Challenging Radiofrequency Catheter Ablation of Idiopathic Ventricular Arrhythmias Originating From the Left Ventricular Summit Near the Left Main Coronary Artery

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**Background**—Radiofrequency catheter ablation (RFCA) of idiopathic ventricular arrhythmias (VAs) originating from the basal portion of the left ventricular (LV) summit, which is divided from the apical LV (A-LV) summit by the great cardiac vein (GCV), is challenging. This study investigated the efficacy of RFCA and electrocardiographic and electrophysiological characteristics of these VAs.

**Methods and Results**—Forty-five consecutive patients with symptomatic idiopathic LV summit VAs were studied. RFCA was successful within the main trunk of the GCV in 16 patients and within a branch of the GCV traversing the basal LV (B-LV) summit in 7. Transpericardial RFCA was successful on the epicardial surface in the A-LV summit in 6 patients and was abandoned in 14 with the B-LV summit VAs because of the close proximity to the coronary arteries and thick fat pads. RFCA was successful at the aortomitral continuity in 3 patients (2 with a failed transpericardial RFCA), and left coronary cusp in 1. The RFCA success rate of the A-LV summit VAs including the GCV VAs was 100% (22/22), whereas that of the B-LV summit VAs was 48% (11/23). The B-LV summit VAs could be differentiated from the A-LV summit VAs by left bundle branch block pattern, QRS duration ≤175 ms, precordial transition ≥V1, and maximum deflection index of ≥0.55.

**Conclusions**—This study revealed that ≥50% of the B-LV summit VAs could be eliminated by a direct approach through a GCV branch running below the proximal left coronary arteries and a remote approach from the adjacent endocardial sites. (*Circ Arrhythm Electrophysiol. 2016;9:e004202. DOI: 10.1161/CIRCEP.116.004202.*)

Key Words: catheter ablation ◼ coronary artery ◼ epicardium ◼ endocardium ◼ ventricular tachycardia

Diopathic ventricular arrhythmias (VAs) can arise from the epicardial surface of the left ventricle (LV). The most common site of origins of idiopathic epicardial VAs is the LV summit that anatomically lies superior to the aortic portion of the LV ostium and occupies the most superior portion of the LV. VAs originating from this region can be eliminated by radiofrequency catheter ablation (RFCA) applied within the great cardiac vein (GCV) or by using a transpericardial approach. However, when VAs originate from the basal portion of the LV summit near the left main coronary artery, catheter ablation of these VAs is challenging because of the close proximity to the coronary arteries and thick epicardial fat pad that covers that region. The techniques that can be used to successfully and safely ablating these basal LV summit VAs remain a major concern in the management of patients with these arrhythmias. Several techniques have been attempted to approach those LV summit VA origins, such as catheter ablation from the endocardial sites adjacent to the LV summit and through the GCV. However, the efficacy of RFCA using these multiple approaches remains unknown. The purpose of this study was to investigate the efficacy of such RFCA and the electrocardiographic and electrophysiological characteristics of the idiopathic VAs originating from the LV summit near the left main coronary artery.

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**Methods**

**Patient Characteristics**

The study population consisted of 45 consecutive patients from a single center (21 men, mean age 51±13 years [range, 18–74 years]) with symptomatic idiopathic sustained ventricular tachycardia (VT) (n=8), nonsustained VT (n=10), or premature ventricular contractions (PVCs; n=27), whose origins were identified in the LV summit. Echocardiography and exercise stress testing or coronary angiography demonstrated no evidence of structural heart disease in any patients. The baseline characteristics including age, sex, LV function, nature of the clinical arrhythmia, and 12-lead ECG during the VAs were recorded. The institutional review board approved the study protocol, and all patients provided written, informed consent for the procedure. All antiarrhythmic drugs were discontinued for at least 5 half-lives before the study.
Electrophysiological Study

For mapping and pacing, a quadripolar catheter was positioned via the right femoral vein at the His bundle region and a deflectable decapolar catheter in the coronary sinus. The coronary sinus catheter was advanced into the GCV as far as possible until the proximal electrode pair recorded an earlier ventricular activation than the most distal electrode pair during VTs.3,4 Mapping and pacing were performed using a 7.5F, 3.5-mm tip irrigated ablation catheter (Navistar ThermoCool; Biosense-Webster, Diamond Bar, CA) introduced from the right femoral vein (for the right ventricle and GCV) or right femoral artery (for the endocardial LV outflow tract [LVOT]). As a guide for mapping within the GCV, an angiogram of the GCV was obtained by a contrast injection through an Amplatz angiographic catheter and/or the irrigation lumen of the ablation catheter3 (Figure 1). The initial 10 patients with a successful RFCA within the GCV in the previous study1 were excluded from this study because a GCV angiogram was not obtained in those patients.

During the procedures in the endocardial LVOT, intravenous heparin was administered to maintain an activated clotting time of >300 seconds. When few PVCs were observed at the beginning of the electrophysiological study, induction of the VT or PVCs was attempted by burst ventricular pacing with the addition of an isoproterenol infusion.

Activation mapping was performed in all cases to identify the earliest site of ventricular activation during the VT or PVCs. Pace mapping was performed using the distal bipolar electrodes at a pacing cycle length of 500 ms and at the minimum stimulus amplitude required for consistent capture (up to a maximum output of 20 mA and pulse width of 2.0 ms). The pace map score was determined as the number of leads with identical heights of the R wave/depth of the S wave (R/S) ratio match (12 represented a perfect R/S ratio match in all 12 leads) and the number of leads with fine notching matches in the 12-lead ECG as previously reported (perfect pace mapping was equal to 24 points). An excellent pace map was defined as a pace map with a score of ≥20.

When the earliest ventricular activation preceded the QRS onset by at least 20 ms and/or an excellent pace map was demonstrated within the GCV, one irrigated radiofrequency (RF) application was delivered at this site. If RF ablation at this site transiently suppressed the VTs, a couple of RF applications were added at adjacent sites. When there were no sites with such an early activation or excellent pace map within the GCV, epicardial mapping via a subxiphoid approach was performed with the irrigated ablation catheter to seek an earlier ventricular activation or excellent pace map on the LV epicardial surface as previously reported.2–4 When a more suitable site for ablation was identified on the epicardial surface, irrigated RF ablation was delivered at that site. If the first irrigated RF ablation from the GCV was unsuccessful, a second irrigated RF ablation was delivered at the site with the earliest

**WHAT IS KNOWN**

- Radiofrequency catheter ablation of idiopathic VTs originating from the basal portion of the LV summit, which is divided from the apical LV summit by the GCV, is challenging.
- Several techniques have been attempted to approach these LV summit VT origins such as catheter ablation from the endocardial sites adjacent to the LV summit and through the GCV.
- The efficacy of radiofrequency catheter ablation using these multiple approaches remains unknown.

**WHAT THE STUDY ADDS**

- Approximately 50% of the basal LV summit VTs could be eliminated by a direct approach through a GCV branch running below the proximal left coronary arteries and a remote approach from endocardial sites such as the aortomitral continuity and left coronary cusp.
- Basal LV summit VTs could be differentiated from apical LV summit VTs by a left bundle branch block pattern, QRS duration ≤175 ms, precordial transition later than or equal to V1, and maximum deflection index of ≥0.55.

**Figure 1.** Fluoroscopic images exhibiting the coronary sinus (CS) venogram (A) and venogram of a communicating branch (CB) of the great cardiac vein (GCV; B) and left coronary arteriogram (C) with the ablation catheter at the successful ablation site. The venograms were obtained by a contrast injection through an Amplatz angiographic catheter (A) and the irrigation lumen of the ablation catheter (B). Top and bottom, Left and right anterior oblique projections. Note that the communicating branch of the GCV was located lower than the distal part of the GCV and anterior interventricular vein (AIVV) because the communicating branch runs on the epicardial surface, whereas the GCV runs through or on the epicardial fat that covers the epicardial surface. ABL indicates ablation catheter; and HB, His bundle.
ventricular activation on the endocardial side (from the aortomitral continuity [AMC] and/or left coronary cusp [LCC]).

Irrigated RF ablation was delivered in the power-control mode starting at 20 W in the GCV and 30 W at the AMC, LCC, and on the epicardial surface with irrigation flow rates of 30 mL/min. The RF power was titrated ≤30 and 50 W, respectively. The goal of the RF applications was to achieve a decrease in the impedance of 8 to 10 Ω and with care taken to limit the temperature to <45 °C. During the epicardial RFCA using transvenous and transpericardial approaches, simultaneous left coronary angiography was performed to ensure the location of the ablation catheter relative to the left coronary arteries and to detect evidence of thermal injury to the vessel (Figure 1). An RF application was avoided within 5 mm of a coronary artery. This was confirmed by a coronary angiogram showing that the ablation catheter was >5 mm away from the coronary artery in either the right anterior oblique or left anterior oblique projection. When an acceleration or reduction in the frequency of the VT or PVCs was observed during the first 10 seconds at the AMC and LCC and 20 seconds in the GCV of the application after the RF power was titrated up to the maximum target, the RF delivery was continued for 30 to 60 seconds. Otherwise, the RF delivery was terminated, and the catheter was repositioned. The end point of the catheter ablation procedure was the elimination and noninducibility of VT or PVCs during an isoproterenol infusion (4 μg/min) and burst pacing from the right ventricle (to cycle lengths as short as 300 ms).

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 left anterior descending coronary artery (LAD) superior to the first septal perforating branch anteriorly to the left circumflex coronary artery laterally (Figure 1). Thus, the radius of this arc was the distance from the bifurcation of the left coronary arteries to the first septal perforator. The main trunk of the GCV bisects the LV summit into a basal portion that is in close proximity to the proximal left coronary arteries with a thick overlying epicardial fat and an apical portion that may be accessible to epicardial catheter ablation. The basal and apical portions of the LV summit have been defined as the inaccessible and accessible areas, respectively, in a previous study,3 but they were defined as the basal and apical areas, respectively, in this study because the basal portion of the LV summit was found to be accessible in some cases. The main trunk of the GCV was included in the apical area of the LV summit for the analysis.

Definition of the LV Summit VAs
A recent study revealed the presence of LVOT VAs originating from an intramural origin.3 These VAs could be successfully eliminated by sequential or simultaneous unipolar RFCA from the AMC and GCV. This study excluded these intramural LVOT VAs to focus on only VAs originating from epicardial sites. Therefore, in this study, LV summit VAs were defined as LVOT VAs with the earliest ventricular activation on the epicardial side and with the earliest endocardial ventricular activation relative to the QRS onset later than −10 ms.9

Electrocardiographic Analysis
Simultaneous 12-lead electrocardiograms during VAs and pace mapping were recorded digitally at sweep speeds of 200 mm/s in all patients for an offline analysis. The QRS morphologies including a bundle branch block pattern, the axis, and the configuration in leads I and V₅ were examined. In lead I, the presence of an R wave was the main concern because the absence of R waves suggested a VA origin located in the LV free wall. In lead V₅, the main concern was the presence of S waves, which were considered to be a characteristic and convenient electrocardiographic finding of AMC VAs probably because the S waves in lead V₅ are consistent with a right bundle branch block pattern, usually present in VAs with LV endocardial origins.

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 maximum deflection time was measured from the QRS onset to the maximum 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 the maximum deflection in any precordial lead by the QRS duration.4 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.

Follow-Up
All patients with an acute success of the catheter ablation were followed up without any antiarrhythmic drugs. Follow-up after the procedure included clinic visits with 12-lead electrocardiograms, 24-hour Holter monitoring, and telephone calls to all patients and their referring physicians. All patients reporting symptoms underwent 24-hour Holter monitoring or event monitoring to document the cause of the symptoms.

Statistical Analysis
Continuous variables are expressed as the group mean±1 SD or median with the first and third quartile (Q1–Q3). Comparisons of the continuous variables between the 2 groups were analyzed with the Student t test or Mann–Whitney U test. The categorical variables expressed as numbers and percentages in the different groups were compared with a χ² test and Yates correction if necessary. The P values were 2 sided, and statistical significance was selected at P<0.05.

Results
Mapping and Ablation
RFCA was successful within the main trunk of the GCV in 16 patients and within a branch of the GCV in 7 patients (Figure 2). An impedance rise within the GCV branch was observed in only one case. In this case, at the site of the earliest ventricular activation, the catheter was wedged, and RF energy could not be delivered because of a high impedance. By slightly pulled back the ablation catheter, RF energy could be delivered, and the VA was successfully eliminated. Because an angiogram of the GCV revealed that the GCV branch ran through the basal area of the LV summit, the VAs successfully ablated within the GCV branch were considered basal LV summit VAs. RFCA within the GCV was unsuccessful in 2 patients, although the earliest ventricular activation preceding the QRS onset by 26 and 30 ms was recorded within the GCV, and the pace map at those sites was excellent. In one of these 2 patients, a high impedance limited the RF energy delivery within the GCV, which transiently suppressed the VAs. RFCA was then successful at the AMC below the basal area of the LV summit where the local ventricular activation relative to the QRS onset was ~4 ms (Figure 3). In the other patient, the QRS morphology had changed after the RF ablation within the GCV. RFCA within the LCC where the local ventricular activation was 12 ms later than the QRS onset suppressed the PVCs during the procedure, resulting in a late success. Transpericardial mapping was performed in 20 patients. Among those 20 patients, RFCA was successful on the epicardial surface in the apical area of the LV summit in 6 patients. Transpericardial RFCA was abandoned in 14 patients because the VA origins were identified at the basal area of the LV summit with close proximity to the coronary arteries and thick fat pads. In 2 of these 14 patients, RFCA was successful at the AMC below the basal area of the LV summit where the local ventricular activation relative to the QRS onset was 0 ms.

Only 1 patient required 2 procedures within 2 months to achieve a successful catheter ablation of the VAs within the GCV. All patients with an acute success of the catheter ablation of VAs have been free from any recurrence of the VAs during
Figure 2. A. Electrocardiograms exhibiting a premature ventricular contraction (PVC) and an excellent pace map (PM) at the successful ablation site within a communicating branch (CB) of the great cardiac vein (GCV) at the left ventricular summit (left) and cardiac tracings and fluoroscopic images exhibiting the successful ablation site (middle and right). B, Cardiac tracings exhibiting the successful ablation. ABL indicates ablation catheter; CS 1 to 5, the first (most distal) to fifth (most proximal) electrode pairs of the coronary sinus catheter; LAO, left anterior oblique view; HB, His bundle; RAO, right anterior oblique view; RF, radiofrequency; V-QRS, the local ventricular activation time relative to the QRS onset, and X d, p, the distal and proximal electrode pairs of the relevant catheter.
the follow-up period (median=55 months and Q1–Q3=35–71 months). The success rate of catheter ablation of VAs originating from the apical area of the LV summit including the GCV was 100% (22/22), whereas that of VAs originating from the basal area of the LV summit was 48% (11/23). No complications occurred.

Comparison of the Clinical Characteristics and Electrocardiographic and Electrophysiological Parameters Between the VAs Originating From the Basal and Apical Areas of the LV Summit

The clinical characteristics and electrocardiographic and electrophysiological parameters of the VAs originating from the basal LV summit that were successfully ablated are shown in Table 1. There were no significant differences in the clinical characteristics between the VAs originating from the basal and apical areas of the LV summit (Table 2). The electrocardiographic and electrophysiological parameters are compared between the VAs originating from the basal and apical areas of the LV summit as shown in Table 3. A left bundle branch block (LBBB) pattern was prevalent during the VAs originating from the basal LV summit, whereas a right bundle branch block pattern was more common in those from the apical LV summit. The precordial transition was significantly later during the VAs originating from the basal LV summit than those from the apical LV summit ($P=0.044$). The QRS duration, R wave amplitude ratio in leads III to II, Q wave amplitude ratio in leads aVL to aVR, and local ventricular activation time relative to the QRS onset at the successful ablation site were significantly greater during the VAs originating from the apical LV summit than those from the basal LV summit ($P=0.037$, $0.012$, and $0.005$, respectively).

Comparing the LV summit VAs, an LBBB pattern, QRS duration ≤175 ms, precordial transition later than or equal to V1, and MDI of ≥0.55 predicted the VAs originating from the basal LV summit with a sensitivity of 65%, 87%, 74%, and 61%; specificity of 77%, 50%, 68%, and 64%; positive predictive accuracy of 75%, 65%, 71%, and 64%; and negative predictive accuracy of 68%, 79%, 71%, and 61%, respectively (Table 4).

Discussion

Major Findings

In the past decade, advances in the procedural equipment and techniques, and an improved understanding of anatomy and electrophysiological mechanisms, have allowed catheter ablation to cure most idiopathic VAs arising from the endocardium and epicardium. However, catheter ablation of idiopathic
Yamada et al  Idiopathic LV Summit VAs Arising From Near the LMT

VAs arising from the basal LV summit remains challenging because of anatomic barrier such as the left coronary arteries and thick overlying epicardial fat pads (Figure 4), and the basal LV summit has often been considered inaccessible for catheter ablation. This study revealed that ≈50% of the VAs origins in the basal LV summit could be eliminated by the direct approach through a branch of the GCV running below the bifurcation of the LAD and left circumflex coronary artery or a remote approach with a transmural RF application from endocardial sites such as the AMC and LCC.

In a previous study, several VAs that were successfully ablated within the GCV branch were included into the apical LV summit VAs. This study reevaluated the electrocardiographic and electrophysiological characteristics of the LV summit VAs by reclassifying cases from the previous study and adding new cases. The results of this study revealed that the electrocardiographic and electrophysiological characteristics of the basal and apical LV summit VAs were consistent with the anatomic background that the basal LV summit is located more septally and higher than the apical LV summit. The basal LV summit VAs were characterized by a dominance of an LBBB pattern, shorter QRS duration, greater R-wave amplitude in the inferior leads, smaller III/II and aVL/aVR ratios, later precordial transition, and earlier ventricular activation in the His bundle region during the VAs as compared with the apical LV summit VAs. When an early ventricular activation is recorded within the distal GCV during the VAs, an LBBB pattern, QRS duration ≤175 ms, precordial transition later than or equal to V1, and MDI of ≥0.55 could differentiate the basal LV summit VAs from the apical LV summit VAs, although the accuracy was relatively limited presumably because of the close anatomic relationship between the 2 sites.

Anatomic Consideration
An anatomic study has demonstrated the presence of a GCV branch running below the bifurcation of the LAD and left circumflex coronary artery. The GCV runs anteriorly along the mitral annulus and connects to the anterior interventricular vein that runs down along the LAD on the lateral side (Figure 4). A GCV branch that is termed the communicating

Table 1. Characteristics of the Patients With Successful Ablation of the Ventricular Arrhythmias Arising From the Basal LV Summit

<table>
<thead>
<tr>
<th>Case</th>
<th>ABL Site</th>
<th>Age</th>
<th>Sex</th>
<th>Type</th>
<th>QRS duration</th>
<th>QRS Morphology</th>
<th>I</th>
<th>aVR</th>
<th>aVL</th>
<th>TZ</th>
<th>V-QRS (PM)</th>
<th>No. of RF</th>
<th>Fluoro Time</th>
<th>CM</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>GCV branch</td>
<td>37</td>
<td>F</td>
<td>PVC</td>
<td>162</td>
<td>LR</td>
<td>QS</td>
<td>QS</td>
<td>QS</td>
<td>V2&gt;V3</td>
<td>−0.54</td>
<td>1</td>
<td>14</td>
<td>−</td>
</tr>
<tr>
<td>2</td>
<td>GCV branch</td>
<td>54</td>
<td>F</td>
<td>PVC</td>
<td>152</td>
<td>LR</td>
<td>QS</td>
<td>QS</td>
<td>QS</td>
<td>V2&gt;V3</td>
<td>−0.52</td>
<td>3</td>
<td>10</td>
<td>−</td>
</tr>
<tr>
<td>3</td>
<td>GCV branch</td>
<td>54</td>
<td>F</td>
<td>NSVT</td>
<td>172</td>
<td>RR</td>
<td>QS</td>
<td>QS</td>
<td>rS</td>
<td>&lt;V6</td>
<td>+0.45</td>
<td>1</td>
<td>14</td>
<td>−</td>
</tr>
<tr>
<td>4</td>
<td>GCV</td>
<td>51</td>
<td>F</td>
<td>PVC</td>
<td>175</td>
<td>LR</td>
<td>rs</td>
<td>QS</td>
<td>QS</td>
<td>V2&gt;V3</td>
<td>−0.56</td>
<td>1</td>
<td>8</td>
<td>−</td>
</tr>
<tr>
<td>5</td>
<td>GVC branch</td>
<td>60</td>
<td>F</td>
<td>PVC</td>
<td>171</td>
<td>LR</td>
<td>rs</td>
<td>QS</td>
<td>QS</td>
<td>V2&gt;V3</td>
<td>−0.46</td>
<td>3</td>
<td>15</td>
<td>−</td>
</tr>
<tr>
<td>6</td>
<td>GVC branch</td>
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<td>M</td>
<td>NSVT</td>
<td>168</td>
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<td>rs</td>
<td>QS</td>
<td>rS</td>
<td>V2</td>
<td>−0.62</td>
<td>2</td>
<td>32</td>
<td>−</td>
</tr>
<tr>
<td>7</td>
<td>GVC branch</td>
<td>25</td>
<td>F</td>
<td>NSVT</td>
<td>162</td>
<td>LR</td>
<td>rS</td>
<td>QS</td>
<td>rS</td>
<td>V2&gt;V3</td>
<td>−0.63</td>
<td>1</td>
<td>14</td>
<td>−</td>
</tr>
<tr>
<td>8</td>
<td>AMC</td>
<td>42</td>
<td>F</td>
<td>PVC</td>
<td>160</td>
<td>RR</td>
<td>rs</td>
<td>QS</td>
<td>QS</td>
<td>&lt;V6</td>
<td>+0.51</td>
<td>8</td>
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<td>−</td>
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<tr>
<td>9</td>
<td>LCC</td>
<td>72</td>
<td>M</td>
<td>NSVT</td>
<td>150</td>
<td>RR</td>
<td>QS</td>
<td>QS</td>
<td>QS</td>
<td>&lt;V6</td>
<td>−0.53</td>
<td>12</td>
<td>37</td>
<td>−</td>
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<tr>
<td>10</td>
<td>AMC</td>
<td>68</td>
<td>M</td>
<td>PVC</td>
<td>170</td>
<td>RR</td>
<td>rsrs</td>
<td>QS</td>
<td>QS</td>
<td>&lt;V6</td>
<td>−0.49</td>
<td>0</td>
<td>17</td>
<td>+</td>
</tr>
<tr>
<td>11</td>
<td>AMC</td>
<td>60</td>
<td>M</td>
<td>SVT</td>
<td>137</td>
<td>LR</td>
<td>rs</td>
<td>QS</td>
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<td>QS</td>
<td>−0.47</td>
<td>5</td>
<td>20</td>
<td>−</td>
</tr>
</tbody>
</table>

ABL indicates ablation; AMC, aortomitral continuity; CM, cardiomyopathy; F, female; GCV, great cardiac vein; LCC, left coronary cusp; LR, left bundle branch block+right axis; LV, left ventricle; M, male; MDI, maximum deflection index; N/A, not available; NSVT, nonsustained ventricular tachycardia; PM, pace map score; PVC, premature ventricular contraction; RF, radiofrequency applications; RR, right bundle branch block+right axis; SVT, sustained ventricular tachycardia; TZ, transition zone; and V-QRS, the local ventricular activation time relative to the QRS onset.

Table 2. The Basic Demographics and Clinical Characteristics

<table>
<thead>
<tr>
<th>Basal Summit (n=23)</th>
<th>Apical Summit (n=22)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>51±14</td>
<td>51±13</td>
</tr>
<tr>
<td>Male sex, n (%)</td>
<td>10 (43)</td>
<td>11 (50)</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>M=62, Q1–Q3=56.5–68.5</td>
<td>M=61, Q1–Q3=57–66.3</td>
</tr>
<tr>
<td>Tachycardia-induced CM, n (%)</td>
<td>4 (17)</td>
<td>5 (23)</td>
</tr>
</tbody>
</table>

Clinical arrhythmia, n (%) 0.834

Data are expressed as the mean±SD, number with the percentage, or median with the first and third quartiles (Q1–Q3). CM indicates cardiomyopathy; LVEF, left ventricular ejection fraction; PVC, premature ventricular contraction; and VT, ventricular tachycardia.

Reference
1. An anatomic study has demonstrated the presence of a GCV branch running below the bifurcation of the LAD and left circumflex coronary artery. The GCV runs anteriorly along the mitral annulus and connects to the anterior interventricular vein that runs down along the LAD on the lateral side (Figure 4). A GCV branch that is termed the communicating
branch begins from the junction between the GCV and anterior interventricular vein, travels through the basal LV summit below the bifurcation of the LAD and left circumflex coronary artery, and connects to the conus branch on the right side (Figure 4).10 Because the communicating branch runs on the epicardial surface, there is no significant fat between the branch and epicardial surface, and RFCA within this branch can often eliminate basal LV summit VAs. On the contrary, because the distal part of the GCV and anterior interventricular vein run through or on the epicardial fat, which covers the epicardial surface, RFCA within these veins may be limited by a high impedance. There may be a concern about safety during catheter ablation within this branch because of the close proximity to the left main coronary artery. The proximal portion of the left coronary arteries is covered by a thick fat pad that limits the efficacy of transpericardial RFCA. The fat pad also infiltrates underneath the left main coronary artery (Figure 4). Because the fat pad with a high impedance lies between the left main coronary artery and the communicating branch, RFCA within the communicating branch could be safely performed without any damage to the coronary artery.

When LV summit VA origins are located between the LCC and communicating branch, a remote approach from the LCC and AMC would be an alternative for the catheter ablation of basal LV summit VAs. The superior end of the LV myocardium attaches to the LCC with the basal LV summit on the epicardial side and AMC on the endocardial side. If the basal LV summit VA origins are located near the LCC, RFCA within the LCC could reach those VA origins and eliminate them. The LV myocardium tapers toward its superior end. Therefore, if a transmural RF lesion can be created from the AMC, it could reach those VA origins and eliminate the basal LV summit VAs.

Previous Studies
Previous studies reported the efficacy of endocardial RF ablation from the LCC and AMC in the catheter ablation of LV summit VAs.16 However, in one of them, epicardial catheter ablation through the GCV or a transpericardial approach was never performed.17 The other study included a significant number of cases with intramural LVOT VAs because

Table 3. The Comparison of the Electrocardiographic and Electrophysiological Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Basal Summit (n=23)</th>
<th>Apical Summit (n=22)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total QRS duration of the VA, ms</td>
<td>161 (153–169)</td>
<td>177 (167–187)</td>
<td>0.032</td>
</tr>
<tr>
<td>QRS morphology, n (%)</td>
<td></td>
<td></td>
<td>0.006</td>
</tr>
<tr>
<td>LBBB+LIA</td>
<td>1 (4)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>LBBB+RIA</td>
<td>14 (61)</td>
<td>5 (23)</td>
<td></td>
</tr>
<tr>
<td>RBBB+LIA</td>
<td>2 (9)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>RBBB+RIA</td>
<td>6 (26)</td>
<td>17 (77)</td>
<td></td>
</tr>
<tr>
<td>Precordial transition, n (%)</td>
<td></td>
<td></td>
<td>0.044</td>
</tr>
<tr>
<td>≤V₁</td>
<td>7 (30)</td>
<td>15 (68)</td>
<td></td>
</tr>
<tr>
<td>V₂</td>
<td>2 (9)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>V₂−V₃</td>
<td>13 (57)</td>
<td>7 (32)</td>
<td></td>
</tr>
<tr>
<td>Maximum R-wave amplitude in the inferior leads, mV</td>
<td>M=1.44, Q1–Q3=1.22 to 1.60</td>
<td>M=1.21, Q1–Q3=0.93 to 1.44</td>
<td>0.037</td>
</tr>
<tr>
<td>R-wave amplitude ratio in leads III to II</td>
<td>M=1.08, Q1–Q3=0.99 to 1.18</td>
<td>M=1.22, Q1–Q3=1.09 to 1.35</td>
<td>0.012</td>
</tr>
<tr>
<td>Maximum deflection index</td>
<td>0.56±0.06</td>
<td>0.51±0.08</td>
<td>0.012</td>
</tr>
<tr>
<td>S wave in lead V₆, n (%)</td>
<td>5 (22)</td>
<td>9 (41)</td>
<td>0.165</td>
</tr>
</tbody>
</table>

V-QRS, ms

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>PPV, %</th>
<th>NPV, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left bundle branch block</td>
<td>65</td>
<td>77</td>
<td>75</td>
<td>68</td>
</tr>
<tr>
<td>QRS duration ≤175 ms</td>
<td>87</td>
<td>50</td>
<td>65</td>
<td>79</td>
</tr>
<tr>
<td>Transition zone ≥V₁</td>
<td>74</td>
<td>68</td>
<td>71</td>
<td>71</td>
</tr>
<tr>
<td>Maximum deflection index ≥0.55</td>
<td>61</td>
<td>64</td>
<td>64</td>
<td>61</td>
</tr>
</tbody>
</table>

LV indicates left ventricle; NPV, negative predictive value; PPV, positive predictive value; and VA, ventricular arrhythmia.
the averaged earliest endocardial ventricular activation was 
−20 ms.6 In this study, the endocardial RF ablation was performed only when all possible epicardial approaches were unsuccessful, and intramural LVOT VAs were excluded. Therefore, we think that this study revealed the real outcome of the RFCA of basal LV summit VAs by using currently available approaches.

**Conclusions**

This study revealed that ≥50% of the basal LV summit VAs could be eliminated by a direct approach through a GCV branch running below the proximal left coronary arteries and a remote approach from endocardial sites such as the AMC and LCC. Basal LV summit VAs could be differentiated from apical LV summit VAs by an LBBB pattern, QRS duration ≤175 ms, pre-cordial transition later than or equal to V7, and MDI of ≥0.55.

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**Disclosures**

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**References**

Challenging Radiofrequency Catheter Ablation of Idiopathic Ventricular Arrhythmias Originating From the Left Ventricular Summit Near the Left Main Coronary Artery
Takumi Yamada, Harish Doppalapudi, Silvio H. Litovsky, H. Thomas McElderry and G. Neal Kay

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