Background—Percutaneous epicardial ablation of ventricular arrhythmias arising from the left ventricular summit is limited by the presence of major coronary vessels and epicardial fat. We report the outcomes of percutaneous epicardial mapping and ablation of ventricular arrhythmias arising from the left ventricular summit and the ECG features associated with successful ablation.

Methods and Results—Between January 2003 and December 2012, a total of 23 consecutive patients (49±14 years; 39% men) with ventricular arrhythmias arising from the left ventricular summit underwent percutaneous epicardial instrumentation for mapping and ablation because of unsuccessful ablation from the coronary venous system and multiple endocardial LV/right ventricular sites. Successful epicardial ablation was achieved in 5 (22%) patients. In the remaining 18 (78%) cases, ablation was aborted for either close proximity to major coronary arteries or poor energy delivery over epicardial fat. The Q-wave amplitude ratio in aVL/aVR was higher in the successful group, with a ratio of >1.85 present in 4 (80%) patients in the successful group versus 2 (11%) in the unsuccessful group (P=0.008). The ratio of R/S wave in V1 was greater in the successful group, with 4 (80%) patients in the successful group having a R/S ratio of >2 in V1 versus 5 (28%) in the unsuccessful group (P=0.056). None of the patients in the successful group had an initial q wave in lead V1, as opposed to 6 (33%) in the unsuccessful group. The presence of at least 2 of the 3 ECG criteria above predicted successful ablation with 100% sensitivity and 72% specificity.

Conclusions—Epicardial instrumentation for mapping and ablation of ventricular arrhythmias arising from the left ventricular summit is successful only in a minority of patients because of close proximity to major coronary arteries and epicardial fat. A Q-wave ratio of >1.85 in aVL/aVR, a R/S ratio of >2 in V1, and absence of q waves in lead V1 help identify appropriate candidates for epicardial ablation. (Circ Arrhythm Electrophysiol. 2015;8:337-343. DOI: 10.1161/CIRCEP.114.002377.)

Key Words: catheter ablation ■ epicardial mapping ■ tachycardia, ventricular
WHAT IS KNOWN

• The left ventricular summit represents a common site of origin of idiopathic ventricular arrhythmias.
• Successful elimination of arrhythmias arising from the left ventricular summit can be achieved with ablation from adjacent structures.
• When ablation from adjacent sites is unsuccessful, a percutaneous pericardial approach has been suggested to achieve arrhythmia suppression.

WHAT THE STUDY ADDS

• This study reports on a series of patients in whom percutaneous pericardial access for epicardial mapping and ablation was attempted for arrhythmias arising from the left ventricular summit.
• Outcomes were poor overall, with an acute success rate of 22% and a long-term arrhythmia-free survival of 17%, likely because of the presence of epicardial fat at the tissue-catheter interface. In about two-thirds of cases, radiofrequency delivery in the epicardial location was aborted because of proximity to major epicardial coronary vessels.
• ECG features were found to be helpful in identifying cases where epicardial ablation may have a higher chance of success.

the LVS was defined as the triangular portion of the epicardial left ventricular outflow tract with the apex at the bifurcation between the left anterior descending and the circumflex coronary arteries and the base formed by an arc connecting the first septal perforator branch of the left anterior descending coronary artery with the circumflex coronary artery (Figure 1). The LVS is bisected by the GCV in to 2 regions: 1 closer to the apex of the triangle (ie, inaccessible area) and 1 at the base of the triangle (ie, accessible area). All the anatomic definitions were based on orthogonal fluoroscopy, intracardiac echocardiography, and intraprocedural coronary angiography.

All patients had failed treatment with at least 1 antiarrhythmic drug and atrioventricular nodal blocking agents; these were discontinued for at least 5 half-lives before the procedure. All patients signed a written informed consent according to the institutional guidelines of the University of Pennsylvania Health System. The baseline characteristics including the age, sex, nature of the clinical arrhythmia, and 12-lead ECG of the VAs were recorded.

Electrophysiological Study and Catheter Ablation

Patients presented to the cardiac electrophysiology laboratory in the fasting state. Conscious sedation was used as the preferred approach. General anesthesia with an inhaled anesthetic (typically sevoflurane) was used for obtaining pericardial access and during epicardial mapping and ablation. Atrial and ventricular burst pacing with and without intravenous isoproterenol (≤12 μg/min) was used to provoke the VAs in case of suppression after general anesthesia induction. Catheters were positioned in the heart using fluoroscopic guidance. A 6-F quadripolar catheter with 5-mm interelectrode distance (Bard Inc, Delran, NJ) was placed at the RV apex. A 7-F decapolar catheter with 5-mm electrodes and 2-mm interelectrode distance was advanced to the coronary sinus and at the GCV–AIV junction. A deflectable 8-F mapping/ablation catheter that had a 3.5-mm irrigated tip and a 2-mm ring electrode separated by 1 mm (Thermocool, Biosense Webster, Diamond Bar, CA) was advanced to the RV (transvenous approach), LV (retrograde aortic approach), coronary venous system, and epicardial space for mapping. Access to the pericardial space and epicardium was obtained using the approach described by Sosa et al.7 A 64-element phased-array intracardiac echocardiography catheter (AcuNav, Acuson, Mountain View, CA) was used to assist catheter manipulation, assess distance from the ostium of the left main coronary artery, monitor radiofrequency energy delivery, tissue-catheter contact, and complications. In addition, intracardiac echocardiography was used together with the 3-dimensional electroanatomic mapping system to define the anatomic details of structures adjacent to the epicardial LVS. During mapping in the LV and coronary cusp region, intravenous heparin was used to achieve an activated clotting time of ≥250 seconds. The site of origin of the VA was determined based on detailed activation and pace mapping. In particular, during activation mapping, the local activation time was consistently measured from the onset of the electrogram (earliest positive or negative deflection) of the distal bipole of the mapping catheter to the earliest onset of the QRS complex in any of the 12 ECG leads. Activation times were measured by 2 independent observers and displayed on a 3-dimensional electroanatomic map (CARTO; Biosense Webster, Diamond Bar, CA) as well as the electrophysiology recording system (Prucka; GE, Houston, TX). Pace-map match was visually judged by 2 observers, with each of the 12 ECG leads assessed for the QRS vector and major notching or deflections from baseline. An ideal match required identical QRS complexes between the paced beats and native VA in 12 of 12 ECG leads. Coronary angiography was performed to delineate the ostium and the course of the left coronary artery before ablation. Radiofrequency energy was only delivered if the site was >5 mm from a coronary artery. During radiofrequency energy delivery, if a suppression/elimination of VAs occurred within the initial 30 seconds, the application was maintained and carefully titrated for ≥60 seconds, targeting an impedance drop of 10 to 15 Ohms with a maximum temperature of 45°C and a maximum power of 40 watts. After ablation, atrial and ventricular burst pacing with and without intravenous isoproterenol (≤12 μg/min) was used to assess arrhythmia inducibility. Acute success was defined as inability to induce the clinical VAs (ventricular premature depolarization or ventricular tachycardia) at the end of the procedure and no recurrence during 24 hours of postprocedural hospital ECG monitoring.

Figure 1. Computer tomographic image depicting the anatomy of the left ventricular summit (LVS). The LVS is a triangular region of the LV epicardium with the apex at the bifurcation between the left anterior descending (LAD) and left circumflex (LCx) coronary arteries and the base formed by the arc connecting the first septal perforator branch of the LAD with the LCx (white dotted line and arrows). The LVS is bisected by the great cardiac vein (GCV) which separates it into 2 regions: (1) closer to the apex of the triangle (blue dotted line) and (2) a more lateral toward the base of the triangle (yellow dotted line). The former is less accessible for catheter ablation in the epicardial aspect because of proximity to coronary arteries and the presence of thicker layer of epicardial fat. AIV indicates anterointerventricular vein; and LM, left main coronary artery.
ECG Analysis
Detailed ECG analysis was performed offline on the Prucka CardioLab recording system (GE, Houston, TX) with the recordings displayed at a speed of 100 and 200 mm/s. Analysis of ECG tracings was performed according to previously reported measurements applied to the outflow tract region. In particular, the following ECG features were assessed: (1) QRS duration; (2) pseudodelta wave, intrinsicoid deflection time, maximum deflection index; (3) R wave amplitude in leads II, III, and aVF (and ratio between R wave in lead II/lead III); (4) Q wave amplitude in leads aVL and aVR (and ratio between Q wave in lead aVL/lead aVR); (5) R wave amplitude in leads V1, V2, and V3; (6) S wave amplitude in leads V1, V2, and V3; and (7) presence of a QS complex in lead I. Three QRS complexes were measured in each patient to confirm reproducibility, and the mean of the values was used.

Follow-Up
Antiarrhythmic drugs were not reinitiated if ablation was acutely successful. Post procedure, patients remained overnight in the hospital under continuous ECG monitoring. Beyond that, patients were followed up in the outpatient clinic at our institution or by their referring physician. Information about symptoms, VA burden using 24-hour Holter, or auto-triggered transtelephonic ECG monitoring was assessed 2 to 6 months after discharge.

Statistical Analysis
Descriptive statistics are reported as mean±SD or median and interquartile range for continuous variables and as absolute frequencies and percentages for categorical variables. Between-group comparisons were performed with the Mann–Whitney U test and Fisher exact test as appropriate. The Wilcoxon matched-pairs signed-rank test was used to compare activation mapping and ablation (impedance drops) data (repeated measurements on a single sample). To formally assess the degree of interobserver agreement for ECG analysis, 2 observers independently measured a total of 96 ECG parameters from 6 patients randomly selected. Interobserver disagreement was considered present when the timing difference exceeded 5 ms and the voltage difference exceeded 0.1 mV. All tests were 2-sided, and a P value <0.05 was considered statistically significant. Statistical analyses were performed with the Stata 12.1 statistical package (Stata Corporation, College Station, TX).

Results
Study Population
Patients participating in the study underwent the procedure between January 2003 and December 2012. Over this time frame 23 patients (age 50±14 years, mean left ventricular ejection fraction 55±14%) manifested VAs arising from the LVS in whom epicardial mapping and ablation was performed after failed ablation from multiple adjacent locations including the LV/RV endocardium, the coronary cusp region, and within the GCV–AIV. All patients had frequent drug-refractory symptomatic VAs, with 5 (22%) patients manifesting LV dysfunction (ejection fraction ≤50%; 3 with ejection fraction ≤40%). The predominant presenting arrhythmia was sustained ventricular tachycardia in 7 (30%), nonsustained ventricular tachycardia in 5 (22%), and frequent ventricular premature depolarizations in 11 (48%). In 11 of 23 (48%) patients, procedural cardiac magnetic resonance imaging (n=8) or contrast-enhanced cardiac computed tomography (n=3) were performed. Decision to perform preprocedural cardiac magnetic resonance imaging or cardiac computed tomography was based on clinical suspicion of underlying structural heart disease, typically presence of LV dysfunction (LV ejection fraction <50%), or physician preference. Preprocedural imaging revealed areas of delayed enhancement abnormalities in 4 of 11 (36%) cases, which were patchy and multifocal and seemed to extend to the LV summit region. The baseline clinical characteristics of the patients are summarized in Table 1.

Mapping of the Epicardial LVS
In all patients epicardial activation or pace mapping confirmed origin of the VAs from the LVS. In particular, detailed multisite activation mapping was possible in 15 of 23 (65%) cases. In 13 of these 15 cases, the epicardial LVS was the earliest site of activation, with an average activation time of –31±10 ms (range, –40 to –20 ms) pre-QRS. In 2 of 15 cases, the epicardial LV summit was either equally early (–35 ms) or slightly later (–28 ms versus –30 ms pre-QRS) compared with the anterior interventricular vein. In the latter case, however, pace mapping was perfect in the epicardial LVS. Of note, the activation times septal to the GCV–AIV were better compared with those lateral to the GCV–AIV (–17±20 ms pre-QRS versus –3±26 ms pre-QRS, respectively; P=0.004). Although delayed enhancement abnormalities in the LVS region were seen on cardiac magnetic resonance imaging in 4 cases, these were not corroborated by voltage mapping (normal distribution of bipolar and unipolar voltages); the mechanism for the VAs seemed to be focal (abnormal automaticity or triggered activity) in all patients.

Outcomes of Epicardial Ablation
Radiofrequency delivery on the epicardium was attempted only in 14 (61%) patients; in the remaining 9 (39%) cases, radiofrequency delivery was aborted because of close proximity to either the left anterior descending or circumflex coronary artery. Of the 14 patients in whom radiofrequency energy delivery was attempted, it was acutely successful in suppressing the VAs in only 5 (22%; Figure 2). No significant difference in the average impedance drop with radiofrequency application was found between the successful versus unsuccessful cases (median drop, 8 Ohms [interquartile range, 4–10] Ohms). The predominant arrhythmia was sustained ventricular tachycardia in 7 (30%), nonsustained ventricular tachycardia in 5 (22%), and frequent ventricular premature depolarizations in 11 (48%). In 11 of 23 (48%) patients, procedural cardiac magnetic resonance imaging (n=8) or contrast-enhanced cardiac computed tomography (n=3) were performed. Decision to perform preprocedural cardiac magnetic resonance imaging or cardiac computed tomography was based on clinical

Table 1. Clinical Characteristics of the Patients Included in the Study

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall (n=23)</th>
<th>Successful Ablation (n=5)</th>
<th>Unsuccessful Ablation (n=18)</th>
<th>PValue*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>50±14</td>
<td>50±12</td>
<td>50±15</td>
<td>0.911</td>
</tr>
<tr>
<td>Male sex, n (%)</td>
<td>9 (39)</td>
<td>1 (20)</td>
<td>8 (44)</td>
<td>0.611</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>55±14</td>
<td>65±4</td>
<td>53±14</td>
<td>0.021</td>
</tr>
<tr>
<td>Predominant presenting arrhythmia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sustained VT, n (%)</td>
<td>7 (30)</td>
<td>2 (40)</td>
<td>5 (28)</td>
<td>0.621</td>
</tr>
<tr>
<td>Nonsustained VT, n (%)</td>
<td>5 (22)</td>
<td>3 (60)</td>
<td>2 (11)</td>
<td>0.048</td>
</tr>
<tr>
<td>Frequent VPDs, n (%)</td>
<td>11 (48)</td>
<td>0 (0)</td>
<td>11 (61)</td>
<td>0.037</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; VPDs, ventricular premature depolarizations; and VT, ventricular tachycardia.

*Comparison between successful and unsuccessful ablation cases.
5–17 Ohms] versus 8 Ohms [interquartile range, 4–13 Ohms], respectively; \( P = 0.783 \). In all the successful cases, the VAs were localized at the base of the LVS triangle (most lateral aspect; Figure 1).

In 1 patient pericardial effusion/tamponade developed while mapping in the coronary venous system (likely per – foration of the GCV); this was drained with pericardiocentesis without further fluid reaccumulation and the procedure was completed. No other complications were observed. The clinical characteristics of patients with successful epicardial ablation compared with those with unsuccessful ablation are presented in Table 1.

**ECG Analysis**

**General Characteristics**

The right bundle branch block morphology with predominantly positive R wave in lead V1 was observed in 13 (57%) patients. The remaining patients (n=10) manifested a left bundle branch block morphology with a precordial R-wave transition in lead V2 or V3 in 7 (70%) cases, and beyond lead V3 in 3 (30%) cases. A QS complex in lead I was observed in 7 of 23 (30%) patients. For the entire cohort, average QRS duration was 149±24 ms, with a mean maximum deflection index of 0.52±0.09, a mean intrinsicoid deflection time of 85±22 ms, and a pseudodelta wave of 52±12 ms. The interobserver agreement in designating concordance for ECG measurements was 92%.

**Comparison Between Successful and Unsuccessful Epicardial Ablation**

A comparison of ECG characteristics between patients with successful epicardial ablation and those with unsuccessful ablation is reported in Table 2 and Figures 3 and 4. A right bundle branch block morphology of the V A was present in 4 (80%) successful and 9 (50%) unsuccessful cases (\( P = 0.339 \) for comparison). In patients manifesting left bundle branch block morphology, no significant difference between successful and unsuccessful cases was found in terms of precordial transition (\( P > 0.99 \) for comparison). The Q-wave ratio in leads aVL/aVR was significantly greater in patients with successful epicardial ablation compared with that in the unsuccessful group (2.63±1.31 versus 1.39±0.58, respectively; \( P = 0.017 \)), with 4 of 5 (80%) successful ablation cases having a ratio >1.85 (versus 2/18 [11%] unsuccessful cases, \( P = 0.008 \) for comparison). An R-wave to S-wave ratio in lead V1 >2 was present in 4 of 5 (80%) patients in the successful group compared with 5 of 18 (28%) cases of unsuccessful ablation (\( P = 0.056 \) for comparison). None of the patients in the successful group had an initial q wave in lead V1 versus 6 of 18 (33%) in the unsuccessful group. A QS complex in lead I was present in 3 (60%) successful and 4 (22%) unsuccessful cases.

### Table 2. ECG Characteristics in the Successful and Unsuccessful Ablation Cases

<table>
<thead>
<tr>
<th>Variable</th>
<th>Successful Ablation (n=5)</th>
<th>Unsuccessful Ablation (n=18)</th>
<th>( P ) Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qualitative findings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBBB morphology, n (%)</td>
<td>4 (80)</td>
<td>9 (50)</td>
<td>0.339</td>
</tr>
<tr>
<td>LBBB morphology, n (%)</td>
<td>1 (20)</td>
<td>9 (50)</td>
<td>0.339</td>
</tr>
<tr>
<td>QS in lead I, n (%)</td>
<td>3 (60)</td>
<td>4 (22)</td>
<td>0.142</td>
</tr>
<tr>
<td>Initial q in V1, n (%)</td>
<td>0 (0)</td>
<td>6 (33)</td>
<td>0.272</td>
</tr>
<tr>
<td>Duration, ms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>QRSd</td>
<td>147±16</td>
<td>149±26</td>
<td>0.970</td>
</tr>
<tr>
<td>IDT</td>
<td>76±5</td>
<td>88±24</td>
<td>0.079</td>
</tr>
<tr>
<td>MDI</td>
<td>0.51±0.07</td>
<td>0.52±0.11</td>
<td>0.794</td>
</tr>
<tr>
<td>Limb leads, mV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R in II</td>
<td>1.25±0.52</td>
<td>1.51±0.38</td>
<td>0.456</td>
</tr>
<tr>
<td>R in III</td>
<td>1.59±0.61</td>
<td>1.68±0.46</td>
<td>0.823</td>
</tr>
<tr>
<td>R in aVF</td>
<td>1.33±0.59</td>
<td>1.57±0.41</td>
<td>0.433</td>
</tr>
<tr>
<td>Ratio R in II/III</td>
<td>0.79±0.16</td>
<td>0.93±0.20</td>
<td>0.248</td>
</tr>
<tr>
<td>Q in aVL</td>
<td>1.03±0.41</td>
<td>0.91±0.29</td>
<td>0.391</td>
</tr>
<tr>
<td>Q in aVR</td>
<td>0.46±0.23</td>
<td>0.71±0.22</td>
<td>0.052</td>
</tr>
<tr>
<td>Ratio Q in aVL/aVR</td>
<td>2.63±1.31</td>
<td>1.39±0.58</td>
<td>0.017</td>
</tr>
<tr>
<td>Ratio Q in aVL/aVR &gt;1.85, n (%)</td>
<td>4 (80)</td>
<td>2 (11)</td>
<td>0.008</td>
</tr>
<tr>
<td>Precordial leads, mV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R in V1</td>
<td>0.51±0.32</td>
<td>0.40±0.32</td>
<td>0.331</td>
</tr>
<tr>
<td>R in V2</td>
<td>0.97±0.54</td>
<td>0.77±0.63</td>
<td>0.296</td>
</tr>
<tr>
<td>R in V3</td>
<td>1.86±0.83</td>
<td>1.45±0.71</td>
<td>0.456</td>
</tr>
<tr>
<td>S in V1</td>
<td>0.18±0.16</td>
<td>0.47±0.36</td>
<td>0.057</td>
</tr>
<tr>
<td>S in V2</td>
<td>0.65±0.50</td>
<td>0.90±0.67</td>
<td>0.551</td>
</tr>
<tr>
<td>S in V3</td>
<td>0.40±0.12</td>
<td>0.55±0.49</td>
<td>0.970</td>
</tr>
<tr>
<td>Ratio R/S in V1</td>
<td>5.98±5.85</td>
<td>1.81±2.23</td>
<td>0.100</td>
</tr>
<tr>
<td>Ratio R/S in V1 &gt;2, n (%)</td>
<td>4 (80)</td>
<td>5 (28)</td>
<td>0.056</td>
</tr>
<tr>
<td>Ratio R/S in V2</td>
<td>2.28±1.47</td>
<td>2.34±3.23</td>
<td>0.456</td>
</tr>
</tbody>
</table>

*Comparison between successful and unsuccessful ablation cases.
No significant differences between the successful and unsuccessful groups were detected in terms of total QRS duration (147±16 ms versus 149±26 ms, respectively; \( P = 0.970 \)), maximum deflection index (0.51±0.07 versus 0.52±0.11, respectively; \( P = 0.794 \)), intrinsicoid deflection time (76±5 ms versus 88±24 ms, respectively; \( P = 0.079 \)), pseudodelta wave (44±5 ms versus 55±12 ms, respectively; \( P = 0.086 \)), and R-wave ratio in leads II/III (0.79±0.16 versus 0.93±0.20, respectively; \( P = 0.248 \)).

**ECG Correlates of Successful Epicardial Ablation**

The 3 criteria that were tested to assess the value of the ECG to help identify patients who had successful epicardial ablation were (1) Q-wave ratio in leads aVL/aVR >1.85, (2) R/S wave ratio in lead V1>2, and (3) lack of initial q wave in lead V1. The presence of at least 2 of these 3 ECG criteria was associated with successful epicardial ablation with 100% sensitivity and 72% specificity.

**Follow-Up**

After a median follow-up of 36 months (interquartile range, 36 months), 3 of the 5 patients who had successful epicardial ablation were free from any recurrent VTs; one of these patients was maintained on propafenone. Another patient had a reduction in the ventricular premature depolarization burden after ablation (from 23% to 7.8% burden on 24-hour Holter monitoring) and has remained off antiarrhythmic drug therapy. Finally, 1 patient had recurrent ventricular tachycardia, which was controlled with amiodarone.

**Discussion**

The present study was specifically designed to assess the outcomes of percutaneous epicardial mapping and ablation for VTs originating from the LVS and to evaluate the ECG features associated with successful epicardial ablation. To the best of our knowledge, our study includes the largest population to date with LVS arrhythmias undergoing percutaneous epicardial mapping and ablation. The major findings are as follows: (1) the outcome of catheter ablation for LVS arrhythmias from the epicardium is poor, with an acute success rate of 22% and a long-term arrhythmia-free survival of 17% and (2) ECG features including a Q-wave ratio in leads aVL/aVR >1.85, and R/S wave ratio in lead V1 of >2, and the lack of an initial q wave in lead V1 may help identify cases where epicardial ablation may have a higher chance of success.

**Epicardial Catheter Ablation of VTs From the LVS**

Catheter ablation of VTs arising from the LVS can be challenging, given the proximity to critical structures such as major coronary vessels and inability to deliver effective radiofrequency lesions because of the presence of a thick layer of epicardial fat. In many of these cases, radiofrequency delivery from...
adjacent sites, such as the endocardial LV/RV, coronary cusp region or coronary venous system is successful in eliminating the arrhythmias.3–5 In this regard, intracardiac echocardiography together with 3-dimensional electroanatomic mapping is particularly valuable in defining the anatomic details of structures adjacent to the epicardial LVS; however, in this series, intracardiac echocardiography did not enhance ablation success. In a recent study by our group, successful ablation of VAs from the inaccessible LVS area was achieved from the left coronary cusp in 56% of cases.3 Similarly, Yamada et al,1 who first described the anatomy and electrophysiological characteristics of arrhythmias arising from the LVS in a series of 27 patients, reported successful ablation from the GCV–AIV region in >70% of cases. When ablation from adjacent sites fails, a percutaneous epicardial access for mapping and ablation may be considered.1,6 Thus far, the published experience with epicardial mapping and ablation of LVS arrhythmias is scant. For instance, in the study by Yamada et al,1 only 9 of 27 (33%) patients underwent epicardial mapping and ablation which was successful in 4 of 9 (44%) cases. In our study, in about two-thirds of cases, radiofrequency delivery in the epicardial location was aborted because of proximity to major epicardial coronary vessels. When radiofrequency ablation was attempted after verifying safe distance from major coronary vessels, it was successful in eliminating the arrhythmia only in a small subset of these patients. We think this is likely because of the presence of epicardial fat at the tissue-catheter interface,15,16 although we did not find significant differences in average impedance drops with radiofrequency application in successful versus unsuccessful cases. Prior studies have shown that epicardial fat represents a major limitation to achieving adequate lesions by radiofrequency energy17; in these cases, a different source of ablation energy, such as cryoablation, may be more efficacious.18 In rare cases, minimally invasive surgical ablation has also been shown to be effective in treating LVS arrhythmias, which were successfully targeted after mobilizing the coronary artery away from the site of cryoablation in the area of interest.19 However, the degree of incremental benefit with these techniques compared with conventional percutaneous radiofrequency ablation warrants validation in larger studies. None of the patients included in our series underwent cryoablation or surgical ablation.

ECG Features Associated With Successful Epicardial Ablation

The accessible area of the LVS is located inferiorly and laterally to the GCV, where the anatomic separation between the left anterior descending and circumflex coronary arteries is maximal and there is lower chance of having a thick layer of epicardial fat (Figure 1).20,21 Indeed, the 3 ECG features found to be more prevalent in successful versus unsuccessful cases (ie, Q-wave ratio aVL/aVR >1.85, R/S ratio in V1>2, and lack of q wave in V1) and reflect a more lateral site of origin of the VAs (distant from the midline and the apex of the LVS triangle). Accordingly, more patients in the successful group had a QS pattern in lead I, which again points to a more lateral (and epicardial) origin of the VAs.12 The low ablation success from the epicardial location in our series suggests that the majority of these VA were originating from the inaccessible location of the LVS. This is corroborated by the observation that the activation times septal to the GCV/AIV were better compared with those lateral to the GCV/AIV. In our study, the presence of at least 2 of the 3 ECG criteria was associated with successful epicardial ablation with a 100% sensitivity and 72% specificity. Our findings support the usefulness of ECG analysis to select cases with the greatest chance of success of epicardial ablation in the LVS region.

![Figure 4. Comparison of 12-lead ECG of the successful and unsuccessful epicardial ablation groups.](http://circep.ahajournals.org/issue)
Limitations
This was a single-center observational study. The decision to obtain percutaneous epicardial access for mapping/ablation was made by the operator during the procedure after failed ablation attempts from multiple adjacent structures; this may have introduced subjective bias. Although, in our series, the epicardial LVS was accessed in all patients without difficulty, in some cases the left atrial appendage may prevent adequate epicardial mapping in the LVS region. Our institution is a tertiary referral centers for the treatment of VAs and so the characteristics of the study population may not be generalized. Given the small number of patients, our findings with respect to ECG correlates of success should be viewed as hypothesis generating only and deserve to be validated prospectively in a larger and independent sample of patients.

Conclusions
In patients manifesting ventricular arrhythmias arising from the LV summit, the outcome of epicardial ablation is poor in the majority of cases because of proximity to major coronary vessels and presence of epicardial fat. ECG features pointing to a more leftward/lateral site of origin of the VA in this location (ie, Q-wave ratio of >1.85 in aVL/aVR, a R/S ratio of >2 in V1, and absence of q waves in lead V1) may help identify appropriate candidates for successful epicardial ablation.

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Disclosures
None.

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Percutaneous Epicardial Ablation of Ventricular Arrhythmias Arising From the Left Ventricle: Outcomes and Electrocardiogram Correlates of Success
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