Subxiphoid Surgical Approach for Epicardial Catheter-Based Mapping and Ablation in Patients With Prior Cardiac Surgery or Difficult Pericardial Access

Kyoko Soejima, MD; Gregory Couper, MD; Joshua M. Cooper, MD; John L. Sapp, MD; Laurence M. Epstein, MD; William G. Stevenson, MD

Background—Percutaneous epicardial mapping and ablation are successful in some patients with ventricular epicardial reentry circuits but may be impossible when pericardial adhesions are present, such as from prior cardiac surgery. The purpose of this study was to evaluate the feasibility of direct surgical exposure of the pericardial space to allow catheter epicardial mapping and ablation in the electrophysiology laboratory when percutaneous access is not feasible.

Methods and Results—In 6 patients with prior cardiac surgery or failed percutaneous pericardial access, a subxiphoid pericardial window was attempted. In all 6 patients, manual lysis of adhesions exposed the epicardial surface of the heart through a small subxiphoid incision and allowed placement of an 8F sheath into the pericardial space under direct vision. Access to the diaphragmatic surface of the heart with ablation catheters was achieved in all patients, and catheter manipulation to the lateral and anterior walls was possible in 4 patients. Three-dimensional electroanatomic voltage maps revealed low-amplitude regions in the inferior or posterior left ventricular epicardium. A total of 16 ventricular tachycardias were induced, and 14 were abolished by radiofrequency ablation. Ablation was limited by intrapericardial defibrillator patches adherent to the likely target region in 2 patients. All patients had chest pain consistent with pericarditis early after the procedure that resolved within a few days. There were no other complications.

Conclusions—A direct surgical subxiphoid epicardial approach in the electrophysiology laboratory is feasible for patients with difficult pericardial access who require ablation of epicardial arrhythmia foci. (Circulation. 2004;110:1197-1201.)

Key Words: tachycardia □ ablation □ epicardium

The majority of ventricular tachycardias (VTs) after myocardial infarction can be ablated with radiofrequency (RF) ablation from endocardium. However, ≈15% of patients have 1 or more VTs that originate from the epicardium; this occurs particularly in patients with prior inferior wall infarction.1 In patients with dilated cardiomyopathy, epicardial reentry circuits may be even more common.2,3 Sosa and coworkers4 described a percutaneous method of inserting a mapping and ablation catheter into the pericardial space, even in the absence of pericardial fluid, which has allowed successful catheter ablation. A percutaneous approach may be difficult or not feasible when prior cardiac surgery has created pericardial adhesions. The purpose of the present study was to evaluate the feasibility of using a direct subxiphoid surgical approach to expose the pericardial space in the electrophysiology laboratory for epicardial mapping and ablation in patients with failed percutaneous epicardial access or patients with prior cardiac surgery.

Methods

Patient Population
From April 2002 to February 2004, epicardial mapping and ablation were attempted in 31 patients. Percutaneous access to the epicardial space was attempted in 26 patients and was achieved in 24 patients. Surgical access to the epicardium was attempted in 6 patients who met the following criteria: recurrent VT, failed endocardial catheter ablation due to absence of identifiable endocardial target sites, and the fact that percutaneous epicardial ablation was either not attempted because of prior cardiac surgery (3 patients) or was unsuccessful because of inability to define and enter the epicardial space (3 patients, including 2 with prior cardiac surgery). Characteristics of the patients are shown in the Table. Mapping and ablation were performed according to procedures approved by the human subjects protection committee after patient consent was obtained.

Pericardial Exposure
Under general anesthesia with endotracheal intubation in the electrophysiology laboratory, a 3-inch vertical incision was made in the midline epigastrium. The abdominal fascia was opened in the linea alba, veering to the left of the xiphoid process superiorly. The pericardium was exposed and opened horizontally, parallel to the
diaphragmatic reflection. The pericardiotomy was extended to the patient's left to improve visualization of the ventricles. Blunt dissection of adhesions was performed to fully expose the diaphragmatic and posterior epicardium. Then, an 8F sheath was inserted into the pericardial space. Through the sheath, a 7F mapping and ablation catheter with a 4-mm tip (Navi Star, Biosense-Webster or Chili catheter, Boston Scientific) was inserted into the pericardial space (Figure 1).

Mapping and Ablation

The epicardial surface was mapped by the method that we have used for endocardial mapping and ablation, with fluoroscopy and an electroanatomic mapping system used to guide catheter location (Carto, Biosense-Webster, Inc). Bipolar electrograms were recorded on the electroanatomic mapping system (filtered at 10 to 400 Hz) and on a separate digital system (filtered at 30 to 500 Hz; Prucka Engineering Inc). Pace mapping and entrainment mapping were performed with unipolar pacing to determine the proximity to the reentry circuit isthmus (Figure 2A). Sinus rhythm maps of peak-to-peak electrogram amplitude (voltage maps) were created that delineated low-voltage regions as those <1.5 mV (Figure 2B). After the ablation target region was selected, coronary angiography was performed to assess proximity to an epicardial coronary artery or bypass graft (Figure 2C). Unipolar pacing at 10 mA and 2-ms pulse width was performed to assess proximity to the phrenic nerve, indicated by diaphragmatic capture. After ablation, the presence of inducible VT was assessed with up to 3 extrastimuli during right ventricular pacing.

RF application was initially performed with a 4-mm electrode catheter, with power titrated to a maximum of 60°C. In all cases, however, the resulting ablation lesions were deemed inadequate, assessed by continued ability to pace and capture at the ablation site (pacing threshold <10 mA). RF ablation was then performed with an internally irrigated catheter (Chilli catheter, Boston Scientific). After cooling to 28°C to 30°C, RF power was titrated upward from 20 W to a maximum of 50 W to achieve a catheter temperature of 40°C to 45°C for 60 to 120 seconds. Repeated applications were made at the target site until the unipolar pacing threshold exceeded 10 mA with 2-ms pulse width.

After ablation, the pericardial sheath was removed, the surgical incision was closed, and a Jackson-Pratt drain was left in the pericardial space overnight and removed the next morning, if there was no active drainage. A cephalosporin antibiotic was administered as long as the drain was in place.

Results

In all 6 patients, the pericardium was accessed successfully via a small subxiphoid incision. In 2 patients, surgical exposure allowed saphenous vein grafts to the posterior descending coronary artery to be seen and avoided. In the 5 patients with prior surgery, dense adhesions were sharply and bluntly divided, after which pericardial adhesions confined movement of the catheter to the inferior and posterior region of the pericardium exposed by dissection in 2 patients. In the remaining 3 patients, the catheter could be gently advanced beyond the region of initial dissection without additional dissection. In the 1 patient who did not have prior surgery, adhesions were confined to the diaphragmatic portion of the pericardium and were the likely reason for failure of percutaneous pericardial access. Average duration of the surgical procedure required to achieve access to the epicardium was 39.7 ± 5.8 minutes (range 35 to 50 minutes).

RF Ablation

Three-dimensional electroanatomic voltage maps revealed low-amplitude regions involving the inferior or posterior left ventricular epicardium in all 6 patients (Table). In all 3 patients with incessant VT on initial arrival at the electrophysiology laboratory, VT terminated spontaneously, either after the general anesthesia (1 patient) or after a mechanical bump during blunt dissection of pericardial adhesions (2

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (y)</th>
<th>Gender</th>
<th>LVEF, %</th>
<th>Heart Disease</th>
<th>Surgery</th>
<th>Scar Location</th>
<th>No. of VTs (VT cycle length, msec)</th>
<th>No. of RF Ablations*</th>
<th>Acute Result (Yes/No)</th>
<th>Recurrence Follow-Up, d</th>
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<tbody>
<tr>
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<td>63/M</td>
<td>20</td>
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<td>None</td>
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<td>1 (450); incessant</td>
<td>9/9</td>
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<td>No/675</td>
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<tr>
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<td>CAD</td>
<td>CABG</td>
<td>Inferior</td>
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<tr>
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<td>Epicardial ICD</td>
<td>Interobasal</td>
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<td>Modified</td>
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<tr>
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<td>CAD</td>
<td>CABG</td>
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<td>18/18</td>
<td>No VT</td>
<td>No/430</td>
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</tr>
<tr>
<td>5</td>
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<td>45</td>
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<td>45</td>
<td>NICMP</td>
<td>Repair of RV perforation</td>
<td>Interobalateral</td>
<td>2 (290, 280)</td>
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<td>No VT</td>
<td>No/106</td>
<td></td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; M, male; NICMP, nonischemic cardiomyopathy; CAD, coronary artery disease; F, female; and RV, right ventricular.

*Total number of RF lesions/epicardial RF lesions.
patients), which allowed initial epicardial mapping during sinus rhythm.

In all patients, 1 or more VT circuits involving a low-amplitude region (<1.5 mV) were identified and abolished with RF ablation. In all cases, RF ablation with a standard 4-mm electrode ablation catheter generated a temperature ≥60°C at a relatively low-power application of <15 W and failed to increase the unipolar pacing
threshold at the ablation site to >10 mA. Further RF lesions were then placed with an ablation catheter cooled by internal irrigation. By a mean of 18.0±9.1 RF applications (range 9 to 31), all inducible VTs were abolished in 4 patients, and 1 or more VTs were rendered no longer inducible, although other VTs remained in 2 patients.

In 3 patients, additional endocardial left ventricular mapping was performed after initial epicardial mapping and ablation when a VT remained inducible after epicardial ablation. In 1 patient, VT was slowed from the epicardium and was abolished with additional ablation at the adjacent endocardial site, after prior failed endocardial ablation alone (patient 3). In 2 patients, a second, different VT was identified and ablated successfully from the endocardium (patients 2 and 5). During endocardial mapping and ablation, heparin was administered intravenously to maintain an activated clotting time >250 seconds, after the mapping catheter was inserted into the left ventricle, which was after the epicardial mapping and ablation had been performed. No bleeding complication was observed.

Total duration of the procedure, including the pericardial access and ablation, was 318.0±84.6 minutes (range 198 to 430 minutes). Fluoroscopy time was 48.2±24.3 minutes (range 25 to 86 minutes).

Complications
All patients had pleuritic chest pain for the initial 2 days after the procedure that was managed with acetaminophen or a nonsteroidal antiinflammatory agent. In 1 patient, chest pain persisted for 5 days. Pericardial drainage was minimal, and an echocardiogram 2 to 5 days after the procedure showed no reaccumulation of fluid. The 3 patients who received endocardial RF lesions were anticoagulated with intravenous heparin infusion initiated 6 hours after femoral sheath removal and were observed for several hours before removal of the drain. The pericardial drain was left in overnight and removed the next morning if there was no active drainage. In 1 patient, the drain was left in for 2 days because a small amount of drainage was observed. There were no other complications. Patients were discharged 2 to 11 days after the procedure.

Follow-up ranged from 106 to 675 days (Table). All 3 patients with incessant VT were free from incessant VT; 1 had a recurrence of VT terminated by an implantable cardioverter defibrillator (ICD), with no further episodes after resumption of a previously ineffective antiarrhythmic medication. In 2 patients, in whom VT was modified, previously ineffective antiarrhythmic agents were continued after the ablation. Two patients have had recurrent, infrequent VT (Table).

Discussion
This study showed that direct surgical epicardial exposure is useful for patients with epicardial VT circuits who have pericardial adhesions due to the prior cardiac surgery or epicardial ICD patches. Catheter mapping techniques and electroanatomic mapping allowed identification of low-voltage regions consistent with myocardial scar, and entrainment mapping and pace mapping were able to identify reentry circuit isthmus sites. RF catheter ablation was successful in abolishing the epicardial VT circuits that were identified.

Nonsurgical epicardial catheter ablation by a percutaneous approach was first reported by Sosa et al. 6 Most of the cases were Chagas or nonischemic cardiomyopathy, but Sosa et al subsequently also reported feasibility in patients with prior myocardial infarction. 7 Although the approach they described does not require the presence of pericardial fluid, a potential space must be present where the needle reaches the pericardium. Pericardial adhesions from prior cardiac surgery can make pericardial access difficult or impossible with this approach. Approximately 54% of patients with VT due to prior infarction seen at our institution have had prior coronary artery bypass surgery (unpublished data), and some have had epicardial ICD patch electrodes placed, as was the case in 2 patients in the present series. Pericardial adhesions are common consequences of these procedures.

We were able to gain access to the pericardial space in all of our patients. In 4 patients, adhesions outside the area of initial dissection were relatively limited. In 2, blunt dissection of adhesions was required to reach any region of the heart. Our success likely was facilitated by the fact that all of these patients had inferior wall scars that caused VT. Svenson et al 9 reported that most patients who needed epicardial or a combined epicardial and endocardial approach had inferior myocardial infarctions. Access to the anterior wall would require more extensive dissection in some patients, which might require a more extensive surgical procedure than is feasible in the electrophysiology laboratory.

Epicardial mapping and ablation can be performed in the operating room under direct surgical vision and has the advantage of allowing surgical cryoablation or laser ablation. A larger incision often is used for this purpose. 8,9 Our approach with limited exposure in the electrophysiology laboratory offers some advantages. Electroanatomic mapping systems, not generally available in the operating room, can be used to help define the abnormal regions and circuits. If desired, endocardial catheters can be used simultaneously for mapping and recording atrial His bundle electrograms and for endocardial mapping. Definition of the location of the epicardial coronary arteries relative to ablation sites is critical for safety. Although we could not directly visualize epicardial coronary arteries, coronary angiography was performed easily. It is conceivable that thoracoscopic approaches could be developed to allow direct inspection of potential ablation areas.

Conclusions
A direct surgical subxiphoid epicardial approach in the electrophysiology laboratory is feasible and can allow successful epicardial ablation for some patients with difficult pericardial access due to pericardial adhesions.

References
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