The left ventricular outflow tract (LVOT) is the most common site of the origin of idiopathic ventricular arrhythmias (VAs) after those arising from the right ventricular outflow tract (RVOT).1–8 Although endocardial radiofrequency catheter ablation (RFCA) is highly successful for idiopathic RVOT VAs,1 epicardial catheter ablation is sometimes required to cure LVOT VAs because the muscle of the LVOT is thicker than that of the RVOT.5,7–13 Anatomically, the sites of successful catheter ablation of idiopathic LVOT VAs include the aortic root, and the endocardium underneath the aortic valve, termed the aortomitral continuity (AMC). In addition, the epicardial surface of the LV, termed the LV summit, is a possible location for LVOT VAs, which can be reached by an ablation catheter within the great cardiac vein (GCV) or may require a transpericardial approach.3–10 The AMC and LV summit face each other at the basal portion of the LV muscle between them.9,10 Recently, the presence of idiopathic LVOT VAs originating from intramural foci located between the AMC and the LV summit, which required catheter ablation from both endocardial and epicardial sides for their elimination, has been reported.14 However, information on the prevalence and electrocardiographic and electrophysiological characteristics of these VAs is limited. The purpose of this study was to determine these features of idiopathic intramural LVOT VAs.

Methods

Patient Characteristics

The study patients were drawn from a total of 304 consecutive patients from a single center with symptomatic idiopathic VAs originating from the ventricular outflow tracts. The sites of these VA origins...
WHAT IS KNOWN

- Idiopathic ventricular arrhythmias (VAs) originating from the left ventricular outflow tract (LVOT) sometimes require catheter ablation from both the endocardial and epicardial sides for their elimination, suggesting the presence of intramural VA foci.
- Information on the prevalence and clinical, electrocardiographic and electrophysiological characteristics of idiopathic intramural LVOT VAs is limited.

WHAT THE STUDY ADDS

- Idiopathic intramural LVOT VAs are not rare, and require more radiofrequency ablation for their elimination than the endocardial and epicardial LVOT VAs, rendering catheter ablation of LVOT VAs more challenging.
- The electrocardiographic and electrophysiological characteristics of the idiopathic intramural LVOT VAs were midrange between those of the idiopathic endocardial and epicardial LVOT VAs, and more similar to those of the idiopathic epicardial LVOT VAs.
- Idiopathic intramural LVOT VAs are not rare, and require more radiofrequency ablation for their elimination than the endocardial and epicardial LVOT VAs, rendering catheter ablation of LVOT VAs more challenging.

Electrophysiological Study

For mapping and pacing, a quadripolar catheter was positioned via the right femoral vein at the HIS bundle region, and a 6 or 7-French deflectable decapolar catheter in the coronary sinus. The coronary sinus catheter was advanced into the GCV as far as possible until the distal electrode pair recorded an earlier ventricular activation than the most distal electrode pair during the VAs (Figure 1). Mapping and pacing were performed using a 7.5-French, 3.5-mm tip irrigated ablation catheter (Navistar Thermocool, Biosense Webster, Diamond Bar, CA) introduced from the right femoral vein (for the RVOT and GCV) or right femoral artery (for the endocardial LVOT). During the procedures in the endocardial LVOT, intravenous heparin was administered to maintain an activated clotting time of >300 s. 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 RVOT or apex with the addition of an isoproterenol infusion.

Mapping and RFCA

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. Pace mapping was also 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 score for the pace mapping was determined as the number of leads with an identical height 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), as well as the number of leads with a fine notching ratio match 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 which obtained a score of >20.

When the earliest ventricular activation preceded the QRS onset by at least 20 ms or an excellent pace map was demonstrated in the AMC and GCV, irrigated unipolar RF current was applied at this site. When there were no sites with such an early activation or excellent pace map in these regions, epicardial mapping via a subxiphoid approach was performed with an irrigated ablation catheter to seek an earlier ventricular activation or excellent pace map on the LV epicardial surface beside the GCV as previously reported. When a more suitable site for ablation was identified on the epicardial surface beside the GCV, irrigated unipolar RF current was applied at that site. If the first irrigated unipolar RF application from the AMC or GCV was unsuccessful, a second irrigated unipolar RF application was delivered at the site with the earliest ventricular activation on the opposing side (sequential

Figure 1. Fluoroscopic images exhibiting the catheter positions. ABL indicates the ablation catheter; GCV, the great cardiac vein; HB, His bundle; LAO, left anterior oblique view; and RAO, right anterior oblique view.
unipolar ablation; Figure 1). If the sequential endocardial/epicardial ablation configuration was unsuccessful in eliminating the VA, irrigated unipolar RF current was simultaneously applied to the distal electrodes of 2 catheters using 2 RF generators (Stockert, BioSense Webster) at the same sites that were used for the sequential configuration (simultaneous unipolar ablation). Irrigated RF current was delivered in the power-control mode starting at 20 W in the GCV and 30 W at the AMC and on the epicardial surface beside the GCV with irrigation flow rates of 30 mL/min. The RF power was independently titrated to ≤30 W and 40 W, respectively. The goal of RF applications was to achieve a decrease in the impedance of 8 to 10Ω and with care taken to limit the temperature to <45°C as monitored from both RF generators. During the epicardial catheter ablation using transvenous and transpericardial approaches, simultaneous left coronary angiography was performed every 15 s 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. An RF 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 s of the application, the RF delivery was terminated, and the catheter was repositioned. The end point of the catheter ablation was the elimination and noninducibility of VT or PVCs using electronic calipers by 2 experienced investigators blinded to the site of the 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 site of the VA origin was determined by the successful ablation site. When the VA was successfully eliminated by ablation in the AMC alone, it was considered one from an endocardial origin and the patient was assigned to the Endo group. When the VA was successfully eliminated by epicardial ablation in the LV summit alone through the GCV or pericardial approach, it was classified as one from an epicardial origin and the patient was allocated to the Epi group. When catheter ablation from both the endocardial and epicardial sides was required for an elimination of the VA, this VA was defined as one originating from an intramural focus and the patient was included in the intramural group.

**Electrocardiographic Analysis**

The simultaneous 12-lead electrocardiograms during the VAs and pace mapping were recorded digitally at a sweep speed of 100 to 200 mm/s in all patients for off-line analysis. The QRS morphologies, including a bundle branch block pattern, axis, configuration in leads I and V5 or V6, were examined. 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 from left to right, suggesting a VA origin located in the LV free wall. In lead V5 or V6, the main concern was the presence of an S wave, which was considered to be a characteristic and convenient electrocardiographic finding of AMC VAs probably because the S wave in lead V5 or V6 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 the 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. A long precordial MDI, reflecting a 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 to the local ventricular activation time relative to the QRS onset.

**Figure 2.** A flow diagram exhibiting the results of the mapping and catheter ablation. AMC indicates aortomitral continuity; GCV, great cardiac vein; and V-QRS, the local ventricular activation time relative to the QRS onset.
the endocardium and the delayed global ventricular activation resulting from the later engagement of the His-Purkinje network.

**Follow-Up**
Follow-up after the procedure included clinic visits with 12-lead electrocardiograms 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.

**Statistical Analysis**
IBM SPSS Statistics was used for the statistical analyses. Continuous variables are expressed as the group mean±1 SD. Comparisons of the continuous variables between the 2 groups were analyzed with the use of the Student t test. When comparisons involved >2 groups, an ANOVA was used. When group differences were found, a 1-way ANOVA was followed by the Bonferroni correction to test the significance of the difference among the means in all groups. The categorical variables expressed as numbers and percentages in the different groups were compared with a \( \chi^2 \) test and Yates correction if necessary. An overall Fisher exact test for a 2×n table was constructed when comparisons involved >2 groups. Statistical significance was selected at a value of \( P<0.05 \).

**Results**

**Mapping and Ablation**
The results of the mapping and catheter ablation are shown in Figure 2. Transpericardial mapping was performed in 23 patients because the earliest ventricular activation within the GCV preceded the QRS onset by <20 ms. Among those 23 patients, RFCA was unsuccessful in 15 because the VAs presumably originated from an inaccessible area of the LV summit with a close proximity to the coronary arteries and thick fat pads. In 6 patients, RFCA was successful on the epicardial surface beside the GCV in the accessible area of the LV summit. In the remaining 2 patients, no earlier ventricular activation was recorded on the epicardial surface beside the GCV than within the GCV, and eventually a unipolar RFCA from both the AMC and GCV was required. Irrigated unipolar RF ablation only from a single site was successful in the AMC in 30 patients, and in the LV summit in 34. Irrigated unipolar RF ablation from both the AMC and GCV was required for a successful ablation in 18 patients. VAs were eliminated by a sequential catheter ablation in 13 of the 18 patients, whereas a simultaneous RF catheter ablation was required.

**Figure 3.** Electrocardiograms exhibiting the premature ventricular contraction (PVC) and pace maps obtained by pacing from the aorto-mitral continuity (AMC) and great cardiac vein (GCV) (left) and cardiac tracings exhibiting the local ventricular activation times in the AMC and GCV during the PVC (right). Although the pace map was excellent in the AMC, the local ventricular activation time during the PVC was earlier within the GCV (arrowhead) than the AMC. The first ablation from the GCV failed, and the subsequent ablation from the AMC successfully eliminated the PVCs. ABL indicates the ablation catheter positioned in the AMC; CS 1 to 5, the first (most distal) to fifth (most proximal) electrode pairs of the CS catheter positioned in the GCV; HB, His bundle; 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.
for the successful ablation in the remaining 5 patients. In one of the 13 patients with a successful sequential catheter ablation, the local ventricular activation time relative to the QRS onset (V-QRS) during the VAs was earlier in the GCV than the AMC, although a pace map in the AMC was excellent and better than that in the GCV (Figure 3). In this patient, catheter ablation was successful in the AMC after the ablation in the GCV failed. In another patient with a successful sequential catheter ablation, the V-QRS during the PVCs was earlier in the AMC than the GCV, and the first ablation was delivered in the AMC (Figure 3). It failed, but changed the QRS morphology of the PVCs with a longer QRS duration and greater MDI. This new QRS morphology of the PVCs had never been observed before the ablation. The PVCs were successfully eliminated by catheter ablation within the GCV where the V-QRS during the VAs turned to be earlier than that before the first ablation in the AMC. No complications occurred.

Comparison of the Clinical, Electrocardiographic, and Electrophysiological Characteristic Among the VAs Originating From the Endocardial, Epicardial, and Intramural Foci in the LVOT

The patients with epicardial VA foci were significantly younger than those with endocardial VA foci (P=0.0004; Table 1). There were no significant differences in the other clinical characteristics, including the sex, LV ejection fraction, incidence of tachycardia-induced cardiomyopathy, and nature of the clinical arrhythmia among the 3 groups (Table 1). The results of the electrocardiographic and electrophysiological parameters are summarized in Table 2. The electrocardiograms of all intramural LVOT VAs are shown in Figure 5. The representative electrocardiograms of the endocardial, intramural, and epicardial LVOT VAs are shown in Figure 6. In terms of the QRS morphology of the VAs, a pattern of right bundle branch block and right inferior axis QRS morphology was most prevalent in all the 3 groups. All epicardial LVOT VAs exhibited a right inferior axis QRS morphology, whereas 37% of the endocardial LVOT VAs and 6% of the intramural LVOT VAs exhibited a left inferio axis QRS morphology. In lead I, 90% and 72% of the endocardial and intramural LVOT VAs exhibited an rS pattern, respectively, whereas 53% of the epicardial LVOT VAs did. The III/II and aVL/aVR ratios were significantly greater in the epicardial LVOT VAs than the endocardial LVOT VAs (P=0.0202 and P=0.0017, respectively). However, there were no significant differences in the III/II and aVL/aVR ratios between the intramural LVOT VAs and the endocardial or epicardial LVOT VAs. The MDI was significantly greater.

![Figure 4. Electrocardiograms exhibiting the change in the QRS morphology of the premature ventricular contraction (PVC) after the ablation in the aortomitral continuity (AMC) (left) and cardiac tracings exhibiting the ablation sites in the AMC and GCV during the PVC (right).](http://circep.ahajournals.org/)
for the epicardial LVOT VAs than the endocardial LVOT VAs ($P=0.0005$). However, there was no significant difference in the MDI between the intramural LVOT VAs and the epicardial or endocardial LVOT VAs. The V-QRS at the successful ablation site tended to be greater during the epicardial LVOT VAs than the endocardial and intramural LVOT VAs.

### Table 1. Basic Demographics and Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Endo (n=30)</th>
<th>Epi (n=34)</th>
<th>Intramural (n=18)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>63±13</td>
<td>48±13*</td>
<td>56±13</td>
<td>0.0004</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>17 (57)</td>
<td>14 (41)</td>
<td>9 (50)</td>
<td>0.46</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>60.3±10.4</td>
<td>58.4±13.2</td>
<td>60.3±14.3</td>
<td>0.86</td>
</tr>
<tr>
<td>Tachycardia-induced CM, n (%)</td>
<td>5 (17)</td>
<td>7 (21)</td>
<td>4 (22)</td>
<td>0.96</td>
</tr>
<tr>
<td>Clinical arrhythmias, n (%)</td>
<td>0.86</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequent PVCs</td>
<td>19 (63)</td>
<td>18 (53)</td>
<td>9 (50)</td>
<td></td>
</tr>
<tr>
<td>Nonsustained VT</td>
<td>6 (20)</td>
<td>7 (21)</td>
<td>4 (22)</td>
<td></td>
</tr>
<tr>
<td>Sustained VT</td>
<td>5 (17)</td>
<td>9 (26)</td>
<td>5 (28)</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as the mean±SD. CM indicates cardiomyopathy; Endo, endocardial; Epi, epicardial; LVEF, left ventricular ejection fraction; PVC, premature ventricular contraction; and VT, ventricular tachycardia.

*Significant versus Endo.

### Table 2. Comparison of the Electrocardiographic and Electrophysiological Parameters

<table>
<thead>
<tr>
<th></th>
<th>Endo (n=30)</th>
<th>Epi (n=34)</th>
<th>Intramural (n=18)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total QRS duration of VA, ms</td>
<td>169±19</td>
<td>174±20</td>
<td>168±25</td>
<td>0.54</td>
</tr>
<tr>
<td>QRS morphology, n (%)</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LBBB+LIA</td>
<td>2 (7)</td>
<td>0 (0)</td>
<td>1 (6)</td>
<td></td>
</tr>
<tr>
<td>LBBB+RIA</td>
<td>2 (7)</td>
<td>13 (38)</td>
<td>1 (6)</td>
<td></td>
</tr>
<tr>
<td>RBBB+LIA</td>
<td>9 (30)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>RBBB+RIA</td>
<td>17 (56)</td>
<td>21 (62)</td>
<td>16 (88)</td>
<td></td>
</tr>
<tr>
<td>Lead I, n (%)</td>
<td></td>
<td></td>
<td></td>
<td>0.0004</td>
</tr>
<tr>
<td>Q wave</td>
<td>1 (3)</td>
<td>16 (47)</td>
<td>1 (6)</td>
<td></td>
</tr>
<tr>
<td>rS wave</td>
<td>27 (90)</td>
<td>18 (53)</td>
<td>13 (72)</td>
<td>0.18</td>
</tr>
<tr>
<td>Precordial transition, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤V1</td>
<td>15 (50)</td>
<td>19 (56)</td>
<td>13 (72)</td>
<td></td>
</tr>
<tr>
<td>V1–V2</td>
<td>4 (13)</td>
<td>1 (3)</td>
<td>2 (11)</td>
<td></td>
</tr>
<tr>
<td>V2–V3</td>
<td>11 (37)</td>
<td>14 (41)</td>
<td>3 (17)</td>
<td></td>
</tr>
<tr>
<td>R-wave amplitude in the inferior leads, mV</td>
<td>1.36±0.49</td>
<td>1.36±0.51</td>
<td>1.33±0.43</td>
<td>0.97</td>
</tr>
<tr>
<td>R-wave amplitude ratio in the leads III to II</td>
<td>1.10±0.26</td>
<td>1.23±0.19*</td>
<td>1.09±0.22</td>
<td>0.0202</td>
</tr>
<tr>
<td>Q-wave amplitude ratio in leads aVL to aVR</td>
<td>1.14±0.55</td>
<td>1.78±0.79*</td>
<td>1.34±0.76</td>
<td>0.0017</td>
</tr>
<tr>
<td>Maximum deflection index</td>
<td>0.45±0.06</td>
<td>0.52±0.07†</td>
<td>0.49±0.05</td>
<td>0.0005</td>
</tr>
<tr>
<td>S wave in lead V6, n (%)</td>
<td>11 (37)</td>
<td>11 (32)</td>
<td>4 (22)</td>
<td>0.58</td>
</tr>
<tr>
<td>V-QRS, ms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ablation site</td>
<td>−26.6±10.9</td>
<td>−31.3±8.0*</td>
<td>−25.2±9.2</td>
<td>0.0475</td>
</tr>
<tr>
<td>His bundle region</td>
<td>31.0±10.7</td>
<td>38.6±15.6*</td>
<td>27.1±18.1</td>
<td>0.0303</td>
</tr>
<tr>
<td>AV amplitude ratio at the ablation site</td>
<td>0.29±0.44</td>
<td>1.39±1.73*</td>
<td>0.17±0.19</td>
<td>0.0004</td>
</tr>
<tr>
<td>No. of RF applications</td>
<td>1.6±0.8</td>
<td>1.9±1.7</td>
<td>4.6±1.5*</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Data are expressed as the mean±SD. Endo indicates endocardial; Epi, epicardial; LBBB, left bundle branch block; LIA, left inferior axis; RBBB, right bundle branch block; RIA, right inferior axis; RF, radiofrequency; VA, ventricular arrhythmia; and V-QRS, the local ventricular activation time relative to the QRS onset.

*Significant vs each of the other sites.
†Significant vs Endo.
Yamada et al  Idiopathic Intramural LVOT VAs

VAs ($P=0.0475$). The V-QRS at the His bundle region was significantly smaller during the epicardial LVOT VAs than the endocardial and intramural LVOT VAs ($P=0.0303$). The atrial to ventricular electrogram amplitude (A/V) ratio at the successful ablation site was significantly greater during the epicardial LVOT VAs than the endocardial and intramural

Figure 5. Twelve-lead electrocardiograms exhibiting all ventricular arrhythmias (VAs) with the intramural left ventricular outflow tract (LVOT) origins. The first 13 and last 5 electrocardiograms exhibit the intramural LVOT VAs that required sequential and simultaneous catheter ablation from both the endocardial and epicardial sides for their elimination, respectively.
LVOT VAs ($P=0.0004$). However, there were no significant differences in the V-QRS at the successful ablation site and His bundle region and A/V ratio at the successful ablation site between the endocardial and intramural LVOT VAs. The number of RF applications required for a successful ablation was significantly greater in the intramural LVOT VAs than the endocardial and epicardial LVOT VAs ($P<0.0001$). There were no significant differences in the other electrophysiological parameters, including the precordial transition, maximum R-wave amplitude in the inferior leads, and the presence of S waves in lead V6 among the 3 groups.

Follow-Up
During the follow-up period (median, 51 and interquartile range, 28 months) after the successful ablation, all 18 patients with the intramural LVOT VAs remained free of any VAs without any antiarrhythmic drugs. The LV ejection fraction had normalized after the successful ablation of the intramural LVOT VAs in all 4 patients with tachycardia-induced cardiomyopathy. No complications occurred.

Discussion

Major Findings
In the past decade, procedural techniques have advanced, and the understanding of the electrophysiological mechanisms has improved, allowing catheter ablation to cure most idiopathic VAs arising from the endocardium and epicardium. However, catheter ablation of idiopathic VAs arising from the intramural foci remains challenging, and information about idiopathic intramural LVOT VAs is limited. This study revealed the prevalence and clinical, electrocardiographic and electrophysiological characteristics of the idiopathic intramural LVOT VAs by comparing with the endocardial and epicardial LVOT VAs. In this study, $\approx20\%$ of idiopathic LVOT VAs originated from intramural foci, and this prevalence corresponded to approximately half and 60% of that of the idiopathic epicardial and endocardial LVOT VAs, respectively. Because idiopathic intramural LVOT VAs were not rare, and required more RF ablation for their elimination, they rendered catheter ablation of LVOT VAs more challenging.

The Purkinje network is located only in the subendocardium. Because of this anatomic background, ventricular activation from an epicardial origin requires more time to reach the Purkinje network than that from an endocardial origin, resulting in a difference in the electrocardiographic and electrophysiological characteristics between the endocardial and epicardial VAs. Once the ventricular activation from LVOT VA origins reaches the Purkinje network, the activation quickly propagates down the ventricular septum, allowing for an early ventricular activation in the His bundle region, a taller R wave in lead II than lead III and deeper Q wave in lead aVR than lead aVL. However, the ventricular activation from epicardial LVOT VA origins propagates toward the LV lateral wall before the His bundle region is activated, resulting in a decrease in the amplitude of an R wave in lead II and Q wave in lead aVR.
and an increase in the amplitude of an R wave in lead III. These electrophysiological features are consistent with the findings in this study that the III/II and aVL/aVR ratios were significantly greater in the epicardial LVOT VAs than the endocardial LVOT VAs and the V-QRS at the His bundle region was significantly smaller during the epicardial LVOT VAs than the endocardial LVOT VAs. In this study, the V-QRS at the successful ablation site tended to be greater during the epicardial LVOT VAs than the endocardial LVOT VAs. This finding might be explained by the mechanism that it was likely to take a longer time for the activation from an epicardial VA focus to activate a mass of the ventricular muscle large enough to cause a QRS onset because the activation propagated slowly on the epicardial surface when compared with the endocardial surface. In fact, in one case in this study with an intramural LVOT VA, the V-QRS recorded at the AMC when the activation from the VA focus preferentially conducted to the endocardium, was later than the V-QRS recorded within the GCV when the activation from the VA focus preferentially conducted to the epicardium after the RF ablation at the AMC eliminated the endocardial breakthrough (Figure 4). These findings would support the abovementioned mechanism.

In this study, the electrocardiographic and electrophysiological characteristics of the idiopathic intramural LVOT VAs were midrange between those of the idiopathic endocardial and epicardial LVOT VAs, and more similar to those of the idiopathic endocardial LVOT VAs than those of the idiopathic epicardial LVOT VAs. These results are reasonable for several reasons. First, the ventricular activation from an intramural origin would require a midrange time to reach the Purkinje network between that from endocardial and epicardial origins. Second, even if the activation from an intramural VA focus reached the epicardium earlier than the endocardium, the endocardial ventricular activation would be more likely to determine the electrocardiographic and electrophysiological characteristics because the endocardial ventricular activation should propagate more quickly than the epicardial ventricular activation. This mechanism was demonstrated by the findings in this study that the pace map in the AMC was excellent and better than that within the GCV, although the V-QRS during the VAs was earlier within the GCV than the AMC (Figure 3). Third, the activation from some intramural VA foci preferentially conducted to the endocardium. This mechanism was demonstrated by the findings in this study. In one case, although the baseline QRS morphology of the intramural LVOT VAs suggested an endocardial VA origin, the QRS morphology changed after the ablation in the AMC, exhibiting the electrocardiographic characteristics of epicardial LVOT VAs (Figure 4).

In this study, the A/V ratio at the successful ablation site was significantly greater during the epicardial LVOT VAs than the endocardial and intramural LVOT VAs. These findings suggested that the foci of the epicardial LVOT VAs were located more basally than those of the endocardial and intramural LVOT VAs.

Conclusions

Idiopathic intramural LVOT VAs are not rare, and require more RF ablation for their elimination than the endocardial and epicardial LVOT VAs, rendering catheter ablation of LVOT VAs more challenging. The electrocardiographic and electrophysiological characteristics of the idiopathic intramural LVOT VAs were midrange between those of the idiopathic endocardial and epicardial LVOT VAs, and more similar to those of the idiopathic endocardial LVOT VAs than those of the idiopathic epicardial LVOT VAs.

Disclosures

Drs Kay, Plumb, and McElderry have participated in catheter research funded by Biosense-Webster and Irvine Biomedical. Dr Kay has received honoraria from Medtronic, Boston Scientific, and St. Jude Medical. Dr McElderry has received consulting fees from Boston Scientific, St. Jude Medical, and Biosense-Webster. The other authors report no conflicts.

References

Prevalence and Electrocardiographic and Electrophysiological Characteristics of Idiopathic Ventricular Arrhythmias Originating From Intramural Foci in the Left Ventricular Outflow Tract


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