Ventricular Arrhythmias Arising From the Left Ventricular Outflow Tract Below the Aortic Sinus Cusps
Mapping and Catheter Ablation via Transseptal Approach and Electrocardiographic Characteristics

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**Background**—Ventricular arrhythmias (VAs) originating from the anterosuperior left ventricular outflow tract (LVOT) represent a challenging location for catheter ablation. This study investigates mapping and ablation of VA from anterosuperior LVOT via a transseptal approach.

**Methods and Results**—This study included 27 patients with symptomatic VA, of which 13 patients had previous failed ablations. LVOT endocardial 3-dimensional mapping via retrograde transaortic and antegrade transseptal approaches was performed. Previous ECG markers for procedure failure were analyzed. In all patients, earliest activation with low-amplitude potentials was identified at the anterosuperior LVOT 5.1±2.8 mm below the aortic cusp and preceded the QRS onset by 39.5±7.7 ms only via an antegrade transseptal approach using a reversed S curve. In all patients, pace mapping failed to demonstrate perfect QRS morphology match. The anatomic location was below the left coronary cusp in 16, below the left coronary cusp/right coronary cusp junction in 8, and below the right coronary cusp in 3 patients. Radiofrequency energy resulted in rapid disappearance of VAs in all patients. ECG analysis showed aVL/aVR Q-wave amplitude ratio >1.4 in 7, lead III/II R-wave amplitude ratio >1.1 in 10, and peak deflection index >0.6 in 11 patients. There were no complications or clinical VA recurrence during a mean follow-up of 8.4±2.5 months.

**Conclusions**—The anterosuperior LVOT can be reached via a transseptal approach with a reversed S curve of the ablation catheter. The rapid effect from radiofrequency energy indicates that the VA is most likely located under the endocardium. Also, previous ECG markers for procedure failure need further investigation.

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**Key Words:** ablation ■ catheters ■ mapping ■ ventricular arrhythmias

Most ventricular arrhythmias (VAs) in patients without structural heart disease originate from the right ventricular outflow tract (RVOT) or left ventricular outflow tract (LVOT).1–4 Catheter ablation in these anatomic areas has been established as an effective and curative therapy.1–8 In some cases, VA arises from an area in the LVOT located anterosuperior to the aortic-mitral continuity, termed the left ventricular summit (LVS).5,6,9 Ablation of this type of VA is still challenging. Epicardially, it is bound by the left anterior descending artery, left circumflex artery, and the distal part of the great cardiac vein (GCV).9 The anatomic region superior to the GCV has been previously described as an area inaccessible to catheter ablation and the area inferolateral to the GCV as more accessible via epicardial ablation.9 We sought to determine mapping, ablation, and ECG characteristics of VA originating from the anterosuperior LVOT via an antegrade transseptal approach.

**Clinical Perspective on p 455**

**Methods**

**Study Population**
In 2012 to 2013, 27 consecutive patients with symptomatic VAs and with ECGs suggestive of VAs arising from the anterosuperior LVOT underwent radiofrequency ablation at 3 centers: 13 patients at

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Electrophysiological Study

The study with both transaortic and transseptal approaches was approved by our institutional review committee, and all patients provided written informed consent before the procedure. After withdrawal of antiarrhythmic drugs for ≥5 half-lives, all patients underwent electrophysiological evaluation under sedation with intravenous propofol at Asklepios Klinik, Hamburg, in 4 patients and without sedation in the remaining 23 patients. Catheters were placed in the right ventricle via femoral veins and within the distal coronary sinus (CS) via femoral or left subclavian veins. Twelve-lead surface ECGs and intracardiac electrograms were recorded simultaneously by a digital multichannel system (EPMed, St Jude Medical), filtered at 30 to 400 Hz for bipolar electrograms and at 0.05 to 400 Hz for unipolar electrograms. If clinical arrhythmias failed to occur spontaneously, programmed stimulation was performed. The standard protocol consisted of ventricular stimulation at 2 basic drive cycle lengths with ≤2 extrastimuli to a minimum coupling interval of 230 ms. If VA was not inducible at baseline, intravenous isoproterenol infusion (2–5 μg/min) was administered to provoke clinical arrhythmias.

Three-Dimensional Mapping Strategy of the Cardiac Chambers

In patients with frequent premature ventricular contractions (PVC) or ventricular tachycardia, 3-dimensional (3D) electroanatomic mapping of the RVOT and GCV was initially performed via the CS. LV access was achieved via a retrograde transaortic and antegrade transseptal approach in all patients. Transseptal puncture was performed through the anterosuperior LVOT. Transseptal sheaths (SL1 and 1B). In our experience, this approach facilitates access into the LV, in particular to the anterosuperior LVOT. Transseptal sheaths (SL1) were continuously flushed with heparinized saline at 20 mL/h. During the procedure, unfractionated heparin was administered to maintain activated clotting times between 250 and 300 seconds.

Electroanatomic mapping methodology has been previously described.10 In brief, mapping was performed using a steerable 7.5F, D-curve catheter with a 3.5-mm irrigated-tip electrode (Navistar ThermoCool; Biosense Webster, Diamond Bar, CA). Point-by-point mapping was performed to create anatomic maps of the RVOT and GCV via the CS. Earliest ventricular activation was annotated in each chamber. When GCV access was difficult, contrast medium was injected through the irrigated catheter tip to define the course of the CS and facilitate advancement into the GCV.

Complete anatomic mapping of the aortic root annotating the earliest ventricular activation in the right coronary cusp (RCC) or left coronary cusp (LCC) always preceded earliest ventricular activation obtained during LV mapping. Two important clinical reasons led to this strategy: (1) Anatomic distance between the aortic sinus cusp (ASC) and endocardial LVOT should theoretically be <12 mm, unless myocardial hypertrophy is present on echocardiography before ablation. This value was arbitrarily chosen because myocardial thickness >12 mm is considered hypertrophic. If the distance is >12 mm, it indicates that the anterosuperior LVOT was inadequately mapped. (2) During aortic root mapping, the site of earliest ventricular activation in the RCC or LCC helps to locate the anatomic origin below the ASC during LV mapping.

LV mapping was performed initially via the transaortic approach. With this approach, LVOT access was generally difficult, particularly the anatomic area just below the right and left ASC. This was also demonstrated on 3D mapping, with a large distance between the aortic root and LV. In contrast, access into the anterosuperior LVOT below the ASCs can be achieved via the transseptal approach using a reversed S curve of the ablation catheter (Figure 2). Once the mapping catheter was below the ASCs, clockwise and counterclockwise rotation manipulates it toward the RCC and LCC, respectively (Figure 2A–2D). During the procedure, the time from the onset of the surface QRS morphology to the peak sharp deflection in a predefined lead was measured. Subsequently, all measurements of activation timing throughout the procedure were then measured from the onset of the ventricular activation to this peak sharp deflection of the surface ECG to identify the site of earliest activation. The timing from the onset of the ventricular activation to the onset of the QRS is then calculated. Earliest ventricular activation in the anterosuperior LVOT was again annotated on 3D electroanatomic mapping. If the overall site of earliest ventricular activation was found below the ASC, angiography was performed to identify the exact anatomic location. Thus, anatomic sites of VA origins were defined by a combination of aortic root/coronary angiography and 3D mapping. In addition, the distance between the site of successful ablation within the LVOT identified by

Figure 1. Fluoroscopic views (right anterior oblique [RAO] and left anterior oblique [LAO]) demonstrating transseptal puncture through the anteroinferior fossa ovals in a patient with left ventricular outflow tract ventricular arrhythmias. Note that the transseptal puncture is performed at the anterior third between the aorta and lateral shadow of the right atrium (yellow line) and is slightly posterior to the mapping catheter (Map) in the noncoronary sinus cusp (NCC). The black line outlines the heart shadow. Red lines mark the aortic sinus cusps. CS indicates coronary sinus catheter; and TP, transseptal puncture.
the earliest activation during PVC/VA mapping and the closest portion of the ASCs was measured offline.

To visualize the local ventricular electrogram potentials, intracardiac electrograms were amplified, and sites of VA origins were identified by earliest ventricular activation on bipolar recordings during clinical VAs. In addition, pace mapping from bipolar electrodes was performed at sites of earliest ventricular activation. During pace mapping, the lowest pacing output (2–20 mA) and pulse width (0.5–10 ms) were used to capture the ventricular myocardium.

Irrigated Radiofrequency Ablation
Irrigated radiofrequency current was delivered in temperature-controlled mode, with a maximum power of 40 W, temperature limit of 43°C, and flush rate of 20 mL/min. Radiofrequency application was immediately stopped if catheter dislodgement occurred or if clinical PVC/VAs were still present 20 seconds after start of ablation. Time until maximum power and disappearance of clinical VAs was recorded. A safety radiofrequency application, applying the same radiofrequency settings used during the initial successful energy delivery, was deployed in all patients. After successful ablation, intravenous administration of isoprenol and programmed stimulation were performed to reprovoke clinical VAs.

QRS Morphology on Surface ECG During Clinical VAs
Detailed analysis of clinical PVC/VAs was performed offline using either the EPMed System (St Jude Medical) with a recording speed of 50 to 100 mm/s or the 12-lead ECG with a recording speed of 25 to 50 mm/s.

The following parameters were analyzed during clinical PVCs or the first beat of ventricular tachycardia: (1) QRS duration; (2) R-wave amplitudes of inferior leads (II, III, aVF) and R-wave amplitude ratio of lead III/II; (3) QS wave amplitudes in leads aVL and aVR and the ratio of aVL/aVR; (4) peak deflection index (PDI) in inferior leads, defined as duration from QRS onset on surface ECG to latest peak in the inferior leads/total QRS duration; and (5) number and percentage of patients with QS wave ratio of aVL/aVR >1.4, R-wave ratio of lead III/II >1.1, and PDI >0.6. All measurements were independently performed by 3 physicians.

Procedure Success and Follow-Up
Ablation success was defined as (1) absence of spontaneous or provoked clinical VAs at end of procedure, and (2) absence of the latter on 48-hour ECG monitoring postablation off antiarrhythmic drugs. VA burden was documented on 24-hour Holter monitoring before and after ablation. Transthoracic echocardiogram was performed immediately before discharge and 3 and 6 months after ablation in all patients. Follow-up was performed either by referring physicians or in outpatient clinics.

Statistical Analysis
Continuous variables are expressed as mean±SD.
Results

Study Population
Patient and clinical data are shown in Table 1. No structural heart disease was found, except in 2 patients (7.4%) who had a history of coronary artery disease with no disease progression before ablation. No patient had LV hypertrophy on echocardiography. All patients were refractory to ≥1 antiarrhythmic drug before ablation. A history of amiodarone therapy was documented in 2 (7.4%) patients, which was ineffective and stopped 6 months before ablation. Before the index procedure, 13 of 27 (48.1%) patients had failed ablation procedures (1 ablation attempt in 8, 2 attempts in 3, 3 attempts in 1, and 4 attempts in 1 patient). Previously failed targets are shown in Table 1. In these 13 patients with failed ablation, only 3 patients had previously failed ablation attempts at Asklepios Klinik St. Georg. Also, the failed ablations in the LVOT were performed with only the transaortic approach.

Mapping and Ablation
In 27 patients, frequent PVCs or short runs of ventricular tachycardia were present during the entire procedure. Although there was a slight reduction in the frequency of PVCs in the 4 patients ablated with sedation, PVCs still occurred frequently. Therefore, intravenous isoproterenol was not administered before LV mapping.

In the retrograde transaortic maps, closest anatomic distance between the ASCs and LVOT was >12 mm in all 27 patients (Figure 3A and 3B). Complete LVOT mapping was only achieved via transseptal approach (Figure 3C and 3D). No area with pathological potentials and low amplitudes was found below the ASCs during sinus rhythm. On bipolar recording, earliest ventricular activation was identified in the LVOT, anteroseptal to the aortic-mitral continuity, preceding the QRS onset by 39.5±7.7 ms during clinical VAs (Figures 4 and 5; Table 2), whereas ventricular activation on unipolar recordings demonstrated QS morphology with delayed activation compared with bipolar recordings (Figure 4). During sinus rhythm, a small atrial potential was always seen before the ventricular activation (Figure 5). Furthermore, before ablation, pace mapping was performed at the site of earliest ventricular activation, demonstrating mismatch of the QRS morphology compared with the clinical VA in all 27 patients (Figure in the Data Supplement).

Three-dimensional mapping and aortic angiography demonstrated that the anatomic location of VAs was below the LCC in 16 of 27 patients (59.3%; Figure 4B), below the RCC/LCC junction in 8 of 27 patients (29.6%; Figure 5), and below the RCC in 3 of 27 (11.1%) patients. Mean distance from the site of earliest ventricular activation in the LVOT identified via transseptal approach to the closest portion of the ASC was 5.1±2.8 mm. VA originating below the LCC had a mean distance of 7.4±3.5 mm to the LCC and that originating below the LCC/RCC junction and below the RCC had a mean distance of 3.8±0.9 mm and 3.7±1.8 mm, respectively (Table 2).

Irrigated Radiofrequency Ablation
Radiofrequency energy used and clinical effects during radiofrequency ablation are shown in Table 2. Radiofrequency energy was applied only via transseptal approach in these 27 patients. No patient had radiofrequency-induced VA or vagal reaction during ablation. Mean time to clinical VA disappearance was 5.5±3 seconds, with mean maximum power of 38.5±5.8 W (Table 2). Time to maximum power at the site of VA origin was 12.1±7.5 seconds in temperature-controlled mode (Table 2). The VAs were successfully abolished with a mean of 2.3±1.4 radiofrequency applications. No ST-segment or T-wave change was observed during ablation in any of the 27 patients. Procedure time was 127±57 minutes with fluoroscopy time of 12.3±6.4 minutes.

During the ablation procedure, PVCs with a completely different morphology were rarely observed and were only seen in 2 of 27 patients and were not targeted for ablation.

ECG Analysis
During clinical VA, right bundle branch block morphology was present in 3 of 27 patients (11.1%), and in these patients, VA origin was below the LCC (Figure 6A). Left bundle branch block morphology with an early transition at V6 through V1 was seen in the remaining 24 patients (88.9%) (Figure 6B and 6C). During clinical VA, no S wave on V5 and V1 leads was seen in any patient. All patients presented with inferior axis and high R waves in leads II, III and aVF and deep Q waves in aVL and aVR. Mean QRS duration was 157±24 ms with PDI of 0.58±0.06. Detailed analysis

Table 1. Patient Data and Basic Characteristics

<table>
<thead>
<tr>
<th>No. of patients</th>
<th>27</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>47±15 (12–68)</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>16/27 (59.3%)</td>
</tr>
<tr>
<td>Clinical symptoms</td>
<td></td>
</tr>
<tr>
<td>Palpitations</td>
<td>27/27 (100%)</td>
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<tr>
<td>Pre-syncope</td>
<td>1/27 (3.7%)</td>
</tr>
<tr>
<td>Syncpe</td>
<td>0/27 (0%)</td>
</tr>
<tr>
<td>Structural heart disease</td>
<td></td>
</tr>
<tr>
<td>Only PVC</td>
<td>21/27</td>
</tr>
<tr>
<td>PVC, nonsustained VT</td>
<td>4/27</td>
</tr>
<tr>
<td>PVC, nonsustained/sustained VT</td>
<td>2/27</td>
</tr>
<tr>
<td>Left ventricular EF (%) before ablation</td>
<td>57±11</td>
</tr>
<tr>
<td>No of patients left ventricular EF &lt; 50%</td>
<td>5/27 (18.5%)</td>
</tr>
<tr>
<td>No of previously failed ablation</td>
<td>13/27 (48.1%)</td>
</tr>
<tr>
<td>Previously failed targets</td>
<td></td>
</tr>
<tr>
<td>No. of patients at RVOT</td>
<td>7</td>
</tr>
<tr>
<td>No. of patients at LCC</td>
<td>5</td>
</tr>
<tr>
<td>No. of patients at GCV</td>
<td>5</td>
</tr>
<tr>
<td>No. of patients at LVOT</td>
<td>6</td>
</tr>
<tr>
<td>No. of patients at RCC</td>
<td>1</td>
</tr>
</tbody>
</table>

Ventricular arrhythmia burden before ablation 24/48±13385/24 h

EF indicates ejection fraction; GCV, great cardiac vein; LCC, left coronary cusp; LVOT, left ventricular outflow tract; PVC, premature ventricular contractions; RCC, right coronary cusp; RVOT, right ventricular outflow tract; and VT, ventricular tachycardia.
of inferior lead R-wave amplitudes and Q waves in aVR and aVL, as well as lead III/II R-wave amplitude ratio >1.1, aVL/aVR Q-wave amplitude ratio >1.4, and PDI >0.6, is shown in Table 3.

In summary, all patients had an inferior axis with high R waves in leads II, III, and aVF and deep Q waves in aVL and aVR. Nineteen of 27 patients (70.4%) had greater R-wave deflections in lead III compared with lead II, and of these 19 patients, 18 had VA origins below the LCC or LCC/RCC junction. A lead III/II R-wave amplitude ratio >1.1 was observed in 10 of 27 (37.0%) patients, and aVL/aVR Q-wave amplitude ratio >1.4 was noted in 7 of 27 (25.9%) patients. A higher amplitude ratio was found in patients with VA origins below the LCC and LCC/RCC junction, and the lowest amplitude ratio was noted if the site of origin was below the RCC. In addition, PDI >0.6 was found in 11 (40.7%) patients, in whom VAs were located below the LCC in 8, below the LCC/RCC junction in 2, and below the RCC in 1 patient.

Follow-Up

No complications occurred during or after ablation procedures. During a mean follow-up of 8.4±2.5 months, no patients received antiarrhythmic drug therapy. No clinical PVC/VA recurrence was seen on 48-hour Holter monitor after ablation, whereas the mean PVC burden was 78±148/24 hours 3 months after ablation. Furthermore, in patients with LV ejection fraction <50% before ablation, LV ejection fraction on transthoracic echocardiogram normalized 3 months.
Figure 4. Activation mapping, fluoroscopy, and 3-dimensional mapping in a 42-year-old man with 2 previous failed ablation attempts in the right ventricular outflow tract (RVOT) and aortic-mitral continuity. A, Surface ECG leads I, III, V1, and V2, and intracardiac recordings from a catheter within the distal coronary sinus catheter (CS) and mapping catheter at the site of earliest ventricular activation in 6 different sites. From the posteroseptal RVOT, the 3 aortic sinus cusps, great cardiac vein (GCV), and successful ablation site below the left coronary cusp (LCC). Note that (1) earliest ventricular activation relative to the stable reference taken from the peak R wave of lead III during clinical premature ventricular contractions (PVC); (2) the earliest ventricular activation is located at the left ventricular outflow tract (LVOT) below the LCC; (3) unipolar timing is delayed compared with bipolar recordings at the successful ablation site below the LCC; (4) local unipolar potentials at all 6 sites demonstrate QS morphology during ventricular arrhythmias. B. Left (B1 and B2), Right (30°) and left (45°) oblique radiographic views of the mapping catheter (Map) at the successful ablation site just below the LCC during left coronary angiography via a 5F Judkins catheter. Right (B3 and B4), Electroanatomic maps of the same male patient demonstrate the successful ablation point (red tag) just below the LCC. L indicates left coronary cusp; LAD, left anterior descending artery; LAO, left anterior oblique; LCX, left circumflex artery; LL, left lateral; LV, left ventricle; NCC, noncoronary sinus cusp; R, right coronary cusp; and RAO, right anterior oblique.
after ablation in 4 of 5 patients, except in the patient with coronary artery disease.

**Discussion**

**Anatomic Consideration of LVOT**

A thorough understanding of LVOT anatomy is important for proper localization of the VA origin and facilitates catheter ablation. Spatially, the aortic root occupies a central location within the heart, with the LVS anterosuperior to the aortic root. The anteriorly situated RVOT passes slightly superiorly and leftward relative to the aortic root. The conical-shaped RVOT is superior to the aortic valve (Figure 7). The LVOT consists of both muscular and fibrous portions. This is in contrast to the RVOT, which is

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**Table 2. Mapping and Ablation Data**

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>Below LCC</th>
<th>Below Junction of LCC/RCC</th>
<th>Below RCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>27</td>
<td>16/27</td>
<td>8/27</td>
<td>3/27</td>
</tr>
<tr>
<td>No. of previously failed ablation</td>
<td>13/27</td>
<td>7/16</td>
<td>5/8</td>
<td>1/3</td>
</tr>
<tr>
<td>Earliest ventricular activation, ms</td>
<td>39.5±7.7 (30–57)</td>
<td>37.6±8.1 (30–57)</td>
<td>42±8.5 (35–55)</td>
<td>41.3±4.0 (37–45)</td>
</tr>
<tr>
<td>Shortest distance to ASC, mm</td>
<td>5.1±2.8 (1.6–11.3)</td>
<td>7.4±3.3 (4–11.3)</td>
<td>3.8±0.9 (2.8–5)</td>
<td>3.7±1.8 (1.6–4.7)</td>
</tr>
<tr>
<td>Timing to VA disappearance, s</td>
<td>6.9±4.7 (1–19)</td>
<td>7.2±5.4 (2–19)</td>
<td>6.9±3.9 (1–9.2)</td>
<td>5.9±4.3 (1–9)</td>
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<tr>
<td>RF parameter at successful ablation</td>
<td>2.6±1.4 (1–5)</td>
<td>2.3±1.7 (1–5)</td>
<td>2.7±1.2 (2–4)</td>
<td>1.7±1.2 (1–3)</td>
</tr>
<tr>
<td>No. of RF applications</td>
<td>2.6±1.4 (1–5)</td>
<td>2.3±1.7 (1–5)</td>
<td>2.7±1.2 (2–4)</td>
<td>1.7±1.2 (1–3)</td>
</tr>
<tr>
<td>Maximal power, W</td>
<td>38.5±5.8</td>
<td>38.1±6.3</td>
<td>40.3±6.8</td>
<td>37.7±3.2</td>
</tr>
<tr>
<td>Timing to maximal power, s</td>
<td>12.5±7.5</td>
<td>8.4±8.1</td>
<td>12.3±8.7</td>
<td>17±2.6</td>
</tr>
<tr>
<td>Maximal temperature, °C</td>
<td>39.3±3.4</td>
<td>39.3±4.2</td>
<td>39.0±2.9</td>
<td>39.7±2.3</td>
</tr>
</tbody>
</table>

ASC indicates aortic sinus cusp; LCC, left coronary cusp; RCC, right coronary cusp; RF, radiofrequency; and VA, ventricular arrhythmia.
composed entirely of myocardium. The septal portion of the LVOT, although primarily muscular, includes the membranous portion of the ventricular septum. The posterior quadrant of the LVOT consists of an extensive fibrous curtain that extends from the fibrous skeleton of the heart across the anterior leaflet of the mitral valve and supports the aortic valve leaflets at the aortic-mitral continuity. The lateral and anterior LVOT are again muscular structures.\textsuperscript{14} Also, the aortic root is connected to the muscular ventricular septum, with the remaining one third in fibrous continuity with the mitral valve anterior leaflet.\textsuperscript{2,14–16} The RCC and anterior part of the LCC are connected with the ventricular musculature at their bases because the semilunar leaflets are hinged superiorly to the aortic wall but inferiorly to muscle.\textsuperscript{2,14–16} The entire noncoronary sinus cusp does not come in contact with the LV myocardium. Therefore, the noncoronary sinus cusp is exclusively composed of fibrous walls, located between the right and left atria, immediately anterior to the interatrial septum, and is the most posterior of the 3 sinuses (Figures 3–5).\textsuperscript{17}

Epicardially, the left anterior descending artery and left circumflex artery lie superior to the aortic portion of the LVOT and occupy the most superior portion of the LV (Figure 8B, 8C, and 8F), termed the LV summit by McAlpine.\textsuperscript{18} This region near the GCV and the anterior interventricular vein (AIV) is a major source of idiopathic VAs from the LV. Yamada et al\textsuperscript{9} described that the LVS is bisected by the GCV into an area lateral to this structure and is accessible to epicardial ablation and a superior region that is difficult to ablate because of the location of the coronary arteries and

![Figure 6](http://circep.ahajournals.org/)

**Figure 6.** Typical ECGs from 3 patients with ventricular arrhythmias (VAs) originating from the left ventricular outflow tract (LVOT) below the left coronary cusp (LCC), LCC/right coronary cusp (RCC) junction, and the RCC. A, ECG recording from a 15-year-old woman in whom the VA origin was located below the LCC, with 2 previous failed ablation attempts in the great cardiac vein, LVOT, and LCC; B, ECG recording from a 60-year-old woman with VAs originating from below the RCC/LCC junction; C, ECG recording from a 47-year-old woman with VAs originating below the RCC. Note that (1) II, III, and aVF have high amplitudes and there is QS morphology in aVR and aVL during clinical arrhythmias; (2) absolute values of the R-wave amplitudes in II, III and aVF, and the Q-wave amplitude in aVR and aVL are marked on the surface ECG; and (3) there is no S wave in V₅ and V₆, with early transition in the precordial leads before V₃.

**Table 3. QRS Morphology During Ventricular Arrhythmias on Surface ECG**

<table>
<thead>
<tr>
<th></th>
<th>All Below LCC</th>
<th>Below the junction of LCC/RCC</th>
<th>Below RCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>R-wave amplitude in II, mV</td>
<td>1.8±0.4 (1.1–2.6)</td>
<td>1.8±0.4 (1.1–2.4)</td>
<td>1.7±0.3 (1.3–2.2)</td>
</tr>
<tr>
<td>R-wave amplitude in III, mV</td>
<td>2.0±0.4 (1.3–3.0)</td>
<td>2.0±0.5 (1.3–3.0)</td>
<td>2.0±0.4 (1.6–2.5)</td>
</tr>
<tr>
<td>R-wave amplitude III&gt;II</td>
<td>19/27 (70.4%)</td>
<td>11/16 (68.8%)</td>
<td>7/8 (87.5%)</td>
</tr>
<tr>
<td>III/II R-wave amplitude ratio &gt;1.1</td>
<td>10/27 (37.0%)</td>
<td>5/16 (31.3%)</td>
<td>5/8 (62.5%)</td>
</tr>
<tr>
<td>Q-wave amplitude in aVL, mV</td>
<td>1.1±0.2 (0.7–1.4)</td>
<td>1.0±0.2 (0.7–1.4)</td>
<td>1.1±0.2 (0.7–1.4)</td>
</tr>
<tr>
<td>Q-wave amplitude in aVR, mV</td>
<td>0.9±0.3 (0.4–1.5)</td>
<td>0.8±0.3 (0.4–1.3)</td>
<td>0.9±0.4 (0.4–1.5)</td>
</tr>
<tr>
<td>Q-wave amplitude aVL&gt;aVR</td>
<td>20/27 (74.1%)</td>
<td>12/16 (75%)</td>
<td>5/8 (62.5%)</td>
</tr>
<tr>
<td>aVL/aVR Q-wave amplitude ratio &gt;1.4</td>
<td>7/27 (25.9%)</td>
<td>4/16 (25%)</td>
<td>3/8 (37.5%)</td>
</tr>
<tr>
<td>PDI</td>
<td>0.58±0.06 (0.46–0.7)</td>
<td>0.59±0.05 (0.53–0.67)</td>
<td>0.57±0.05 (0.50–0.62)</td>
</tr>
<tr>
<td>No. of PDI &gt;0.6</td>
<td>11/27 (40.7%)</td>
<td>8/16 (50%)</td>
<td>2/8 (25%)</td>
</tr>
</tbody>
</table>

LCC indicates left coronary cusp; PDI, peak deflection index in inferior leads; and RCC, right coronary cusp.
the thick layer of epicardial fat that overlies the proximal portion of these vessels.

**Previous Studies of Catheter Ablation of LVOT**

One third of all idiopathic VAs and ≈70% of idiopathic VAs originating from the LV originate from the LVOT in previous studies. LVOT-VA predominantly occurs in men and usually presents with PVCs, which is in line with our data. Mapping demonstrates that VAs frequently occur from the aortic root and LV endocardial and epicardial myocardium adjacent to the GCV and left anterior descending artery near the mitral annulus. In VAs from the aortic root, the LCC is the most common location, followed by the RCC and RCC/LCC junction. Rarely, VA origins can be located in the noncoronary sinus cusp, based on successful ablation. Also, catheter ablation is effective in abolishing these VAs from the aortic root. However, VAs from the myocardium just below the coronary sinus cusps have been generally difficult to ablate with a published ablation failure rate of 5% to 10%. Several parameters have been previously defined to predict ablation failure.

In previous studies using mapping and ablation from the CS and transaortic approaches, ECG parameters such as aVL/aVR Q-wave amplitude ratio >1.4, lead III/II R-wave amplitude ratio >1.1, and PDI >0.6 were used as criteria to identify ablation failure. In these cases, the epicardial LVOT was proposed as the VA origin. Recently, Jauregui Abularach et al performed ablation at the LCC and adjacent regions to abolish LVOT-VAs below the ASC in 16 patients, of whom 4 had previous failed ablation attempts. The authors found that the anatomic distances between sites of the epicardial veins and the closest ASC were greater in those 7 patients with failed ablation compared with the 9 patients who were successfully ablated (20.4±12.1 mm, P<0.01). Based on these findings, they proposed that an anatomic distance >15 mm was associated with failed ablation and may indicate an epicardial site of origin.

Figure 7. A to F, Complete anatomic mapping of the right ventricle (RV), left ventricle (LV), aorta root, and distal part of the coronary sinus (CS) in various views in patients with left ventricular summit (LVS) ventricular arrhythmias. Note that (1) the left anterior descending artery (LAD), left circumflex artery (LCX), and anterior interventricular vein (AIV) have been graphically added because the AIV was inaccessible with the 7F mapping catheter and coronary artery mapping is not performed; (2) the LVS is marked with black arrows and is close to the aortic cusps bordered by the vessels. AP indicates anteroposterior; GCV, great cardiac vein; L, left coronary cusp; LAO, left anterior oblique; LL, left lateral; N, noncoronary sinus cusp; PA, posteroanterior; RAO, right anterior oblique; RVOT, right ventricular outflow tract; and Sup, superior.

It is reported that ablation within the GCV and AIV and in the epicardial space via subxiphoid approach can abolish VA in some patients. There are several limitations in epicardial ablation targeting LVOT-VAs. First, mapping in the GCV and AIV is generally associated with higher impedances, which can prevent radiofrequency ablation or limit radiofrequency energy. Second, because of the close proximity of the coronary arteries and the thick layer of epicardial fat that overlies these vessels, epicardial LVOT ablation is often not feasible. Finally, radiofrequency ablation may result in inadvertent injury to the coronary arteries if the ablation target is <5 mm away.

**Catheter Ablation of VA Just Below the ASC via Transseptal Approach**

A previous case report described successful VA ablation emanating from the LVOT in a patient with failed retrograde transaortic approach. In the present study, retrograde anatomic mapping during sinus rhythm was initially performed, followed by antegrade transseptal mapping. The anterosuperior LVOT could be reached in all 27 patients via an antegrade transseptal approach using a reversed S curve on the ablation catheter. Importantly, to facilitate the latter approach, the transseptal puncture should target the anteroinferior fossa ovalis. We observed significant differences in the LVOT anatomic map depending on the approach used, indicating that the superoanterior LVOT cannot be reached via retrograde transaortic access. In contrast to previous studies, none of our patients demonstrated a distance between the ablation target and the closest ASC >12 mm. A large distance may be because of nonaccessibility using a retrograde approach, resulting in failed ablation attempts in some patients. To overcome this limitation, it was important to perform a complete aortic root map before LV mapping. In the present study, an average distance between site of earliest activation and closest ASC was 5.1±2.8 mm. Below the LCC, mean distance to the ablation target was slightly greater at 7.4±3.5 mm, which may
explain why in some patients VAs can be ablated with irrigated radiofrequency energy from the LCC.20 Also, because of the close anatomic relationship with the LCC (Figure 4B), our ablation strategy may be used as an alternative approach for ablating VAs from the LCC to avoid potential injury to the left main coronary artery if the distance to the artery is <5 mm or if the ablation catheter is unstable in the LCC.9,29

In the present study, the ablation target was only identified by earliest ventricular activation, which preceded the QRS onset by 39.5±7.7 ms and was not consistent with unipolar electrograms. In addition, pace mapping at the successful ablation site showed mismatch of QRS morphology to clinical VAs (Figure in the Data Supplement), suggesting that ablation should be performed while the patient is awake because sedation may result in suppression of clinical VAs. The above unipolar recording and pace mapping findings were similar to that described in previous studies.5,6,9,20 Another important finding was that irrigated radiofrequency resulted in rapid disappearance of VAs within 10 seconds. The rapid abolishment of VAs by irrigated radiofrequency energy strongly suggests that this type of VA may be located subendocardially. However, complete epicardial mapping via suboxiphoid approach was not performed in any of our patients; therefore, it is still unknown whether the earliest activation was located subepicardially. Nevertheless, the rapid effect during radiofrequency ablation demonstrated that this type of VAs can be successfully abolished via the transseptal approach, whether this is because of an endocardial origin or an epicardial origin in a region with thin myocardium. More importantly, no radiofrequency-related complications occurred via the transseptal approach as a result of the lack of coronary arteries in the endocardium in these 27 patients. Therefore, we strongly recommend that endocardial mapping at the anterosuperior LVOT via both retrograde and transseptal approaches should be performed initially to avoid potential risks of a suboxiphoid epicardial approach and ablation within the GCV and AIV in patients with this type of VA,22–27 because recent criteria suggest that it can be difficult to distinguish between an endocardial or epicardial origin.9,12,13

**ECG Characteristics of VA Below the ASC**

Previous studies attempted to describe ECG criteria to identify the successful ablation site in the LVS.6–9 Because the LVS is located most superior in the LV (Figure 8), VAs originating from this region exhibit high inferior lead R-wave amplitudes and no S wave in V5/V6,5,6,9 and right bundle branch block pattern has been described as a rare phenomenon.5,6,9 In our study, all patients had high inferior lead R-wave amplitudes of 2.0±0.4 mV, and 24 of 27 (88.9%) patients had left bundle branch block morphology, which is consistent with previous reports.5,6,9

Importantly, previous studies proposed an aVL/aVR Q-wave amplitude ratio >1.4,11 lead III/II R-wave amplitude ratio >1.1.11 and PDI >0.612 as criteria to identify VA not amenable to catheter ablation. The site of origin was most likely located in the presumed inaccessible epicardial LVOT. The present study identified an aVL/aVR Q-wave amplitude ratio >1.4 in 7 of 27 (25.9%) patients and lead III/II R-wave amplitude ratio >1.1 in 11 of 27 (40.7%) patients. In patients with deep Q waves in aVL and larger R waves in III, VA almost always originated below the LCC or LCC/RCC junction, which is consistent with our anatomic finding that the origins are more leftward and produce large vectors in lead III and against lead aVL (Figure 8). Also, PDI >0.6 was found in 11 of 27 (40.7%) patients. All patients with previously described ECG marker criteria for ablation failure had successful ablation with rapid clinical VA disappearance during radiofrequency application using the transeptal approach. In our opinion, additional studies are required to answer the question whether previous ECG criteria can be used to identify VA origins that are presumed inaccessible.

**Conclusions**

VAs from the anterosuperior LVOT present with left bundle branch block morphology, inferior axis, and early transition in the precordial leads on surface ECG in the majority of patients. This region is accessible with an antegrade approach from the anteroinferior transseptal puncture site using a reversed S curve on the ablation catheter. Clinically, only activation mapping is feasible because VA morphologies do not match QRS morphologies during pace mapping. The earliest activation demonstrating a low-amplitude potential was located 5.1±2.8 mm below the ASC. Irrigated radiofrequency energy resulted in rapid disappearance of clinical VAs. Therefore, in these patients we strongly recommend that the anterosuperior LVOT should initially be mapped using a combined retrograde and antegrade approach to minimize potential risks of suboxiphoid epicardial approach and ablation within the GCV and AIV. Furthermore, among the 27 patients who underwent successful ablation, 7 (25.9%) had aVL/aVR Q-wave amplitude ratio >1.4, 10 (37.0%) had III/II R-wave amplitude ratio >1.1, and 11 (40.7%) had PDI >0.6, which indicates that previous reported ECG criteria predicting ablation failure should be reinvestigated.

**Acknowledgments**

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**Disclosures**

None.

**References**


Left ventricular outflow tract ventricular arrhythmias (VAs) arise from the aortic sinus cusp, endocardial, or epicardial LV myocardium where coronary arteries are often adjacent. Sites immediately below the aortic sinus cusp have been difficult to ablate. This study included 27 patients with symptomatic VA, of which 13 had previous failed ablations. Left ventricular outflow tract mapping via the transaortic and transseptal approaches was performed. Use of the transseptal approach using a reversed S curve for catheter placement allowed identification of the site of earliest activation at the anterosuperior left ventricular outflow tract 5.1±2.8 mm below the aortic sinus cusp, preceding the QRS onset by 39.5±7.7 ms. Radiofrequency ablation was successful in all cases, despite the frequent presence of ECG markers previously associated with ablation failure. Pace mapping at the successful ablation site did not match the QRS of the VA, thus the presence of VA to allow activation mapping is important. There were no complications or VA recurrences during a mean follow-up of 8.4±2.5 months. A transseptal endocardial approach to the anterosuperior left ventricular outflow tract is an important approach to ablation of these arrhythmias.


Ventricular Arrhythmias Arising From the Left Ventricular Outflow Tract Below the Aortic Sinus Cusps: Mapping and Catheter Ablation via Transseptal Approach and Electrocardiographic Characteristics

Feifan Ouyang, Shibu Mathew, Shulin Wu, Masashi Kamioka, Andreas Metzner, Yumei Xue, Weizhu Ju, Bing Yang, Xianzhang Zhan, Andreas Rillig, Tina Lin, Peter Rausch, Sebastian Deiß, Christine Lemes, Tobias Tönnis, Erik Wissner, Roland Richard Tilz, Karl-Heinz Kuck and Minglong Chen

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In the article “Ventricular Arrhythmias Arising From the Left Ventricular Outflow Tract Below the Aortic Sinus Cusps: Mapping and Catheter Ablation via Transseptal Approach and Electrocardiographic Characteristics” by Ouyang et al, which was published in the June 2014 issue (Circ Arrhythm Electrophysiol. 2014;7:445–455), a correction was needed.

In Table 2, the timing to ventricular arrhythmia disappearance in all patients should be 6.9±4.7 (1–19).

The authors apologize for the error.

The online version of the article has been corrected.
Supplemental Material
Figure Legend

Pacemapping in a 49-year-old male with previous failed ablation attempt and VA origin below the LCC/RCC junction (A), and in a 42-year-old male with 2 previous failed ablation attempts and VA origin below the LCC. Note that (1) the left panel shows clinical VES following a sinus beat; (2) the QRS morphology was narrower in duration and lower in amplitude in the inferior leads compared to that during clinical VES when pacing at the VA origins below the LCC/RCC junction (A) and the LCC (B); (3) the QRS morphology in V1 continuously oscillates during pacing (cycle length 570ms). PVC = premature ventricular contraction; LCC = left coronary sinus cusp; RCC = right coronary sinus cusp