Epicardial Interventions in Electrophysiology
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The utility of the pericardial space in the electrophysiology laboratory for mapping and ablation was not appreciated by cardiac electrophysiologists until percutaneous access to the epicardial space for catheter ablation of ventricular tachycardia (VT) was first described by Sosa and colleagues in patients with Chagas cardiomyopathy in 1997.1 Long regarded as an area accessed only if procedural complications developed, the epicardial space has now become the new frontier in expanding the role of cardiac catheter ablation and therapeutics.2,3 For many years patients with arrhythmias (which could not be ablated endocardially) were treated with an open surgical approach, reminiscent of the first bypass tract surgical resection in a Wolff-Parkinson-White patient in 1969.4,5

The focus of this article is to review the development of epicardial interventions in electrophysiology in the past decade and a half and to provide an update on the current status and future prospects in this field. The anatomy of the epicardial space is discussed initially, because this provides a basis for a review of the relevant imaging aspects. The technique of accessing the space, initially developed for drainage of pericardial effusions by pericardiocentesis and subsequently modified for accessing a normal pericardial space, will also be outlined. We will also discuss mapping and ablation of arrhythmias from the epicardial surface of the heart. The majority of epicardial clinical studies have involved ventricular arrhythmias, with only small series or case reports for supraventricular arrhythmias.

The most recent European Heart Rhythm Association/Heart Rhythm Society consensus document reported that the epicardial space was accessed in 17% of VT ablation procedures, based on a survey of VT tertiary referral centers.6 A recent multicenter study from tertiary referral centers found an overall epicardial access rate of 19% for patients undergoing VT ablation procedures.7 This ranged from 6% in normal hearts, to 16% for ischemic cardiomyopathy, 35% for dilated cardiomyopathy, and 41% for arrhythmogenic right ventricular cardiomyopathy (ARVC). However, as the technique becomes more widely available, these numbers are likely to increase.

Anatomy

With the development of the percutaneous epicardial mapping and ablation approach, knowledge of the pericardial space anatomy is important for cardiac electrophysiologists using this approach for catheter ablation of arrhythmias (Figures 1 and 2, online-only Data Supplement Movies I to VIII and online-only Data Supplement Figure I).8–12

The heart is located within a double-layered membrane known as the pericardium extending from the roots of the great vessels to the central tendon of the diaphragm. It consists of an outer fibrous layer and an inner serous sac that is invaginated by the heart. The serous pericardium has a visceral layer on the epicardial surface and a parietal layer reflected on the outer fibrous layer. The thickness of the parietal pericardium varies from 0.8 to 2.5 mm. The pericardial space is a potential space between the visceral and parietal layers of the pericardium and normally contains <50 mL of serous fluid. The reflection of the visceral pericardium at the posterior surface of the heart results in an oblique sinus bounded by the inferior vena cava and the 4 pulmonary veins and situated posterior to the left atrium and anterior to the esophagus. It is the most posterior pericardial space and is inferior to the transverse sinus from which it is separated by the pericardial reflections. The transverse sinus is located posterior to the ascending aorta and the pulmonary trunk and above the left atrium. Because the pericardial reflections are located posteriorly, the anterior, apical, and lateral surfaces of the ventricles are thus freely accessible within the pericardial space. In addition the posterior and superior surfaces of the left atrium are also accessible.

Nerve Supply

The fibrous pericardium and the parietal layer of serous pericardium that lines it are supplied by the phrenic nerves (located on the anterior lateral pericardial surface) and receive vagal inputs from the esophageal plexus.13 The visceral pericardial layer on the cardiac surface is insensitive to pain. The pain of pericarditis arises in the parietal layer only and is transmitted by the phrenic nerve. The phrenic nerves course along the parietal pericardium and their anatomic location needs to be understood, because it is crucial to protect them during both endocardial and epicardial procedures.

Pericardial Anomalies

Congenital defects of the pericardium, including both partial and, rarely, total absence of the pericardium, are reported in 1:10 000 autopsies and may be associated with other cardiac

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1752
anomalies. In ≈75% of these cases the left pericardium is absent, and the heart and lungs occupy a common serous cavity; in the remainder of cases, there is a partial defect, with a foramen of variable size connecting the pericardial and pleural cavities.\(^\text{14}\)

**Pericardial Imaging**

When contrast is introduced into the pericardial space, it flows freely and outlines the cardiac borders, sinuses, and the pericardial reflections at the roots of the great vessels (Figure 3 and online-only Data Supplement Movies IV and VII). Computed tomography and magnetic resonance imaging can provide information on the pericardial space, specifically the pericardial recesses, pericardial abnormalities, and a wide variety of systemic conditions affecting the pericardial space.\(^\text{15}\) Knowledge of the pericardial recesses, which arise from the sinuses, is important for correct interpretation of pericardial imaging.\(^\text{16,17}\) The superior and inferior aortic recesses arise from the transverse sinus, the posterior cardiac recess arises from the oblique sinus, and the pulmonary vein recesses and the post caval recess arise from the main pericardial cavity.

**Accessing the Pericardial Space**

The normal pericardial space contains 15 to 50 mL of serous fluid. Although this provides for free movement between the visceral and parietal pericardial surfaces, it also provides the key to accessing the space in patients without pathological pericardial effusions. The subxiphoid approach is the best known and most frequently used for percutaneous access; other approaches such as parasternal or apical can also be used.\(^\text{9}\) Additionally, transesophageal,\(^\text{18}\) transatrial,\(^\text{19,20}\) and transbronchial\(^\text{21}\) methods to access the pericardial space have also been described.

**Subxiphoid Approach**

In the standard subxiphoid approach, the skin in the subxiphoid region is sterilized and anesthetized with lidocaine 1\%(Figures 2 through 4, online-only Data Supplement Movies I to VIII and online-only Data Supplement Figure I).\(^\text{2}\) If the procedure is performed under general anesthesia, then lidocaine 1\% may be used for local anesthesia before removal of the sheath. A 17-gauge standard Tuohy needle (marketed by multiple manufacturers, originally designed for epidural access: Havel’s Inc, OH; BD Medical, NJ) is used; either 3 ½ or 6 inches in length is commonly used (see online-only Data Supplement Figure I). With the needle directed to the left shoulder, the skin is punctured between the left border of the subxiphoid process and the left rib cage. The curved part of the needle is generally directed toward the heart. The angle between the needle and the thorax determines the surface of the ventricle accessed; with a steeper angle (with respect to the chest wall), the access tends to be more posterior, whereas a less acute angle will approach the anterior surface. As the needle is advanced, the area between the diaphragm and the chest wall is crossed, and, as the fibrous pericardium is indented, cardiac pulsation may be appreciated. Injection of small amounts of contrast will indicate the location of the needle tip (online-only Data Supplement Movie I), and, once the fibrous pericardium is tented, this can be outlined with contrast (online-only Data Supplement Movie II). Care should be taken at this step to avoid a static needle position, especially if there is downward movement of the diaphragm. If the needle is not moved backward to compensate for the extent the diaphragm moves down, the net movement of the needle would be toward the heart (hence, risking perforation of the heart). If the patient is under general anesthesia, control of respiration at the time of pericardial puncture is helpful. Puncture of the fibrous pericardium is appreciated with a release of resistance on the needle, and injection of contrast at this time will result in a contrast layer outlining the heart in the pericardial space. A guidewire is then advanced into the pericardial space, and, in the left anterior oblique view, it is observed to follow the left cardiac border and preferably cross from the left to right side in front of the great vessels, confirming the pericardial location. In our laboratory, a 4F...
sheath is then advanced over the guidewire, and 5 to 10 mL of contrast is injected into the pericardial space, which will outline the cardiac borders, sinuses, and the roots of the great vessels (online-only Data Supplement Movies II to VIII). The presence of adhesions (online-only Data Supplement Movie X) can also be appreciated at this stage (adhesions may be present even in patients who have not had a previous thoracotomy). A long exchange wire is then introduced into the space over which, in our laboratory, an 8F SL0 sheath (St. Jude Medical, Minnetonka, MN) is then advanced into the space, through which a mapping or ablation catheter can then be introduced. In the normal pericardial space, the catheter will move smoothly over the epicardial surface, allowing for easy mapping and ablation. Deflectable sheaths are also available for use in the pericardial space (Agilis EPI, St. Jude Medical, Minnetonka, MN).

**Surgical Approach**

In patients who have pericardial adhesions, most commonly after previous cardiac surgical procedures, a limited thoracotomy approach can be used to access the pericardial space. Soejima and colleagues\(^3\) first described using a subxiphoid surgical approach with manual lysis of adhesions in 6 patients. Access to the diaphragmatic surface of the heart was achieved in all patients, with catheter manipulation to the lateral and anterior wall possible in 4 patients. Our group has reported on the use of surgical access in a series of 14 patients with previously unsuccessful endocardial ablation.\(^2\) The subxiphoid approach was used in 11 patients, and 3 patients had a limited anterior thoracotomy to access the epicardium. The indications for surgical access were previous cardiac surgery (n=12), previous failed epicardial access (n=1), and need for ablation at a site in close proximity to the phrenic nerve and coronary artery (n=1). Although mapping was limited to the inferior and parts of posterior and lateral walls with the subxiphoid approach, the limited anterior thoracotomy provided access to the apex, anterior, and anterolateral walls. Surgical access with limited thoracotomy proved feasible and safe in the cardiac electrophysiology (EP) laboratory, with the surgical approach tailored to the region of the ventricle to be mapped and ablated. Subxiphoid needle puncture is possible after cardiac surgery as proposed by Sosa et al.\(^3\) It certainly requires more experience and is limited by the risk of multiple right ventricular (RV) punctures and a possible increase in the risk of RV pseudoaneurysm.

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**Figure 2.** A. The figure demonstrates a cross-section of the lower part of the thorax revealing the relationships of the pericardial cavity with the adjacent structures. In addition, the broken arrow illustrates the direction of the pericardiocentesis needle. B. Midsagittal section of a cadaver revealing the pericardial cavity, the diaphragm, and the contents of the abdominal cavity. In addition, the broken arrow shows the direction of the pericardiocentesis needle. A and B (left panel) from Loukas et al\(^9\) with permission from the publisher. Copyright © 2012, John Wiley & Sons. RCA indicates right coronary artery; AIA, anterior interventricular artery.
Epicardial VT

The stimulus to the development of epicardial intervention came from the patient population with Chagas disease and VT (Figures 5 and 6, online-only Data Supplement Movie IX and Table 1). In their center, Sosa and colleagues found that VT was reproducible with the use of programmed stimulation indicating a reentrant mechanism. Left ventricular mapping showed an inferobasal scar in 70% of patients as a likely substrate.2,39 They observed fractionated ventricular electrograms in the coronary sinus catheter in some patients; however, epicardial mapping was limited by venous anatomy. They also hypothesized that endocardial ablation might fail to create transmural lesions reaching epicardial circuits; hence, the indication to investigate the epicardial approach.25

ECG Criteria Suggesting Epicardial VT

Several groups have used their clinical studies to propose ECG criteria that would indicate an epicardial focus or circuit for VT (Figure 7). In 2004, Berruezo and colleagues reported criteria developed with endocardial and epicardial left ventricular (LV) pacing from a group of 9 patients, and further refined with 3 patient groups: a group successfully ablated epicardially after a failed endocardial approach, a second group successfully ablated endocardially, and a third group in whom endocardial ablation failed.40 A pseudodelta wave interval (measured from the onset of ventricular activation to the onset of the earliest rapid deflection in any precordial lead) of >34 ms has a sensitivity of 83% and a specificity of 95%, an intrinsicoid deflection time (measured from the onset of ventricular activation to the peak of the R wave in lead V2) of >85 ms has a sensitivity of 87% and a specificity of 90%, and an RS complex duration of >121 ms has a sensitivity of 76% and a specificity of 85% in identifying an epicardial origin of the VT. The mean QRS duration was 217±24 ms in the epicardial group versus 174±37 ms in the endocardial group, which was statistically significant, but no criterion based on QRS duration was defined; however, all patients with QRS duration >211 ms had an epicardial VT. It is noteworthy that the majority of patients in this study had ischemic cardiomyopathy (>64% for each group). However, the most recent analysis of these criteria for the ischemic substrate suggests that none of these or other criteria could
reliably predict an epicardial VT focus. Although amiodarone treatment resulted in a nonsignificant trend to QRS prolongation, no effect was seen with other antiarrhythmic drugs that were discontinued 5 half-lives before electrophysiological testing. However, in the presence of antiarrhythmic drugs, these criteria are likely even less sensitive and specific.

Bazan et al developed ECG criteria for VT arising from the epicardial RV, and the epicardial LV in the absence of...
myocardial infarction. They found these criteria were region specific, with sensitivity/specificity varying from 14% to 99% and 20% to 94%. In 2010, Valles and coworkers assessed these criteria for a population of nonischemic cardiomyopathy patients using both endocardial and epicardial pacing and clinical ventricular tachycardias. With the use of a set of endocardial and epicardial pace maps from a series of 14 nonischemic cardiomyopathy (NICM) patients, a 4-step algorithm was developed, which was then validated in a cohort of 11 patients (with 14 epicardial and 7 endocardial VTs). The following criteria were developed: (1) absence of inferior Q waves, (2) pseudodelta greater than or equal to 75 ms, (3) maximum deflection index (defined as the ratio of the interval from the onset of ventricular activation to the peak of the QRS in a precordial lead to the QRS duration) of >0.59, and (4) the presence of a Q wave in lead I. When these criteria were applied in a 4-step algorithm, they yielded a sensitivity of 96% and a specificity of 93%. It is noteworthy that most of the VTs in NICM appear to originate from areas of substrate scar in the superior and lateral areas near the mitral valve. For idiopathic and nonischemic substrates compared to the ischemic substrate.

Chagas VT

In their initial series of patients with VT and Chagas disease, Sosa et al found an epicardial circuit for 14 of 18 mappable VTs induced in 10 patients. In 10 of these 14 VTs, the earliest site was epicardial and radiofrequency (RF) delivery at these sites resulted in VT termination in 4.8 ± 2.9 seconds. None of the VTs were reinducible in the 6 patients who underwent epicardial ablation. Most of the VTs (82%) are related to a left-sided inferobasal scar, and a common reentrant circuit involved the surviving tissue between the scar and the mitral isthmus.

In a more recent report from this laboratory on 257 consecutive patients referred for VT ablation, the prevalence of epicardial VT was higher in patients with Chagas disease (37%) than in patients with postmyocardial infarction (28%) and idiopathic dilated cardiomyopathy (24%).

VT in Normal Hearts

In the study of Schweikert et al, 20 of the 30 patients with VT who underwent epicardial mapping had normal hearts. Of these patients, 7 were ablated from the aortic root, 9 were ablated epicardially, 2 were ablated endocardially, and 2 could not be ablated. For the 9 patients ablated epicardially, there were a total of 17 VTs with mean epicardial activation occurring 36 ± 5 ms pre-QRS, requiring a mean of 3 ± 1 epicardial lesions. Of particular note, for the 17 VTs that were
successfully ablated epicardially, successful sites were clustered at the atroventricular or interventricular grooves, along the course of major epicardial vessels. Daniels and coworkers reported that an epicardial LV site of origin was found in 12 of 138 (9%) of patients referred for ablation of idiopathic VT; patients with Sinus of Valsalva VT were not included in the analysis. Eleven patients were successfully ablated, 5 via the coronary venous system, 4 with the percutaneous epicardial approach, and 2 with a surgical approach. It is noteworthy that the sites of successful ablation

### Table 1. Epicardial VT Ablation Studies

<table>
<thead>
<tr>
<th>Author and Ref. No.</th>
<th>Year</th>
<th>N</th>
<th>Substrate (n)</th>
<th>Epicardial Ablation (n)</th>
<th>Ablation Catheters</th>
<th>Ablation Approach</th>
<th>Acute Success</th>
<th>Follow-Up, mo</th>
<th>Intermediate Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sosa25</td>
<td>1998</td>
<td>10</td>
<td>Chagas CM</td>
<td>6</td>
<td>4-mm tip with temp to 60°</td>
<td>Pace mapping</td>
<td>100%</td>
<td>4–9</td>
<td>100%</td>
</tr>
<tr>
<td>Sosa26</td>
<td>2000</td>
<td>14</td>
<td>ICM (14)</td>
<td>7</td>
<td>NR</td>
<td>Thermal mapping</td>
<td>56%</td>
<td>14±2</td>
<td>37%</td>
</tr>
<tr>
<td>Schweikert37</td>
<td>2003</td>
<td>30</td>
<td>Normal heart (20) ICM (7) NICM (3)</td>
<td>8</td>
<td>Cooled tip (Chilli or Thermocool) to 50°C mean 3+1 lesions</td>
<td>Activation mapping</td>
<td>93%</td>
<td>26±13</td>
<td>58%</td>
</tr>
<tr>
<td>Sarabanda28</td>
<td>2005</td>
<td>56</td>
<td>Chagas CM (56)</td>
<td>56</td>
<td>8-mm tip to 70°C</td>
<td>Ent or pace mapping</td>
<td>30%</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Cesario29</td>
<td>2006</td>
<td>20</td>
<td>ICM (12) NICM (8)</td>
<td>6</td>
<td>8-mm Navistar; 40–70 W to 55°</td>
<td>Pace mapping</td>
<td>100%</td>
<td>12±4</td>
<td>75%</td>
</tr>
<tr>
<td>Daniels30</td>
<td>2006</td>
<td>12</td>
<td>Normal</td>
<td>12</td>
<td>4-mm tip, 4-mm irrigated, cryocatheter</td>
<td>Act., Pace mapping</td>
<td>75%</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Aryana31 (magnetic navigation)</td>
<td>2007</td>
<td>24</td>
<td>ICM (13) NICM HCM ARVC Sarcoidosis</td>
<td>3</td>
<td>4-mm tip (n=2); 4-mm irrigated (n=22)</td>
<td>Act., Ent. mapping</td>
<td>97%</td>
<td>7±3</td>
<td>83%</td>
</tr>
<tr>
<td>Garcia32</td>
<td>2009</td>
<td>13</td>
<td>ARVC (13)</td>
<td>13</td>
<td>4-mm tip RF 50 W to 55°C; Closed irrig. or open irrig. to 42°; both up to 50 W</td>
<td>Act. or pace mapping; focal or linear lesions to LPs</td>
<td>92%</td>
<td>18±13</td>
<td>77%</td>
</tr>
<tr>
<td>Schmidt33</td>
<td>2010</td>
<td>56</td>
<td>Normal (18) ICM (11) NICM (16) ARVC (13) Myocarditis (5)</td>
<td>12</td>
<td>Irrigated RF to 43°C and power to 50 W Median 1–12 lesions</td>
<td>Act. and Ent. mapping</td>
<td>69%</td>
<td>12±5</td>
<td>47%</td>
</tr>
<tr>
<td>Sacher7 (multicenter)</td>
<td>2010</td>
<td>134</td>
<td>ICM (51) NICM (39) ARVC (14) Other CM (13) Normal (17)</td>
<td>51</td>
<td>4-mm, 8-mm solid tip Open and closed cooled tip Cryocatheters power 20–50 W</td>
<td>Multiple</td>
<td>NR</td>
<td>23±21</td>
<td>71%</td>
</tr>
<tr>
<td>Nakahara34</td>
<td>2010</td>
<td>33</td>
<td>ICM (17) NICM (16)</td>
<td>7</td>
<td>Open irrig. (Thermocool) 50 W Closed irrig. (Chilli) 8-mm tip (Navistar)</td>
<td>LP and fract. EGMs</td>
<td>82% (ICM)</td>
<td>12±10</td>
<td>82%</td>
</tr>
<tr>
<td>Nadamanee35</td>
<td>2011</td>
<td>9</td>
<td>Brugada (9)</td>
<td>9</td>
<td>3.5-mm Thermocool 30–50 W to 45°, median 35 lesions</td>
<td>LPs and fract. EGMs</td>
<td>78%</td>
<td>20±6</td>
<td>78%</td>
</tr>
<tr>
<td>Bai36 (multicenter)</td>
<td>2011</td>
<td>49</td>
<td>AVRC (49)</td>
<td>26</td>
<td>3.5-mm Thermocool</td>
<td>Act. or Ent. mapping LPs and fract. EGMs</td>
<td>85% (endo. ABl. only 52%)</td>
<td>39±4</td>
<td>85%</td>
</tr>
<tr>
<td>Della Bella37 (multicenter)</td>
<td>2011</td>
<td>218</td>
<td>ICM (85) NICM (67) ARVC (13) HCM (5) Normal (48)</td>
<td>218</td>
<td>Open irrig. Cath. (80% of cases) 20–40 W, irrig. 25 mL/min; power titrated Temp. &lt;45°C or impedance drop &lt;20 ohms. Nonirrigated RF catheters (12%); cryobal. (8%)</td>
<td>Pace or Ent. mapping</td>
<td>72%</td>
<td>17±18</td>
<td>68%</td>
</tr>
<tr>
<td>Dello Russo38</td>
<td>2012</td>
<td>20</td>
<td>Myocarditis</td>
<td>6</td>
<td>Cooled tip with 40 W to 43°</td>
<td>Act., Ent. mapping</td>
<td>100%</td>
<td>Median 28</td>
<td>90%</td>
</tr>
</tbody>
</table>

NR indicates not reported; Intermediate Success, success rate after the defined follow-up period; Act., activation; Cont., continuous; Dias., diastolic; Fract., fractionated; Ent., entrainment; Irrig., irrigation; EGM, electrograms; LP, late potentials; ICM, ischemic cardiomyopathy; NICM, nonischemic cardiomyopathy; ARVC, arrhythmogenic right ventricular cardiomyopathy; RF, radiofrequency; and CM, cardiomyopathy.
were also clustered around the course of the middle cardiac
vein and the anterior interventricular vein, similar to the
findings of Schweikert and colleagues.

Ischemic VT
In the initial report of epicardial catheter ablation in patients
with postmyocardial infarction scar–related VT, Sosa and
colleagues reported their findings in a series of 14 patients.
Epicardial access was obtained in all patients. A total of 30
VTs were induced, of which 18 were mappable and, of these,
7 were interrupted with epicardial ablation (39% of mappable
VTs). In epicardial mapping, the earliest sites were 87
ms before the QRS onset. The overall success rate of
combined endocardial and epicardial ablation in this study
was 37% with a follow-up of 14 ± 2 months.

Our group reported an initial experience in a combined
population of ischemic (n = 12) and nonischemic (n = 8) car-
diomyopathy patients referred for VT ablation to a tertiary
referral center. All patients underwent both endocar-
dial and epicardial mapping; high-density voltage maps of the
substrate were performed and showed that the patients with
ischemic cardiomyopathy tend to have a 2- to 3-fold larger
scar area endocardially than epicardially. Most VTs (89%)
were hemodynamically unstable, and a substrate
modification-based approach was used for ablation. For the
total population, 75% of patients were free of either shocks or
ATP at 1 year (online-only Data Supplement Video IX). Combined epicardial and endocardial mapping allows a
comprehensive delineation of the substrate and allows abla-
tion of later potentials in sinus rhythm. In a recent
study, we found that patients with ischemic cardiomyopathy
tend to have a better outcome following catheter ablation for
VT (82% nonrecurrence at 12 ± 10 months of follow-up)
compared to NICM.

Schmidt and colleagues from Hamburg recently reported
on 59 patients with multiple different substrates who under-
went combined endocardial and epicardial mapping, follow-
ing previous unsuccessful endocardial ablation. Eleven pa-
tients had an ischemic substrate with a mean of 1.6 inducible
VTs, and epicardial ablation was performed in 7 patients.
Using both activation mapping for tolerated VTs and a
substrate-guided approach for unmappable VT, complete
success, defined as no inducible VT, was obtained in 55% of
the ischemic patients. Partial success, defined as noninduc-
ability of the clinical VT, was obtained in the remainder of
the patients. The site of successful ablation was endocardial in
4, epicardial in 4, and both endocardial and epicardial in 3
patients.

Nonischemic VT
Hsia and colleagues first reported that epicardial mapping in
patients with NICM demonstrated abnormal electrograms
(amplitude <1.8 mV) in 20 ± 12% of the total endocardial
surface, usually located near the ventricular base in the
perivalvular region, with a comparable area (25%) of
endocardial low voltage. We also found that patients with
NICM had similarly sized scar areas (voltage <0.5 mV) on
the epicardial and endocardial surfaces, in contrast to ische-
mic cardiomyopathy substrates where the endocardial scar
area was nearly 3 times larger than the epicardial scar area.
Soejima et al reported on a series of 28 patients with VT and
a NICM substrate. Of these, 26 patients underwent endocar-
dial and 8 underwent epicardial electroanatomic mapping. Of
the 19 VT circuit isthmuses identified by entrainment or pace
mapping, 12 were associated with endocardial scar and 7
were associated with an epicardial scar. Epicardial scar areas
were also larger than endocardial scar areas in these patients.
Ablation with an internally irrigated 4-mm electrode success-
fully ablated 6 VTs with an average of 10 RF applications, 35
Watts to 41°C.

In a recent study from the University of Pennsylvania,
Cano et al reported on 22 patients with VT and NICM who

![Flow chart for suggested approach to assessing need for epicardial access and/or ablation. VT indicates ventricular tachycardia; CE, contrast enhanced; Pseudodelta, interval from the onset of ventricular activation to the onset of the earliest rapid deflection in any epicardial lead; IDT, intrinsicoid deflection time, the interval from the onset of ventricular activation to the peak of the QRS in a preordial lead to the QRS duration; MDI, maximal deflection index defined as the ratio of the interval from the onset of ventricular activation to the peak of the QRS in a preordial lead to the QRS duration; ICM, ischemic cardiomyopathy; NICM, nonischemic cardiomyopathy; ARVC, arrhythmogenic right ventricular cardiomyopathy; and CM cardiomyopathy.](image-url)
underwent combined endocardial and epicardial mapping for suspected epicardial VT. Epicardial VTs were targeted in 18 patients on the basis of activation/pacemapping or by targeting split or late potentials. During an average of 18 months of follow-up, there was no recurrence of epicardial VTs in 14 of 18 patients (78%). The mean epicardial area of low voltage (<1.0 mV) was $55.3\pm33.5\,\text{cm}^2$ versus $22.9\pm32.4\,\text{cm}^2$ for the endocardial surface. The electrophysiological substrate in NICM which is characterized by a lower prevalence of late potentials and the success rate for ablation was lower compared to ischemic cardiomyopathy (50% nonrecurrence at 12±10 month of follow-up). 34

Myocarditis
Ventricular arrhythmias may be the initial presentation in myocarditis. 35,36 In the largest series to date, Dello Russo and coworkers recently reported a multicenter study of 20 patients with biopsy-proven myocarditis and drug refractory VT, of which 5 patients presented with VT storm. 38 The mean ejection fraction (EF) was 55%, and all patients underwent endocardial RF mapping and ablation with irrigated catheters. This was successful in 14 patients, whereas the remaining 6 (30%) were successfully ablated epicardially. Subsequently, 2 patients died of acute heart failure unrelated to the VT. Contrast-enhanced cardiac magnetic resonance may be particularly useful for identifying scar location in patients with acute myocarditis. In an magnetic resonance study of patients with myocarditis presenting with heart failure or ventricular arrhythmias, De Cobelli et al 35 found that late enhancement with gadolinium was present in 84% of patients, equally distributed between midwall and subepicardial distributions.

Sarcoidosis/Granulomatous Diseases
In granulomatous cardiac disease, ventricular arrhythmias may also be the first clinical presentation. 38 Koplan et al described a series of 8 consecutive patients with monomorphic VT due to cardiac sarcoidosis, with VT the initial manifestation in 5 of 8 cases. An average of 4±2 VTs were induced in each patient, with areas of low voltage in the RV in all patients, in the LV for 5, and in the epicardium in 2 patients who underwent epicardial mapping. Postablation, all but 1 patient remained inducible for some VT; however, with at least 6 months of follow-up, 4 patients were controlled with immunosuppression and antiarrhythmic drugs. Four patients eventually required cardiac transplant because of recurrent VT. Jefic and colleagues 38 followed a multicenter registry of 42 patients with cardiac sarcoidosis who developed VT. In 9 patients, VT was not controlled with medical therapy (steroids and antiarrhythmic drugs), and RF ablation was performed for this group. A total of 44 VTs were induced in these 9 patients, and the most frequent VT circuit was reentry in the pericricuspid area. One patient required epicardial ablation of a lateral LV site and had no recurrence. In total, 5 of the 9 patients who underwent ablation had no recurrence at 20 months of follow-up.

Arrhythmogenic Right Ventricular Cardiomyopathy
In the first complete description of arrhythmogenic right ventricular cardiomyopathy (ARVC) in 1982, a triangle of dysplasia involving the inferior right ventricle (RV), the RV apex, and the RV outflow tract was defined. 39 Within a year, Guiraudon et al 40 reported a surgical treatment of VTs in these patients by total disconnection of the RV free wall. Marchlinski and colleagues 41 confirmed that patients with ARVC have a predilection for scar in the basal periventricular, apical, and outflow tract areas with the use of epicardial mapping. In a study of 13 patients who underwent endocardial and epicardial mapping after failed endocardial ablation, this group reported that areas of low voltage (<1 mV for epicardium and 1.5 mV for endocardium) were more extensive on the epicardium and demonstrated multicomponent and late potentials. 42 Twenty-seven VTs were targeted on the epicardium, which were opposite ineffective endocardial ablation sites in 11 patients. The approach combined activation, entrainment, and pace mapping with focal or linear lesions targeting late potentials. During an average 18 months of follow-up, 10 of 13 patients (77%) had no VT. In a recent multicenter study, Bai et al compared 2 groups of ARVC patients with VT who underwent endocardial-only ablation (n=23) or endo-epicardial ablation (n=26). The ablation strategy was to target all sites with fractionated or late potentials seen on electroanatomical mapping. After at least 3 years of follow-up, freedom from ventricular arrhythmias or implantable cardioverter defibrillator therapy was 52.2% (12/23) in the endocardial-only ablation group and 84.6% (22/26) in the combined endo-epicardial ablation group. The authors concluded that a combined endo-epicardial ablation strategy was superior for VT ablation in arrhythmogenic RV dysplasia/cardiomypathy patients. More recently, Berruezo et al 42 reported similar results with a combined epicardial-endocardial approach in 11 patients with ARVC, with a recurrence rate of 9% at 11 months of follow-up.

Brugada Syndrome
Epicardial mapping studies by Nagase and colleagues 43 showed that the negative T wave associated with type I Brugada ECG pattern was associated with a prolongation of the action potential in the epicardium in comparison with the endocardium in the region of the RV outflow tract. Nademanee and coworkers 43 recently reported a new application of epicardial ablation to patients with type I Brugada syndrome and recurrent episodes of ventricular fibrillation. Their study reported on 9 patients with a median 4 episodes of VT per month requiring implantable cardioverter defibrillator shocks. Combined endocardial and epicardial mapping of the RV revealed areas of abnormal low voltage (0.94±0.79 mV) with prolonged duration and fractionated electrograms localized to the anterior aspect of the RV outflow tract epicardium. Ablation of these sites rendered VT/ventricular fibrillation noninducible in 7/9 patients (78%), with no clinical recurrences in these patients during a mean follow-up of 20 months. Additionally, following ablation, the ECG pattern normalized in 8/9 patients (89%). This study supports the hypothesis that abnormal delayed depolarization is the mechanism of VT/ventricular fibrillation in Brugada patients with an arrhythmogenic substrate localized to the RV outflow tract epicardial region.
Current State of the Art: European Multicenter Study 2011
Della Bella and colleagues\textsuperscript{37} reported outcomes from 6 tertiary VT referral centers (with an experience of at least 8 epicardial cases) in Europe in 2011. The analysis included 218 patients who underwent epicardial mapping (87% had concurrent endocardial mapping). Catheter ablation was attempted for 289 induced VTs with a strategy based on pace or entrainment mapping and targeting presystolic or middiastolic potentials. RF energy was delivered on the epicardial surface only in 103 VTs (35.6%), on the endocardial surface only in 41 VTs (14.2%), and both endocardially and epicardially for 145 VTs (50.2%). After a mean follow-up of 17 months, the overall recurrence rate was 31.4%; this was not significantly different for the various cardiac substrates: NICM (39.3%), ischemic cardiomyopathy (34.7%), ARVC (30.8%), hypertrophic cardiomyopathy (25%), and idiopathic VT (17.1%). Twenty patients (10.4%) died during the follow-up period (12 of heart failure, 2 of cardiac arrest, and 6 of noncardiac causes), whereas 3 patients underwent heart transplantation.

Epicardial Ablation Using Magnetic Navigation
Aryana et al\textsuperscript{31} reported on the use of remote magnetic navigation to guide endocardial and epicardial mapping and ablation for scar-related VT. In 15 of 24 patients, the substrate was ischemic cardiomyopathy, with substrates of NICM, hypertrophic cardiomyopathy, ARVC, and sarcoidosis in the remainder. Electroanatomical maps using the Stereotaxis system were constructed for the LV, RV, and epicardial surface in 24, 10, and 12 patients, respectively. A total of 77 VTs were targeted, with a manually irrigated catheter used for ablation in 22 procedures; 75 of 77 (97%) of VTs were eliminated. For 21 hemodynamically stable VTs targeted for ablation during VT in 15 patients, 17 (81%) were successfully terminated; for these 15 patients, the successful sites were LV (for ischemic cardiomyopathy and NICM), RV for the sarcoidosis patient, and epicardial RV for the 3 ARVC patients. Four patients underwent a second procedure, 3 with magnetic navigation; there were no VT recurrences during a mean of 7 ± 3 months of follow-up.

Surgical Approach to Epicardial VT Ablation
First developed in the 1970s with the subendocardial excision method for recurrent VT,\textsuperscript{64} but largely abandoned by the 1990s because of high procedural mortality, surgically guided ablation has recently made a comeback with the development of the percutaneous epicardial approach, electroanatomic mapping techniques, and advances in catheter technology. In our series of 14 patients who underwent a surgical approach for epicardial access, 10 had an ischemic cardiomyopathy substrate.\textsuperscript{23} Of these 10, 8 underwent epicardial ablation with the use of a closed irrigation catheter with power settings from 20 to 50 W, titrating for a target temperature of 42°C and monitoring for an impedance drop. We have also reported on a patient with ischemic cardiomyopathy and an LV assist device, who underwent anterior thoracotomy for recurrent VT after unsuccessful endocardial ablation.\textsuperscript{65} The epicardial access allowed ablation by using a surgical ablation tool (Isolator Coolrail, AtriCure Inc) composed of two 30-mm electrodes, with bipolar RF delivered between the 2 electrodes and internal irrigation. This allowed delivery of RF to a broad region, with lesion depth to 4 to 5 mm, and successful ablation of the clinical VT.

Anter at al\textsuperscript{66} recently reported a series of 8 patients with nonschemic cardiomyopathy and refractory VT despite previous endocardial (n = 8) and epicardial (n = 6) ablation. After an unsuccessful percutaneous approach, median sternotomy was performed and surgical cryoablation was delivered directly at sites previously identified by electroanatomic mapping and pacing techniques. Two patients died in the postprocedure hospitalization period because of heart failure and sepsis, while, in the remaining 6 patients, the median time from surgery to discharge was 7 days. Over a mean follow-up of 23 ± 6 months, 4 patients remained free of VT and the number of implantable cardioverter defibrillator shocks per patient declined from 6.6 in the 3 months before surgery to 0.6 in the 3 months after surgery. Surgical ablation tools have also been used in this setting in the EP laboratory for creating ablative lesions.\textsuperscript{65}

The surgical option offers several advantages when dealing with epicardial or midmyocardial circuits that cannot be reached with endocardial or epicardial catheter ablation.\textsuperscript{67} Surgical cryoablation delivers temperatures to \(-160°C\), achieving deeper lesions than catheter cryoablation, whereas direct visualization of the myocardium allows avoidance or mobilization of the coronary arteries and dissection of epicardial fat. As the authors point out, these patients comprise \(\approx5\)% of the patients referred for VT ablation at the severe end of the spectrum, and perioperative mortality remains a concern. Nevertheless, this is now a viable option for select patients, with an experienced cardiac EP and cardiac surgical team.

When to Perform Epicardial Access and Ablation
Multiple factors need to be considered when deciding whether an epicardial approach is needed.\textsuperscript{68} A suggested approach is outlined in Figure 7. This includes ECG analysis of the clinical VT for features suggesting epicardial source, history of a previous failed endocardial ablation, contrast-enhanced computed tomography or magnetic resonance imaging suggesting a subepicardial or midmyocardial scar and an estimation of the likelihood of epicardial VT given the cardiac substrate. It is important to make this decision before starting the case, because planning and logistics are very important in an epicardial access case. In our laboratory, we obtain epicardial access immediately after placing the standard intracardiac catheters, His bundle, RV, coronary sinus, and before accessing the LV via the transseptal route. After epicardial access is obtained, and there is no evidence of epicardial bleeding or complications, the patient is fully heparinized and endocardial LV access is obtained by the transseptal approach (our preferred approach to avoid aortic transit of catheters). We then proceed with epicardial and endocardial voltage mapping.

Epicardial Supraventricular Tachycardias
The epicardial approach is less commonly used for supraventricular tachycardia ablation than for VT ablation, and almost
always used after a patient has already undergone an unsuccessful endocardial ablation (Table 2). Supraventricular tachycardias successfully treated with this approach include accessory pathways, atrial tachycardia, and atrial fibrillation.

### Accessory Pathways

The first reported ablation of an accessory pathway was the use of the open surgical approach in 1969. The development of endocardial RF catheter ablation, most bypass tracts became accessible to catheter ablation (Figure 8 and online-only Data Supplement Video VIII). Although the coronary sinus, or a coronary sinus diverticulum, may provide a route to locate and ablate epicardial pathways, there are some pathways that cannot be reached via the coronary sinus or its branches. Reported cases indicate a wide variety of pathway locations including left posterolateral and posteroseptal, midseptal, right-sided, and right atrial appendage to RV. In some cases, a combined epicardial and endocardial approach has been successfully used for resistant pathways when endocardial or epicardial approaches alone were unsuccessful.  

Epicardial pathway location has been cited as a reason for failed endocardial ablation in 8% of cases. Schweikert and coworkers reported on a series of 10 patients with accessory pathways and previously failed endocardial ablations. The group included both manifest and concealed pathways, with 4 left-sided, 1 midseptal, and 5 right-sided pathways. In 5 cases, the earliest activation was recorded epicardially; 3 were right atrial appendage to RV and were successfully ablated epicardially, whereas a right posteroseptal and a left posteroseptal pathway were successfully ablated endocardially despite an earlier epicardial site. In the case of the left posteroseptal pathway, there was high impedance with epicardial ablation, possibly because of epicardial fat. During a 2-year follow-up, 1 patient had a recurrence and was successfully reablated. There were no procedure-related complications, and there was no difficulty in reaccessing the pericardial space.

Our group has reported on a series of 6 patients with previously unsuccessful endocardial ablation of accessory pathways referred for epicardial ablation. Six patients with 7 accessory pathways were studied; 6 of the pathways were right sided, and of these, 5 were located in the right free wall. All

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**Table 2. Epicardial SVT Ablation Studies**

<table>
<thead>
<tr>
<th>Author and Ref. No.</th>
<th>Year</th>
<th>N</th>
<th>Substrate/Epicardial Access (n)</th>
<th>Epicardial Ablation Performed (n)</th>
<th>Catheter</th>
<th>Ablation Target</th>
<th>Follow-Up, mo</th>
<th>Intermediate Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>Katritsis69</td>
<td>2001</td>
<td>40</td>
<td>Paroxysmal AF (14)</td>
<td>2</td>
<td>25 W to 50°C</td>
<td>Activation mapping</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Schweikert27</td>
<td>2003</td>
<td>18</td>
<td>Pathway (10)</td>
<td>3</td>
<td>Irrigated tip to 50°C</td>
<td>Activation mapping</td>
<td>26±13</td>
<td>66%</td>
</tr>
<tr>
<td>Valderrabano70</td>
<td>2004</td>
<td>6</td>
<td>Pathway (6)</td>
<td>2</td>
<td>50 W at 60–70°C</td>
<td>Activation mapping</td>
<td>4</td>
<td>84%</td>
</tr>
<tr>
<td>Scanavacca71</td>
<td>2006</td>
<td>10</td>
<td>AF (7)</td>
<td>7</td>
<td>30–50 W to 60°C</td>
<td>Evoked Vagal reflex mapping</td>
<td>8</td>
<td>30%</td>
</tr>
<tr>
<td>Pak72</td>
<td>2007</td>
<td>5</td>
<td>AF (5)</td>
<td>5</td>
<td>Open irrigated; 35 W to 50°C</td>
<td>PV isolation</td>
<td>8±6</td>
<td>100%</td>
</tr>
<tr>
<td>Phillips73</td>
<td>2008</td>
<td>2</td>
<td>LAA AT (2)</td>
<td>2</td>
<td>4 mm; 50 W to 50°C</td>
<td>Activation mapping</td>
<td>NR</td>
<td>NR</td>
</tr>
</tbody>
</table>

NR indicates not reported; Intermediate Success, success rate after the defined follow-up period; AF, atrial fibrillation; AFL, atrial flutter; AT, atrial tachycardia; IST, inappropriate sinus tachycardia; LAA, left atrial appendage; and PV, pulmonary vein.
patients also underwent simultaneous endocardial mapping. The successful ablation site was endocardial in 1 patient, epicardial in 4, and simultaneous epicardial and endocardial in 1 patient.

In a series of 89 cases of Wolff-Parkinson-White referred to tertiary centers after initial unsuccessful ablation attempts, the most common reasons for failure were catheter manipulation problems and inadequate mapping. An irrigated tip catheter was required for epicardial pathways (n=7), of which 6 were ablated from the coronary sinus and 1 via percutaneous epicardial access.

**Atrial Tachycardias**

Successful epicardial ablation has also been reported for atrial tachycardias, specifically inappropriate sinus tachycardia and atrial tachycardias, particularly those arising from the left atrial appendage. Schweikert et al reported 4 cases in their series of supraventricular tachycardia cases; one of the two with an earliest epicardial site was ablated endocardially. Although this arrhythmia did not recur, overall there was a 50% recurrence rate for inappropriate sinus tachycardia. A combined epicardial-endocardial approach was used for another reported case of inappropriate sinus tachycardia. Phillips et al reported 2 cases of atrial tachycardia arising from the left atrial appendage that were successfully ablated epicardially following a failed endocardial approach. The complex anatomy of the atrial appendage, which is also more prone to perforation from mechanical pressure, could make the epicardial route the preferred approach for atrial appendage tachycardias.

**Atrial Fibrillation**

There is very limited data on ablation of atrial fibrillation (AF) using an epicardial approach (Figure 9). The reflections of the visceral pericardium prevent complete encircling lesions of the pulmonary veins being achieved by this approach. The vein of Marshall, in some cases, may be a trigger site for induction of AF and can be approached both endocardially via the coronary sinus or epicardially. Hwang and coworkers reported on a series of 28 patients undergoing AF ablation; in 6 patients, direct vein of Marshall recording documented the origin of the AF in the muscle bundle within the ligament of Marshall. RF ablation at the insertion site successfully terminated AF in 4 of these 6 patients. In a study of 40 patients undergoing catheter ablation for paroxysmal AF, catheterization of the vein of Marshall via the coronary sinus was feasible in 14 of 19 patients with foci in the left superior or inferior pulmonary veins. In 2 patients, the sole focus was epicardial and was ablated epicardially via the coronary sinus.

Pak and colleagues reported a series of 5 patients who relapsed after endocardial AF ablation; additional epicardial ablations at pulmonary vein adjacent sites resulted in successful pulmonary isolation. In a recent report on epicardial mapping in patients undergoing a surgical Maze procedure within 8+11 months of an endocardial ablation, 15 of 24 (62.5%) of the pulmonary veins examined showed conduction on the epicardial regions. Choi et al reported a hybrid endocardial-epicardial procedure in a patient with an anterior atrial wall mass and atrial fibrillation guided by computed tomography merged with 3-dimensional electroanatomical mapping. In a recent case of persistent AF, a combined endocardial-epicardial approach was used to create a box set of lesions on the posterior atrial wall isolating all 4 PVs and the posterior wall together.

Scanavacca and coworkers examined a strategy of targeting atrial sites in which high-frequency stimulation (20 Hz) induced vagal reflexes (atrioventricular [AV] block >2 seconds) in 10 patients with paroxysmal AF and no heart disease. Endocardial and/or epicardial RF ablation was performed at these sites in 7 patients, whereas pulmonary vein isolation was performed in 3 patients in whom evoked vagal reflexes were not obtained. AF recurred at 8 months in 5 of the 7 patients (71%) who underwent the vagal selective guided ablation and none of the pulmonary vein isolation patients. Given the small number of patients, further studies are needed to define if this approach has a role in AF ablation procedures.

Although the epicardial approach is appealing for ablation of AF, potentially avoiding the complications of embolic events and tamponade, these advantages must be balanced against the increased risk of damage to the coronary arteries, phrenic nerves, and esophagus. Approaches adopted to protect the phrenic nerves and the esophagus include the use of balloons, saline, and air in the pericardial space. As with ablation of epicardial ventricular circuits, the presence and extent of epicardial fat may prevent effective ablation of epicardial pathways.

**Collateral Injury and Complications**

As would be expected, entering, mapping, and ablation in the pericardial space are associated with multiple potential complications (Figure 10). When the pericardial space is ac-
cessed, the liver, colon, and diaphragm with its vascular supply may be injured. If the needle is advanced too far or too fast, it may enter the RV, the pleural space, or the lung. Use of the curved-tip Tuohy needle will help decrease the risk of entering the RV and direct the guidewire when the epicardial space is entered (Figure 2). Knowledge of the anatomy of the subxiphoid space and the pericardial space and an awareness of potential complications are essential.

In their initial description of the epicardial access technique in a series of 3 patients, Sosa and colleagues reported no complications and specifically noted that echocardiograms 1 day after the procedure showed no pericardial effusion. In a recent summary of their group experience, the most common complications were related to puncture accidents.86 A dry RV puncture occurred in 4.5% of 215 consecutive cases, and a hemopericardium requiring drainage was seen in 7% of cases (200±100 mL blood). Avoiding coronary artery damage is a major concern while ablating in the epicardial space. A coronary angiogram will show the location of the coronary arteries with respect to proposed ablation site. RF energy is not delivered unless the distance between the catheter tip and a visible coronary artery is at least 1 cm, which is based primarily on operator experience2 and on limited studies.87 In only 1 of their 215 consecutive patients did RF delivery result in occlusion of a coronary marginal branch, and a peak creatine kinase of 35 U/L; however, more data are needed to further clarify this important concern. By using a high-output pacing (15 mA, 5-ms pulse) to test at ablation sites possibly close to the phrenic nerve, they had no cases of phrenic nerve injury. Epicardial fat with thickness of >5 mm may insulate the myocardium and attenuate RF lesion formation, even with cooled tip catheters. Pericarditis without pericardial effusion was seen in 30% of the patients postprocedure, which responded to anti-inflammatory drugs. In a group of 29 patients who had a repeat epicardial access, no adhesions were found.

Sacher and colleagues reported a multicenter safety study from 3 tertiary centers performing epicardial VT ablation in 2010. VT ablation required epicardial access in 121 (13%) of 913 procedures. There were 8 (5%) acute major complications related to epicardial access: 7 epicardial bleeding and 1 coronary stenosis. Three delayed complications occurred after 48 hours: 1 major pericardial inflammation, 1 tamponade (10 days), and 1 acute inferior myocardial infarction (2 weeks). No constrictive pericarditis or phrenic nerve injuries were reported. There were 6 complications related to concomitant endocardial ablation: pulmonary embolism (2), pericardial effusion (1), infranodal AV block (1), and groin hematomas requiring blood transfusion (1).

In the European multicenter study reported by Della Bella et al in 2011,37 major complications were observed in 9 (4.1%) of the patients (cardiac tamponade in 8 and abdominal hemorrhage in 1). Complications classified as minor were observed in 17 (7.8%) of the patients, including vascular injury, heart failure, intermittent atrioventricular block, and pneumonia. Ten percent of patients experienced severe postprocedural pericardial pain.

A recent report highlights a series of unusual complications associated with percutaneous epicardial access in a multicenter series of 334 patients in a 5-year period.88 These included subcapsular hepatic hematoma, coronary spasm, and RV pseudo aneurysm, which were all managed conservatively. Surgical intervention was required for cases of liver puncture with intra-abdominal bleeding, pericardial bleeding due to middle cardiac vein laceration, pericardial bleeding due to coronary sinus laceration, and RV abdominal fistula. Other uncommon complications such as a pleuropericardial fistula may occur and should be considered if a new left pleural effusion occurs after the procedure.89

Recently, it has been shown that repeat pericardial access can be performed safely at a median of 110 days after the initial procedure in a series of 30 patients.90 Adhesions were encountered in 7 patients and were easily divided with blunt dissection by a deflectable catheter in 5 of the 7 patients. A 90% acute success rate for the targeted VT was achieved in this study. However, the same center has also recently reported a case of constrictive pericarditis after a fourth
epicardial ablation procedure, resulting in severe heart failure, requiring a pericardiotomy.  

**Methods to Reduce Complications**

Several approaches have been developed to minimize or prevent these epicardial ablation–related complications. As symptoms of pericarditis can occur in up to 30% of patients; nonsteroidal anti-inflammatory medications are usually given postprocedure. Additionally, injection of glucocorticoid into the pericardial space also reduces inflammation in an animal model, and is now routinely used in our center, where we give methylprednisolone 250 mg intrapericardially at the end of the procedure.

Multiple techniques have also been developed for phrenic nerve protection. Our group has adapted a Meditech balloon (Boston Scientific) to displace the phrenic nerve and protect it during RF ablation on the lateral epicardial wall. Figure 10 and online-only Data Supplement Videos XI and XII). Fan et al. reported that, in a series of 10 patients with NICM undergoing epicardial VT ablation, 7 had evidence of phrenic nerve capture near targeted ablation sites. In 1 patient, a peripheral angioplasty balloon, 18×60 mm) was used to displace the phrenic nerve, whereas adjacent sites without phrenic nerve capture were ablated in the other patients. Di Biase et al. compared multiple methods for separating the phrenic nerve from the epicardial surface during epicardial RF ablation. These were as follows: (1) placing a large size (25×40 mm.) peripheral angioplasty balloon in the pericardial space, (2) introducing saline in 20-mL increments, (3) introducing air in 20-mL increments, and (4) introducing a combination of air and saline. At each step, epicardial pacing was performed to look for phrenic nerve stimulation. The saline or air infusions were increased until there was loss of phrenic capture or the systolic blood pressure dropped below 60 mm Hg. Saline failed in all cases, air alone failed in 6 of 8 cases, the balloon could not be accurately placed or failed in 6 of 8 cases, but the air+saline combination was successful in 7 of 8 cases. The authors concluded that inflation with air and saline together provided the best strategy for preventing phrenic nerve injury. The intrapericardial balloon approach can also be used for esophageal protection during ablation of the posterior wall in AF ablation as described by Buch et al. Significant reductions in esophageal temperature were shown in an animal model with the use of this method of esophageal protection. A similar approach has also been used to protect the right phrenic nerve during ablation of atrial tachycardias arising along the crista terminalis.  

Protection of the coronary arteries is a key concern when ablating in the epicardial space. Few data are available in addition to the empirical experience of Sosa and colleagues discussed above. D’Avila et al. studied the effects of RF lesions delivered in the vicinity of coronary arteries and found that the risk of vascular damage varied inversely with vessel size. Larger vessels with internal diameter >0.5 mm demonstrated matrix proliferation in the media, but no intimal proliferation, whereas severe hyperplasia or endovascular thrombosis was seen in vessels <0.5 mm. This implies that larger vessels are protected by increased blood flow and possibly epicardial fat in addition. In a study of excised sheep hearts, Thyer et al. studied the effects of instillation of intracoronary chilled saline (5°C) during RF ablation over the coronary artery. The peak intracoronary temperature was 23.6°C in the chilled versus 54.6°C in the nonchilled vessels, whereas the median distance between the lesion and the artery was 0.42 mm versus 0.0 mm, respectively. Vile-Gonzalez and coworkers delivered RF lesions within 1 mm of the coronary arteries in 7 pigs and examined the acute (20 days) and chronic (70 days) effects. In both the acute and chronic specimens, intimal and medial thickening was seen, with replacement of smooth muscle cells by extracellular matrix. No significant stenosis was observed up to 70 days after ablation. It remains to be seen whether these findings can be applied clinically in the EP laboratory; for now, the best approach appears to be to avoid RF delivery within 1 cm of an epicardial coronary artery.

Finally, new approaches to the pericardial space and new devices may also lessen procedure-related complications. Scanavacca and colleagues recently described a percutaneous transthoracic approach in which the epicardial space was accessed via the atrial appendage. The epicardial space was successfully entered in 17 animals, and epicardial catheter manipulation was performed. Hemodynamic compromise occurred in 2 animals where the puncture was outside the atrial appendage (RV in one and tricuspid annulus in another). In a study of the pressure–frequency patterns in the thorax and pericardium, Mahapatra and coworkers found a distinctive 2-peak pattern in the pericardial space, which may aid development of new access tools. These workers have also designed a novel percutaneous access needle that draws the pericardium away from the surface of the heart before engagement, thus minimizing the risk of ventricular perforation.

**Energy Sources/Special Considerations for Epicardial Ablation**

To date, reported clinical studies have primarily used RF as the energy source for epicardial ablation. Lesion formation with standard RF catheters in the pericardial space will be limited by lack of convective cooling without circulating blood, resulting in high electrode temperatures at low-power settings. D’Avila and colleagues compared the efficacy of standard versus cooled-tip RF catheters for epicardial ablation in goat and pig animal models with healed myocardial infarctions. Standard RF lesions with the use of a 4-mm-tip catheter were 3.7±1.3 mm (25±16.8 W), whereas cooled-tip RF lesions were 6.7±1.7 mm (44±6.8 W). In areas covered by epicardial fat >3 mm, standard RF did not generate any appreciable lesions, whereas the cooled-tip RF lesions were 4.1±2 mm in depth (45±4.4 W). The authors concluded that cooled-tip RF could create epicardial lesions more effectively than standard RF ablation. During RF ablation with open irrigation catheters, the irrigation flow may be up to 30 mL/min, which would require intermittent drainage of the pericardial space. Theoretically, the absence of circulating blood in the pericardial space should be advantageous to the use of cryoablation. Di Biase et al. reported 2 cases, one an atrial tachycardia and the second a VT, in which cryoablation was ultimately successful when RF ablation had failed.
However, as shown by Lustgarten and colleagues, cryoablation may cause epicardial vessel damage by neointimal proliferation, with the probability of vessel damage inversely proportional to the vessel diameter. Suggested explanations included the presence of epicardial fat or better adherence of the cryocatheter. However, the mechanisms are still not clear, and the size of the catheter tip may play a an important role. Other potential energy sources include microwave, laser, and ultrasound. Epicardial mapping also needs to take into consideration epicardial fat, which can mimic low-voltage areas because of the insulating effects of fat. Electrogram characteristics can help delineate differences between true scar versus fat causing a reduction in recorded voltage.

Emerging Pericardial Procedures: Percutaneous Atrial Appendage Closure

In nonvalvular AF, up to 90% of thrombi originate from the left atrial appendage. Left atrial appendage occlusion devices are available, but require transseptal catheterization with associated complications, including air embolism, bleeding, tamponade, and device embolization requiring surgical removal. These problems have been the impetus for the development of new methods of left atrial occlusion by the use of the percutaneous epicardial approach. Friedman and colleagues reported preliminary results using a novel left atrial appendage closure device applied via a single percutaneous sheath in 4 dogs. The device consists of a grasping tool that can record left atrial appendage (LAA) activity and holds the appendage while a preloaded suture loop is advanced over the appendage to the base and fixed in place, occluding the mouth of the appendage. The device was able to achieve appendage closure in all the animals as confirmed by amputation of the appendage in 2 animals and necropsy in all animals. This method has been further modified by the use of a transsectally placed endoluminal balloon in the LAA with a magnet-tipped guidewire and a magnet-tipped wire of opposite polarity in the epicardial space (LARIAT, Sentre Heart) (Lariat refers to a type of knot or lasso). When the magnets are in proximity they align, allowing traction on the appendage as the snare loop is advanced to the base of the LAA. The endovascular guidewire and balloon are then detached, and the suture is tightened around the base of the LAA. In 5 animals in which the endocardial balloon was used, the level of the LAA closure was at the base, although this occurred in only 1 of 4 when it was not used. In all animals, LAA closure was complete without a leak. Lee and coworkers studied 26 dogs with the use of this device. Sixteen dogs were euthanized and found to have the base of LAA closed in all cases; in the remaining animals, examination of the LAA at 1 week, 1 month, and 3 months demonstrated a completely endothelialized origin of the LAA. In the first study in humans, Bartus at al studied 13 patients, either undergoing mitral valve surgery (n = 2) or AF ablation (n = 11). Both of the mitral valve repair patients had complete closure of the LAA determined by visual inspection, whereas this was achieved percutaneously in 10 of the 11 AF patients, with 1 patient requiring a thorascopic procedure to remove the snare owing to pectus excavatum. The procedure was terminated in 1 patient because of poor echocardiographic visualization of the marker balloon. Sixty-day follow-up was reported for 6 patients: 4 had complete LAA closure, and 2 had a 2-mm opening by color flow Doppler. The authors concluded that catheter-based suture ligation of the LAA is feasible in humans, with further investigation needed to determine long-term safety and efficacy.

Conclusions

In the past 15 years, interventional cardiac electrophysiology has ventured into the pericardial space with notable added benefits for treatment of arrhythmias, particularly for VT. In addition, the ability to access the epicardial surface of the heart has enabled mechanistic studies regarding arrhythmia mapping and substrates. Advances in epicardial therapies for atrial arrhythmias have been slower, but present an opportunity to emulate the achievements in treating ventricular arrhythmias.

New methods to make pericardial access easier and a lower risk procedure are being developed. Protection of the coronary arteries and surrounding structures such as the esophagus and phrenic nerves will remain an important feature when working in this space. Future developments in cardiovascular science could create the need for site-specific epicardial delivery of biological therapies.

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Disclosures

The University of California, Los Angeles has intellectual property developed by the authors that relate to epicardial interventions.

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22. Natale A, D’Avila A, Heist EK, 1767


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SUPPLEMENTAL MATERIAL

EPICARDIAL INTERVENTIONS IN ELECTROPHYSIOLOGY

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SUPPLEMENTAL FIGURE 1: Epicardial access needles and landmarks for needle entry: The top panel shows Tuohy epidural needles (6 inches and 3.5 inches) versions. The bottom left panel shows bony landmarks and the location of needle access. The lower middle panel shows the orientation of the tip of the Tuohy needle. The lower right panel shows a Sagittal plane magnetic resonance image of the heart and the chest wall showing anterior and posterior access angles. RV= right ventricle; [Left lower panel image by Patrick J. Lynch, Yale University, Tuohy needle tip image: www.ganfyd.org/no restrictions for non-commercial use]

MOVIE LEGENDS:

Movie 1: Pericardial access Step I: Pericardial staining with an epidural (Tuohy needle), LAO view just prior to epicardial access, showing ‘tenting’ of the pericardium

Movie 2: Pericardial access Step II: Pericardial puncture and contrast extravasation around the heart. This is immediately after the previous video (note mild staining of the myocardial wall that can be prevented by with increased experience and proper fluoroscopy settings, typically high frame rates). This can also be accomplished by wire insertion via the needle without contrast placement.

Movie 3: Pericardial access Step III: Wire advancement into the pericardial space in the LAO view, showing that the wire is extracardiac in the pericardial space.

Movie 4: Pericardial Imaging I: A pigtail catheter is placed deep in the oblique sinus to show contrast in the pericardium.

Movie 5: Pericardial Imaging II: 2 D echocardiography parasternal long axis view of a patient with pericardial effusion (not procedure related) showing fluid in the oblique sinus (arrow). PE= pericardial effusion, LA= left atrium, DAO= descending aorta. (Movie courtesy, John S Child, MD UCLA)

Movie 6: Pericardial Instrumentation I: A long sheath is placed in the pericardium for mapping the tricuspid annulus on the epicardial aspect.

Movie 7: Pericardial Instrumentation II: A ‘soft’ 5 F sheath is used for pericardiography to ensure there are no adhesions.

Movie 8: Pericardial Instrumentation III: Simultaneous epicardial and endocardial mapping of a right free wall accessory pathway with simultaneous right coronary injection. Camera sweeps from LAO to RAO in the cine run to image catheters in several angles.
Movie 9: Epicardial Voltage mapping: Epicardial voltage map from a patient with a large inferior wall scar. Voltage map scale shown in the movie.

Movie 10: Pericardial adhesions: Wire in pericardium 'dissecting' adhesions, this is a patient who had undergone epicardial mapping in an outside institution and was referred for repeat ablation.

Movie 11: Phrenic protection I: Pacing at ablation site prior to ablation shows phrenic capture.

Movie 12: Phrenic protection II: Pacing at ablation site after balloon placement prior to ablation shows lack of phrenic capture.

Movie 13: Direct Pericardial Imaging: A fiberoptic scope can be placed in the pericardium to image the heart to identify fat and other structures.

Movie 14: Vein of Marshall Mapping: A microelectrode catheter is placed within the vein of Marshall endocardially and a multielectrode catheter is placed epicardially.
SUPPLEMENTAL FIGURE 1: Epicardial Access Needles and Landmarks for Needle Entry

Direction of Needle Entry

- Curved end faces Heart
- Open end away from Heart toward right inferior quadrant 3-6 O’clock viewed from caudal view

Attenuated Needle

- RV
- Liver

Anatomical Landmarks
- Apex of lung
- Clavicle
- Coracoid process
- Acromion
- Right border of the heart
- Axillary fold
- Manubrium
- Thyroid cartilage
- Thyroid gland
- Trachea
- Humerus
- Border of left ventricle
- Dome of diaphragm
- Xiphoid cartilage
- Costal arch
- Infra斯特ernal angle
- Pleural reflexion

Inferior
Anterior