Idiopathic Ventricular Arrhythmias Originating from the Left Ventricular Summit
Anatomic Concepts Relevant to Ablation

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Background—The summit of the left ventricle (LV) is the most superior portion of the epicardial LV bounded by an arc from the left anterior descending coronary artery, superior to the first septal perforating branch to the left circumflex coronary artery. Ventricular arrhythmias (VAs) originating from this region may present challenges for catheter ablation.

Methods and Results—We studied 27 consecutive patients with VAs originating from the LV summit. The great cardiac vein (GCV) divides this region between an inferior area accessible to ablation and a superior, inaccessible area. Successful ablation was achieved within the GCV in 14 patients and on the epicardial surface in 4. Ventricular prepotentials were recorded at the successful ablation site in 80% of these patients. In 5 patients, ablation was abandoned because of inaccessibility of the catheter to the myocardium or high impedance with radiofrequency application within the GCV. In the remaining 4 patients, epicardial mapping suggested VA origins in a region of low voltage that was located superior to the GCV (inaccessible area), and ablation was abandoned because of close proximity to the coronary arteries or high impedance. A right bundle-branch block, transition zone, R-wave amplitude ratio in leads III to II, Q-wave amplitude ratio in leads aVL to aVR, and S waves in lead V6 accurately predicted the site of origin.

Conclusions—LV summit VAs may be ablated within the GCV or inferior to the GCV on the epicardial surface, though sites superior to the GCV are usually inaccessible to ablation. (Circ Arrhythm Electrophysiol. 2010;3:616-623.)

Key Words: idiopathic ventricular arrhythmia ▪ left ventricular summit ▪ great cardiac vein ▪ epicardial ▪ radiofrequency catheter ablation

The prevalence, ECG, and electrophysiological features and response to catheter ablation of idiopathic ventricular arrhythmias (VAs) originating from the left ventricle (LV) have been increasingly established in the last decade.1–13 Although endocardial catheter ablation is usually successful, epicardial ablation using transvenous or transpericardial approaches may sometimes be required in these VAs.5–8,11–13 A region of the LV epicardial surface bounded by the left anterior descending coronary artery (LAD) and left circumflex coronary artery (LCx) that lies superior to the aortic portion of the LV ostium occupies the most superior portion of the LV and has been termed the LV summit by McAlpine.14 This region near where the great cardiac vein (GCV) ends and the anterior interventricular cardiac vein (AIVV) begins is one of the major sources of epicardial idiopathic VAs. The LV summit is bisected by the GCV into an area lateral to this structure, which is accessible to epicardial catheter ablation (the accessible area) and a superior region that is inaccessible to catheter ablation because of the close proximity of the coronary arteries and the thick layer of epicardial fat that overlies the proximal portion of these vessels (the inaccessible area). This study was undertaken to determine the ECG and electrophysiological features and results of the catheter ablation of idiopathic epicardial VAs originating from the LV summit.

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Methods

Patient Characteristics
The study population was drawn from 221 consecutive patients (132 men; mean age, 53±15 years; range, 14 to 82) with symptomatic idiopathic sustained ventricular tachycardia (VT) (n=84), nonsustained VT (n=37), or premature ventricular contractions (PVCs)
(n=100) originating from the LV, who were pooled from the University of Alabama at Birmingham, Aichi Prefectural Cardiovascular and Respiratory Center, Nagoya Dai-ni Red Cross Hospital, and Nagoya University Graduate School of Medicine. Echocardiography and exercise stress testing or coronary angiography demonstrated no evidence of structural heart disease in any patient. The sites of origin of the VA included the aortic root in 70 (31.7%) patients, aortomitral continuity in 18 (8.1%), epicardial surface of the LV in 32 (14.5%) (LV ostium in 27 and crux in 5), mitral annulus in 33 (14.9%), fascicles of the left bundle-branch in 40 (18.1%), papillary muscles in 23 (10.4%), and other sites in 5 (2.3%). The subjects of the present study were the 27 patients (13 men, 46±11 years, 24 to 70) with a site of the VA origin in the epicardial LV ostium that was determined by successful catheter ablation or presumed by electrophysiological study. The baseline characteristics including the age, sex, nature of the clinical arrhythmia, and 12-lead ECG during the VAs were recorded. Each patient gave written informed consent, and all antiarrhythmic drugs were discontinued for at least 5 half-lives before the study.

Electrophysiological Study
For mapping and pacing, multipolar catheters were positioned at the His bundle region, coronary sinus, and right ventricular apex. Mapping in the GCV and AIVV was usually performed using a 6F or 7F decapolar catheter advanced from the right femoral vein or internal jugular vein. This catheter was advanced until the electrode pair 3 to 4 recorded earlier ventricular activation than the most distal electrode pair 1 to 2 during the VAs. When this was impossible, a 2.3F multielectrode catheter (PATHFINDER, CARDIMA, Fremont, Calif.) was advanced through a 7F Amplatz angiographic catheter via the right femoral vein for mapping within these veins. Endocardial mapping and pacing in the ventricular outflow tracts were performed using a 7F, 4- or 5-mm-tip ablation catheter via the right femoral vein and artery. During the procedures in the LV, intravenous heparin was administered to maintain an activated clotting time >250 seconds. When few PVCs were observed at the beginning of the electrophysiological study, induction of the Vt or PVCs was attempted by burst pacing from the RV outflow tract (RVOT) or apex with the addition of an isoproterenol infusion.

Mapping and Radiofrequency Catheter Ablation
Activation mapping was performed in all cases to identify the earliest site of ventricular activation during the Vt or PVCs. In some patients, when the Vt or PVCs were frequent, electroanatomic mapping was performed as previously reported.15,16 Pace mapping was also performed using the distal bipolar electrodes at a pacing cycle length of 500 ms and stimulus amplitude of 1 mA greater than the late diastolic threshold (up to a maximum output of 20 mA and pulse width of 2.0 ms). The score for the pace mapping was determined from the R/S ratio and notch of the R wave in the 12-lead ECG as previously reported (perfect pace mapping was equal to 24 points).17 An excellent pace map was defined as a pace map that obtained a score of ≥20.

When endocardial catheter ablation was unsuccessful or the local ventricular activation during the VAs was earlier in the GCV or AIVV than at any endocardial site, epicardial catheter ablation using transvenous or transpericardial approaches was considered. When the local ventricular activation within the GCV or AIVV preceded the QRS onset by more than 20 ms and pacing from the site of the earliest ventricular activation within the GCV or AIVV produced an excellent match to the QRS complex of the VAs, catheter ablation within the GCV or AIVV was attempted. Otherwise or when the radiofrequency ablation within the GCV or AIVV failed to eliminate the VAs, epicardial mapping and ablation via a subxiphoid approach were performed. Nonirrigated radiofrequency current was delivered with a target temperature of 55°C and maximum power output of 30 W. Irrigated radiofrequency current was delivered in the power-control mode starting at 20 W in the GCV and AIVV and 30 W on the epicardial surface with an irrigation flow rate of 30 mL/min. The radiofrequency power was titrated to as high as 30 W and 40 W, respectively, with the goal being to achieve a decrease in the impedance of 8 to 10 Ω and with care taken to limit the temperature to <40°C. During the epicardial catheter ablation using transvenous and transpericardial approaches, simultaneous left coronary angiography was performed every 15 seconds to ensure the location of the ablation catheter relative to the left coronary arteries and to minimize the risk of thermal injury to that vessel. A radiofrequency application was never delivered within 5 mm of a coronary artery. When an acceleration or reduction in the frequency of the Vt or PVCs was observed during the first 10 seconds of the application, the radiofrequency delivery was continued for 30 to 60 seconds. Otherwise, the radiofrequency delivery was terminated, and the catheter was repositioned. The end point of the catheter ablation was the elimination and noninducibility of Vt or PVCs during an isoproterenol infusion (2 to 4 μg/min) and burst pacing from the right ventricle (to a cycle length as short as 300 ms). After epicardial catheter ablation, left coronary angiography was repeated to ensure that there was no evidence of injury to the coronary arteries.

Follow-up after the procedure included clinic visits with 12-lead ECGs and 24-hour ambulatory (Holter) monitoring and telephone calls to all patients and their referring physicians. All patients who reported symptoms were given a 24-hour Holter monitoring or event monitor to document the cause of the symptoms. Successful catheter ablation was defined as no recurrence of any VAs during >6 months of follow-up.

Anatomic Definition of the LV Summit
The LV summit was defined based on fluoroscopy and coronary angiography as the region on the epicardial surface of the LV near the bifurcation of the left main coronary artery that is bounded by an arc from the LAD superior to the first septal perforating branch anterior to the LCx laterally (Figure 1). Thus, the radius of this arc is the distance from the bifurcation of the left coronary arteries to the first septal perforator. The GCV bisects the LV summit into a superior portion that is in close proximity to the proximal coronary arteries and overlying epicardial fat (the inaccessible area) and an inferior portion that may be accessible to epicardial catheter ablation (the accessible area).

ECG Analysis
The simultaneous 12-lead ECGs during the VAs and pace mapping were recorded digitally at a sweep speed of 100 to 200 mm/s in all patients for offline analysis. The QRS morphologies including a bundle-branch block pattern, axis, configuration in leads I and V5 or V6 were recorded. In lead I, the presence of an R wave was the main concern because the absence of an R wave represents an activation vector directed completely from left to right, suggesting a VA origin located in the LV free wall. In lead V6 or V9, the main concern was the presence of an S wave, which was considered to be a characteristic and convenient ECG finding of aortomitral continuity VAs.5,19,20 Probably because the S wave in lead V6 or V9 is consistent with a right bundle-branch block pattern, usually present in VAs with an LV endocardial origin.

The QRS duration, maximal R-wave amplitude in the inferior leads, and maximum deflection time in the precordial leads were measured with electronic calipers by 2 experienced investigators blinded to the site of the origin. The QRS duration was measured as the interval between the earliest deflection of the ventricular complex in any of the 12 simultaneous leads to the latest offset in any lead and maximum deflection time from the QRS onset to the maximum (+) or (−) deflection in each precordial lead. If there were discrepancies between those results, they were adjudicated by a third investigator. The maximum deflection index (MDI) was calculated by dividing the shortest time to maximum deflection in any precordial lead by the QRS duration. The ratio of the Q-wave amplitude in leads aVL to aVR (aVL/aVR) and that of the R-wave amplitude in leads III to II (III/II) were also calculated. An MDI of >0.54 was used as a predictor of an epicardial focal VA origin according to previous reports.5,14,19 A long precordial MDI, reflecting delayed initial activation of the LV, is considered to discriminate between an epicardial and endocardial VA origin because of the slower spread of the activation from the VA origin on the epicardial surface relative
pericardial approach was performed without any attempts of GCV or AIVV, epicardial mapping and ablation via a pericardial approach were performed. In the remaining 20 patients, radiofrequency ablation was attempted within the GCV or AIVV (Figure 3). Radiofrequency ablation was successful in 13 of the 20 patients with a nonirrigated ablation catheter in 4, irrigated ablation catheter in 8, and both in 1 patient (9 in the GCV and 4 in the AIVV). In 2 patients, it was unsuccessful with slight reduction in the PVC frequency, and epicardial mapping and ablation via a pericardial approach were added. In 5 of the 20 patients, radiofrequency ablation within the GCV or AIVV was abandoned despite an almost perfect pace map because of the inaccessibility of the ablation catheter to the site of the earliest activation in one patient or high impedance in 4 patients (2 in the GCV and 2 in the AIVV). No epicardial mapping or ablation via a pericardial approach was performed in these patients because an irrigation catheter was not available in 3 Japanese patients and 2 patients with PVCs refused an epicardial procedure. Because the site of the VA origin was suggested to be within the GCV or AIVV, these 5 patients were combined with the patients with a successful ablation in the GCV and AIVV in a further study. In a total of 9 patients (5 patients without any early local ventricular activation or an excellent pace map within the GCV or AIVV and 4 patients with an unsuccessful ablation in the GCV, AIVV, LCC, or RVOT), epicardial mapping and ablation via a pericardial approach were performed. In 4 of these patients including 1 patient with unsuccessful ablation within the GCV, successful ablation was achieved on the epicardial surface located lateral and inferior to the GCV and AIVV (the accessible area) (Figures 1 and 4). In another 4 of these patients including 2 patients with unsuccessful ablation within the LCC and RVOT, epicardial mapping revealed the earliest ventricular activation in the region that was bounded by the LAD and LCx superior to the GCV and AIVV (the inaccessible area).
In all 4 patients, radiofrequency ablation was abandoned because of the close proximity to the coronary arteries and high impedance (>300 Ω), although it was attempted in 2 patients (Figure 2). In the inaccessible area, a low amplitude (<0.5 mV) of the local ventricular electrogram was noted (Figure 2). In the remaining patient with unsuccessful ablation within the AIVV, epicardial mapping revealed no earlier ventricular activation on the epicardial surface than within the AIVV and epicardial ablation via a pericardial approach was abandoned. However, in this patient, the PVCs completely disappeared approximately 5 hours after the procedure, and the successful ablation site of the PVC origin in this patient was suggested to be within the AIVV. Finally, successful ablation was achieved in 9 in the GCV, 5 in the AIVV, and 4 in the accessible area. Ventricular prepotentials were recorded at the successful ablation site in 15 (83%) of these patients (Figure 3). In all of the 4 patients with VAs presumably originating from the inaccessible area, pacing from any endocardial or epicardial sites never produced an excellent pace map. Specifically, the R-wave amplitude of the spontaneous VAs in the inferior leads was higher than could be achieved with pacing at any endocardial or epicardial site (Figure 5). Fluoroscopy and coronary angiography demonstrated that the sites of determined and presumed VA origins were located within the LV summit in all 27 patients. During the follow-up period (median, 12; interquartile range, 16 months) after the successful ablation, all 18 patients remained free of any VAs without any antiarrhythmic drugs. No complications occurred.

**Comparison of Clinical, ECG, and Electrophysiological Parameters of VAs Originating From the LV Summit**

The results of the clinical, ECG and electrophysiological parameters of these VAs are summarized in Tables 1 and 2.

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**Figure 3.** Twelve-lead ECGs obtained during PVCs and pacing from the successful ablation site within the GCV (left panels) and cardiac tracings obtained at the successful ablation site during the PVCs (right panels). Note that ventricular prepotentials (arrowheads) were recorded at the successful ablation site within the GCV. ABL (HB) d,p indicates the distal and proximal electrode pairs of the ablation (HB) catheter; CS 1 to 5, first to fifth electrode pairs of the coronary sinus catheter; PM, pace map; and V-QRS, local ventricular activation time relative to the QRS onset.

**Figure 4.** Successful ablation of VAs originating from the accessible area in the LV summit. Abbreviations are as in Figure 3.
The patient symptoms associated with the VAs were palpitations, fatigue, and dizziness, but no patients reported syncope. The maximal amplitude of the R wave in the inferior leads was highest for VAs with a presumed origin in the inaccessible area, second in those with an origin in the GCV and AIVV, and lowest in those with an origin in the accessible area (P=0.092). A right bundle-branch block pattern of the VAs was observed in all patients with an origin in the accessible area and was dominant in those with an origin in the GCV and AIVV but rare in those with an origin in the inaccessible area. The transition zone of the VAs that was earlier than in lead V1 was dominant in the patients with an origin in the accessible area and GCV and AIVV, whereas it was never observed in those with an origin in the inaccessible area. The III/II and aVL/aVR amplitude ratios were significantly higher in the VAs with an origin in the accessible area than in those with an origin in the GCV and AIVV and the inaccessible area (P<0.010 and P=0.005, respectively). No S waves in lead V5 or V6 were observed in any of the VAs with an origin in the inaccessible area and were observed in one fourth of the VAs with an origin in the GCV and AIVV and the accessible area. There were no significant

Table 1. Clinical, ECG, and Electrophysiological Characteristics

<table>
<thead>
<tr>
<th>Origin</th>
<th>Age</th>
<th>Sex, Male/Female</th>
<th>Type (SVT/NSVT/PVC)</th>
<th>QRSd, ms</th>
<th>QRS Morphology</th>
<th>Transition</th>
<th>Lead I</th>
<th>R Amp in Inferior Leads, mV</th>
</tr>
</thead>
<tbody>
<tr>
<td>GCV+/AIVV</td>
<td>49</td>
<td>9/10</td>
<td>5/4/10</td>
<td>175</td>
<td>RBBB+</td>
<td>V1&lt;; 13</td>
<td>QS; 4</td>
<td>1.5</td>
</tr>
<tr>
<td>(n=12+7)</td>
<td>(42 to 55)</td>
<td></td>
<td></td>
<td>(166 to 184)</td>
<td>LBBB+</td>
<td>V2;V3&lt;; 5</td>
<td>rS; 15</td>
<td>(1.2 to 1.8)</td>
</tr>
<tr>
<td>Accessible area</td>
<td>42</td>
<td>1/3</td>
<td>1/2/1</td>
<td>182</td>
<td>RBBB+</td>
<td>V3&lt;; V3&lt;; 1</td>
<td>qrs; 3</td>
<td>1.0</td>
</tr>
<tr>
<td>(n=4)</td>
<td>(35 to 48)</td>
<td></td>
<td></td>
<td>(139 to 225)</td>
<td>RBBB+</td>
<td>V4&lt;; V4&lt;; 4</td>
<td>QS; 1</td>
<td></td>
</tr>
<tr>
<td>Inaccessible area</td>
<td>51</td>
<td>2/2</td>
<td>1/2/1</td>
<td>181</td>
<td>LBBB+</td>
<td>rS; 2</td>
<td>rsr'; 1</td>
<td>(0.3 to 1.7)</td>
</tr>
<tr>
<td>(n=4)</td>
<td>(34 to 67)</td>
<td></td>
<td></td>
<td>(150 to 212)</td>
<td>LBBB+</td>
<td>V1&lt;; V1&lt;; 4</td>
<td>QS; 1</td>
<td></td>
</tr>
</tbody>
</table>

P value          | 0.568| 0.682| 0.638| 0.758| 0.052| 0.126| 0.006| 0.092|

LBBB (RBBB) indicates left (right) bundle-branch block; LIA (RIA), left (right) inferior axis; NSVT, nonsustained ventricular tachycardia (VT); QRSd, QRS duration; R amp, maximum R-wave amplitude; and SVT, sustained VT.

Continuous variables are expressed as the group mean with 95% confidence intervals in parentheses.
Table 2. Clinical, ECG, and Electrophysiological Characteristics

<table>
<thead>
<tr>
<th>Origin</th>
<th>III/II Ratio</th>
<th>aVL/aVR Ratio</th>
<th>S (+) in V5 or V6</th>
<th>MDI (% of &gt;0.54)</th>
<th>Pre-P (+)</th>
<th>V-QRS, ms</th>
<th>No. of Radiofrequency Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>GCV+ - AIVV (n=12+7)</td>
<td>1.2 (1.1 to 1.3)</td>
<td>1.5 (1.2 to 1.8)</td>
<td>5 (26%)</td>
<td>0.53 (0.49 to 0.57)</td>
<td>47%</td>
<td>16 (84%)</td>
<td>–32 (–28 to –36) 2.1 (1.4 to 2.8)</td>
</tr>
<tr>
<td>Accessible area (n=4)</td>
<td>1.4 (1.2 to 1.6)</td>
<td>2.3 (1.6 to 3.0)</td>
<td>1 (25%)</td>
<td>0.53 (0.49 to 0.57)</td>
<td>50%</td>
<td>3 (75%)</td>
<td>–38 (–18 to –58) 3.0 (1.7 to 4.3)</td>
</tr>
<tr>
<td>Inaccessible area (n=4)</td>
<td>1.1 (0.9 to 1.3)</td>
<td>0.9 (0.6 to 1.2)</td>
<td>0 (0%)</td>
<td>0.54 (0.38 to 0.70)</td>
<td>50%</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

aVL/aVR ratio indicates the ratio of the Q-wave amplitude in leads aVL to aVR; Pre-P, prepotential; V-QRS, local ventricular activation time relative to the QRS onset at the successful ablation site; and III/II ratio, R-wave amplitude ratio in leads III to II.

Continuous variables are expressed as the group mean with 95% confidence intervals in parentheses.

Table 3. Sensitivity, Specificity, and Positive and Negative Predictive Accuracies of the ECG Characteristics for a Ventricular Arrhythmia Origin

<table>
<thead>
<tr>
<th>QRS Characteristics</th>
<th>Subject</th>
<th>Site of Prediction</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBBB</td>
<td>LV summit (n=27)</td>
<td>GCV+ - accessible area (n=23)</td>
<td>78%</td>
<td>75%</td>
<td>95%</td>
<td>38%</td>
</tr>
<tr>
<td>Transition zone &lt;V1</td>
<td>LV summit (n=27)</td>
<td>GCV+ - accessible area (n=23)</td>
<td>70%</td>
<td>100%</td>
<td>100%</td>
<td>36%</td>
</tr>
<tr>
<td>aVL/aVR ratio &gt;1.1</td>
<td>LV summit (n=27), Avg=1.56, 95% CI=1.56 ±0.27</td>
<td>GCV+ - accessible area (n=23), Avg=1.56 ±0.27</td>
<td>87%</td>
<td>100%</td>
<td>100%</td>
<td>57%</td>
</tr>
<tr>
<td>S waves in V5 or V6</td>
<td>LV summit (n=27)</td>
<td>GCV+ - accessible area (n=23)</td>
<td>74%</td>
<td>100%</td>
<td>100%</td>
<td>40%</td>
</tr>
<tr>
<td>III/II ratio &gt;1.25</td>
<td>GCV+ - accessible area (n=23), Avg=1.23, 95% CI=1.23 ±0.08</td>
<td>Accessible area (n=4), Avg=1.23 ±0.08</td>
<td>100%</td>
<td>74%</td>
<td>44%</td>
<td>100%</td>
</tr>
<tr>
<td>aVL/aVR ratio &gt;1.75</td>
<td>GCV+ - accessible area (n=23), Avg=1.68, 95% CI=1.68 ±0.29</td>
<td>Accessible area (n=4), Avg=1.68 ±0.29</td>
<td>100%</td>
<td>74%</td>
<td>44%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Avg indicates average; CI, confidence interval; NPV, negative predictive value; and PPV, positive predictive value. Other abbreviations are as in Tables 1 and 2.
ablation may save procedure and fluoroscopic time and avoid some complications.

Because the LV summit is bounded by the LAD and LCx coronary arteries and the inaccessible area is separated from the accessible area by the GCV and AIVV, a catheter within these veins is essential for mapping and coronary angiography is necessary to protect the coronary arteries from inadvertent damage by radiofrequency ablation. Biplane fluoroscopic images and left coronary angiography may be recommended to identify whether the site of origin is within the accessible or inaccessible regions of the LV summit.

Comprehensive mapping within the GCV and AIVV should be performed first for LV summit VAs. However, when mapping within these venous structures does not suggest an origin within these veins, epicardial mapping via a pericardial approach should be considered before radiofrequency energy is applied.

Based on the anatomy of this region, several ECG parameters may be helpful for predicting the site of origin. When LV summit VAs exhibit a right bundle-branch block pattern, transition zone earlier than lead V1, an aVL/aVR amplitude ratio of >1.1, and S waves in V4 or V5, those VAs are likely to be cured by catheter ablation within the GCV, AIVV or the accessible area. When LV summit VAs exhibit a III/II amplitude ratio of >1.25 and an aVL/aVR amplitude ratio of >1.75, those VAs are likely to require a pericardial approach for ablation. Comparison of amplitude of the R waves in the inferior leads during between VAs and pace mapping from the GCV and AIVV may be helpful for predicting the VA origins in the inaccessible area in the LV summit. Because the inaccessible area is located most superiorly in the LV, followed by the GCV and AIVV, VAs originating from the inaccessible area may exhibit higher amplitude of the R waves in the inferior leads than the QRS complexes obtained by pacing from any other sites in the LV, even from the GCV and AIVV. When the amplitude of the R waves in the inferior leads is higher during the VAs than during pacing from the GCV and AIVV, this finding may suggest VAs with an origin in the inaccessible area. Ventricular prepotentials were often recorded at the successful ablation sites of the LV summit VAs. This finding was consistent with the other LV ostial VAs.2–4,8,9,11 These ventricular prepotentials may be very helpful for guiding the site of successful ablation of LV summit VAs.

In 1 of the patients in this study, the VAs were eliminated by the delayed effect of the radiofrequency ablation within the AIVV. During radiofrequency ablation, lesion formation may extend even though radiofrequency energy delivery has been terminated.20 Therefore, the site of the VA origin in that patient might have been intramural rather than epicardial.

**Study Limitations**

This study had dual problems with the statistical tests. First, multiple testing and multiple probability values were generated. Second, statistical tests were applied to 2 small comparison groups (n = 4 in each), resulting in a low power for detecting any differences. Therefore, the possibility could not be excluded that the statistical significance of differences in this study might have been due to chance alone.

**Conclusions**

The LV summit is the most common site of idiopathic epicardial LV VA origins. LV summit VAs are most commonly ablated within the GCV or AIVV but sometimes from the epicardial surface more lateral to these venous structures. Several LV summit VAs arise from an area that is inaccessible to catheter ablation, bounded by the left coronary arteries and superior to the GCV and AIVV.

**Disclosures**

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**References**

CLINICAL PERSPECTIVE

Success of catheter ablation for focal ventricular arrhythmias is dependent on anatomic location. In this case series, we characterize the anatomy and focal arrhythmias originating from the left ventricular summit, which is the most superior portion of the left ventricle, including the epicardial surface between the proximal left anterior descending and left circumflex coronary arteries. The great cardiac vein bisects the left ventricular summit into (1) a superior portion that is in close proximity to the proximal coronary arteries and beneath epicardial fat and is inaccessible to catheter ablation and (2) an inferior portion that is accessible to catheter ablation. Catheter ablation can be successfully performed within the great cardiac vein, which lies deep in the epicardial fat or in the accessible area below the great cardiac vein, where the epicardial fat is less prominent. A ventricular prepotential is usually recorded at the site of successful ablation. Origin from the inaccessible region explains failure of catheter ablation for some focal left ventricular arrhythmias.
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