest activation times from the epicardium. All of these APs were along the posterior atrioventricular groove. This could reflect a more exuberant presence of pericardial fat in this region that prohibited effective delivery of energy with radiofrequency ablation.

Potential Complications and Technical Issues

One concern is the potential for damage to the epicardial vessels during this procedure, either with the epidural needle or with delivery of epicardial radiofrequency lesions. Neither problem was observed in our series. Other investigators have used various methods to avoid ablation in the region of the epicardial vessels, such as radiographic landmarks, placement of catheters or injection of contrast within the coronary sinus to define the atrioventricular groove, and left coronary angiography.

We performed coronary angiography in approximately half of the patients who underwent VT ablation in our series. None of the ablation sites were on or immediately adjacent to a coronary artery. However, a target site near an epicardial coronary vessel was not considered prohibitive for application of ablation lesions. Interestingly, for the 17 VTs that were successfully ablated with epicardial lesions, the site of successful ablation appeared to be located near the atrioventricular or interventricular grooves, along the course of major epicardial vessels (Figure 4). This was true for patients with ischemic cardiomyopathy as well as those with normal hearts. Although epicardial vessel damage during ablation remains a concern, recent animal data have shown that radiofrequency lesions delivered over epicardial vessels produce significant damage only in the smallest epicardial coronary arteries.

A potential limitation of percutaneous, pericardial instrumentation involves patients with prior cardiac surgery in whom postoperative, pericardial adhesions could limit access to the pericardial space. Although patients with prior cardiac surgery were not excluded from consideration for percutaneous, pericardial instrumentation, none of the patients in this
series had undergone prior cardiac surgery. A potential advantage of the percutaneous, epicardial approach is avoidance of endovascular complications that might be encountered with conventional endocardial techniques, such as vascular injury, valve damage, and embolism from coagulum or dislodged plaque during left-sided ablation procedures. In addition, the use of intravenous heparin and its associated complications could be avoided.

**Conclusions**

Percutaneous, pericardial instrumentation for epicardial catheter mapping and ablation is feasible and safe and may be applicable to a variety of cardiac arrhythmias in which standard endocardial ablation techniques have failed. The technique appears to be particularly effective for VTs, including patients without structural heart disease. A significant number of failed ablations with standard endocardial ablation methods might represent an epicardial arrhythmia substrate, and percutaneous, epicardial mapping and ablation should be considered an adjunctive approach for such patients. Further study is required to better define the types of arrhythmias best suited to this approach.

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**References**


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