Idiopathic Ventricular Arrhythmias Originating From the Aortic Root
Prevalence, Electrocardiographic and Electrophysiologic Characteristics, and Results of Radiofrequency Catheter Ablation

Takumi Yamada, MD,* H. Thomas McElderry, MD,* Harish Doppalapudi, MD,* Yoshimasa Murakami, MD,‡ Yukihiko Yoshida, MD,§ Naoki Yoshida, MD,‡ Taro Okada, MD,‡ Naoya Tsuboi, MD,§ Yasuya Inden, MD,# Toyoaki Murohara, MD,# Andrew E. Epstein, MD,* Vance J. Plumb, MD,* Satinder P. Singh, MD,† G. Neal Kay, MD*
Birmingham, Alabama; and Ichinomiya and Nagoya, Japan

Objectives
This study investigated the prevalence and electrocardiographic and electrophysiologic characteristics of aortic root ventricular arrhythmias (VAs).

Background
Idiopathic VAs originating from the ostium of the left ventricle may be ablated at the base of the aortic cusps.

Methods
We studied 265 patients with idiopathic VAs with an inferior QRS-axis morphology.

Results
The successful ablation site was within (or below) the aortic cusps in 44 patients (16.6%). The site of the origin was the left coronary cusp (LCC) in 24 (54.5%), the right coronary cusp (RCC) in 14 (31.8%), the noncoronary cusp (NCC) in 1 (2.3%), and at the junction between the LCC and RCC (L-RCC) in 5 (11.4%) cases. The maximum amplitude of the R-wave in the inferior leads was significantly greater with an LCC than with an RCC origin (p < 0.05). The ratio of the R-wave amplitude in leads II and III was significantly greater with an LCC than with an RCC origin (p < 0.01) and was significantly smaller in the NCC than in the other sites (p < 0.0001). The ventricular deflection in the His bundle electrogram was significantly later relative to the surface QRS with an LCC or L-RCC origin than with an RCC or NCC origin (p < 0.0001). The ratio of the atrial-to-ventricular deflection amplitude was significantly greater in the NCC than in the other sites (p < 0.0001). No other factors predicted the site of origin.

Conclusions
Idiopathic VAs are more common in the LCC than in the RCC and rarely arise from the NCC. The electrocardiogram is useful for differentiating the site of origin. (J Am Coll Cardiol 2008;52:139–47) © 2008 by the American College of Cardiology Foundation

The myocardium around the ventricular outflow tract is a major source of idiopathic ventricular tachycardias (VTs) or premature ventricular contractions (PVCs) (1–8). Radiofrequency catheter ablation is a safe and reliable technique for curing those ventricular arrhythmias (VAs) (2–8). Several previous reports have revealed differences in the electrocardiographic characteristics of VAs originating from the right ventricular outflow tract (RVOT), left ventricular outflow tract (LVOT), and aortic root regions (4–6,9). However, for VAs originating from the aortic root, information on the relationships between the location of their origin and prevalence and the electrocardiographic and electrophysiologic characteristics is limited. The present study was undertaken to examine the features that predict the site of origin in the aortic root.

Methods

Patient characteristics. The study population consisted of 265 consecutive patients (71 men, mean age 53 ± 15 years [range 16 to 80 years]) with symptomatic idiopathic sustained VT (n = 34), nonsustained VT (n = 56), or PVCs (n = 175) originating from the ventricular outflow tract. During the clinical arrhythmia, the surface electrocardio-
gram revealed a uniform QRS morphology with an inferior axis in all patients. Echocardiography and exercise stress testing or coronary angiography demonstrated no evidence of structural heart disease in any patients. Each patient gave written informed consent, and all antiarrhythmic drugs were discontinued for ≥5 half-lives before the study.

Electrophysiologic study. Six-French quadripolar catheters were introduced from the right femoral vein and placed across the tricuspid valve to record the His bundle (HB) activation and in the right ventricular (RV) apex for pacing. Mapping and pacing were performed using a 7-F 4-mm-tip ablation catheter introduced from the right femoral vein (for the RVOT) or right femoral artery (for the LVOT or aortic root). When few PVCs were observed at the beginning of the electrophysiologic study, induction of the VT or PVCs was attempted by burst pacing from the RVOT or apex with the addition of an isoproterenol infusion. During mapping of the femoral artery (for the LVOT or aortic root). When few PVCs were observed at the beginning of the electrophysiologic study, induction of the VT or PVCs was attempted by burst pacing from the RVOT or apex with the addition of an isoproterenol infusion. During mapping of the LVOT or aortic sinuses (ASCs), intravenous heparin was administered to maintain an activated clotting time of >250 s.

Mapping and radiofrequency catheter ablation. Activation and pace mapping were performed in all cases to identify the VA origin site. In some patients, when VT or PVCs were frequent, electroanatomic mapping was performed as previously reported (10–12) (Fig. 1). Pace mapping was performed at a pacing cycle length of 500 ms and stimulus amplitude of 1 mA greater than the late-diastolic threshold. When the earliest site of activation was mapped to a site above the aortic valve, selective angiography of the coronary artery and aorta was performed before ablation to assess the anatomic relationships between those structures and the location of the ablation catheter. In cases where the distinction between the aortic sinuses could not be determined by angiography alone, intracardiac echocardiography also was used to identify the site of the ablation catheter. If the ablation site was close to the ostium of the coronary artery, radiofrequency ablation was performed with an angiographic catheter positioned within the coronary artery ostium with frequent hand injections of contrast (every 15 s). The wall of the ASCs as well as coronary artery flow was observed carefully by injecting an adequate volume of contrast to overflow into the ASC. A radiofrequency application was never delivered to an area within 5 mm of the angiographic catheter. Radiofrequency applications were delivered with a target temperature of 60°C and

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**Figure 1** Successful Ablation Site of PVCs Originating From the L-RCC

The first beat is a sinus beat and the second is a premature ventricular contraction (PVC). At the successful ablation site, 2 ventricular activation components and no atrial activations were recorded during sinus rhythm (left panel). The sequence of the 2 components was reversed during the PVCs. The first of the 2 components preceded the QRS onset by 65 ms. The aortogram showed that the ablation catheter was located in the junction of the left and right coronary cusps (L-RCC) (center panels). Note that the tip of the ablation catheter was positioned at the L-RCC by deflecting the loop of the ablation catheter in the left ventricular cavity. The activation map during the PVCs revealed the earliest activation at the L-RCC (right panels). ABLd = distal electrode pair of the ablation catheter; ABLp = proximal electrode pair of the ablation catheter; ABLuni = distal unipolar electrode of the ablation catheter; AP = anteroposterior; LAO = left anterior oblique view; LCC = left coronary cusp; LL = left lateral; LVOT = left ventricular outflow tract; RAO = right anterior oblique view; RCC = right coronary cusp; RVOT = right ventricular outflow tract.
maximum power output of 50 W at sites exhibiting the earliest bipolar activity and/or a local unipolar QS pattern during the VT or PVCs. When an acceleration or reduction in the incidence of VT or PVCs was observed during the first 10 s of the application, the radiofrequency delivery was continued for 30 to 60 s. Otherwise, the radiofrequency delivery was terminated, and the catheter was repositioned. If the earliest ventricular activation was observed with an HB potential or close to the HB region during the VT or PVCs, radiofrequency energy was delivered at least 5 mm away from the site recording the largest HB potential, using an initial power of 30 W. The end point of the catheter ablation was the elimination and noninducibility of VT or PVCs during an isoproterenol infusion (2 to 4 μg/min) and burst pacing from the right ventricle (to a cycle length as short as 300 ms).

When VAs were suspected to originate from the epicardial side around the LVOT, mapping was performed in the great cardiac vein or anterior interventricular cardiac vein using a mapping or ablation catheter introduced via the right femoral or internal jugular vein. If mapping in those cardiac veins revealed the earliest activation preceding the onset of the QRS during the VAs and an excellent pace map, the VAs were considered to originate from the left ventricular (LV) epicardium.

**Follow-up.** Follow-up was performed at 2 weeks, 1 month, and every 3 months thereafter using 24-h Holter monitoring and 12-lead electrocardiograms. All patients who reported symptoms were given an event monitor to document the cause of the symptoms.

**Statistical analysis.** 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 analysis of variance (ANOVA) was used. When group differences were found, a 1-way ANOVA was followed by the Fisher least significant difference method to test the significance of the difference among the means in all groups. The categoric variables expressed as numbers and percentages in the different groups were compared with a chi-square test and Yates correction if necessary. An overall chi-square test for a 2 × n table was constructed when comparisons involved >2 groups. Statistical significance was selected at a p value of <0.05.

**Results**

**Overall mapping, catheter ablation, and follow-up.** Successful ablation was acutely achieved in 260 of 265 patients. In the remaining 5 patients, an LV epicardial origin was identified that was not ablated. The successful ablation site was located in the aortic root in 44, RVOT in 199, LVOT in 11, LV epicardium in 3, and pulmonary artery in 3 of the patients. Eventually, in 8 of 265 patients, the VA origins were deemed to be epicardial. Two procedures were required to eliminate the VAs in the aortic root in 2 patients, RVOT in 15, and LVOT in 1. The number of radiofrequency applications required for a successful ablation was significantly larger in the VAs with an RVOT or LVOT origin than in those with an aortic root origin (4.0 ± 1.4 or 3.8 ± 0.8, respectively, vs. 2.2 ± 0.9; p < 0.0001 and p = 0.0005, respectively). Over a mean follow-up of 18 ± 12 months after the last procedure, VAs with the same QRS morphology as previously targeted recurred in 12 (11 [6%] in the RVOT and 1 [9%] in the LVOT; 5 [9%] were sustained VTs, 1 [2%] nonsustained VT, and 8 [5%] PVCs), and those VAs were excluded from the analysis because their origins were not fully defined. In the remaining 253 patients without a VA recurrence, the origin was determined to be in the aortic root in 44, RVOT in 188, LVOT in 10, LV epicardium in 8, and pulmonary artery in 3. The basic demographics of those patients are given in Table 1. Three patients with an RVOT, LVOT, or left coronary cusp (LCC) VA origin were complicated by cardiomyopathy induced by frequent PVCs. In all cases, the LV ejection fraction was <45% at baseline and normalized within 3 months after the elimination of the VAs.

**Catheter ablation of VAs with an origin in the aortic root.** Successful ablation was achieved in all 44 patients (100%) with aortic root VAs. This represented 17.3% of the successfully ablated patients or 16.6% of all patients. The VA origin was determined to be in the LCC in 24 (54.5%), right coronary cusp (RCC) in 14 (31.8%), junction between the left and right coronary cusps (L-RCC) in 5 (11.4%), and noncoronary cusp (NCC) in 1 (2.3%) patient. The distance from the tip of the ablation catheter to the left or right coronary artery ostia was 12.9 ± 3.1 mm (range 7.5 to 17.2 mm) and 10.5 ± 3.0 mm (range 8.3 to 18.1 mm), respectively.

In the catheter ablation of the L-RCC VAs, the tip of the ablation catheter was positioned at that site by deflecting the loop of the ablation catheter in the LV cavity in 3 patients or on the NCC in the remaining 2 (Fig. 1). In all patients with an RCC or NCC origin, the earliest RV activation preceding the QRS onset was recorded in the HB region, whereas in the patients with an LCC or L-RCC origin, the local ventricular activation in the RV HB region never preceded the QRS onset (Figs. 1 to 3). In 2 of our initial patients with a VA origin in the RCC or NCC contiguous to the RCC, radiofrequency applications were first delivered near the HB region in the right ventricle, because the local ventricular activation preceded the QRS onset (Fig. 2) and a good pace map (score = 10 of 12) was obtained. These radiofrequency applications did not interrupt the VAs, but caused a slight change in the QRS morphology, with a prolongation of the QRS duration and attenuation and delay of the high-amplitude near-field ventricular electrogram in the RV HB region. The low-amplitude far-field ventricular electrogram preceding the QRS onset was then separated from the near-field ventricular electrogram after the QRS onset in the RV HB region (Fig. 2). Successful radiofrequency ablation was achieved in the NCC, where
the local ventricular activation was recorded simultaneously with the activation of the far-field ventricular electrogram in the RV HB region (Fig. 2). The amplitude of the atrial deflection in the local electrogram was greater than that of the ventricular deflection only when the site of origin was within the NCC (Fig. 2). During 13 of 14 RCC VAs (the

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<th>Table 1</th>
<th>Basic Demographics of the Patients With Successful Ablation</th>
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<tr>
<td>Origin</td>
<td>Age (yrs)</td>
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<tr>
<td>Ao root (n = 44)</td>
<td>53 ± 14</td>
</tr>
<tr>
<td>RVOT (n = 188)</td>
<td>54 ± 15</td>
</tr>
<tr>
<td>LVOT (n = 10)</td>
<td>59 ± 6</td>
</tr>
<tr>
<td>LV epi (n = 8)</td>
<td>49 ± 15</td>
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<tr>
<td>PA (n = 3)</td>
<td>54 ± 14</td>
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AF = atrial fibrillation; AFL = atrial flutter; Ao = aortic; AP = angina pectoris; Con. = conventional mapping; DM = diabetes mellitus; EA = electro-anatomic mapping; epi = epicardium; F = female; HL = hyperlipidemia; HT = hypertension; LV = left ventricular; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; M = male; NSVT = nonsustained ventricular tachycardia; PA = pulmonary artery; PSVT = paroxysmal supraventricular tachycardia; PVC = premature ventricular contraction; RVOT = right ventricular outflow tract; SSS = sick sinus syndrome; SVT = sustained ventricular tachycardia; VT = ventricular tachycardia.

Radiofrequency applications delivered near the His bundle (HB) region where the local ventricular activation preceded the QRS onset by 25 ms (left panel) caused a slight change in the QRS morphology with the prolongation of the QRS duration and an attenuation and delay of the high-amplitude near-field ventricular electrogram in the HB region (double arrowheads, center panel). The low-amplitude far-field ventricular electrogram (single arrowheads, center panel) preceding the QRS onset was then separated from the near-field ventricular electrogram after the QRS onset in the HB region. Successful radiofrequency ablation was achieved in the noncoronary cusp (NCC) where the local ventricular activation was recorded simultaneously with the activation of the far-field ventricular electrogram in the HB region (center panel). Note that the atrial electrogram with an amplitude greater than that of the ventricular electrogram was recorded at the successful ablation site during sinus rhythm (right panel). HBd = distal electrode pair of the His catheter; HBp = proximal electrode pair of the His catheter; VT = ventricular tachycardia; other abbreviations as in Figure 1.
exception being 1 patient in which radiofrequency ablation was first attempted at the RV HB region), a low-amplitude far-field ventricular electrogram preceding the high-amplitude near-field ventricular electrogram was recognized in the RV HB region (Fig. 3). In only 3 of 14 RCC VAs, an HB electrogram was recorded at the successful ablation site (Fig. 3). During the catheter ablation of those VAs, no junctional rhythm or AH or HV delays were observed.

In none of the cases with aortic root VAs could a sustained VT be terminated during successful radiofrequency applications. Even in the cases with clinically documented sustained VT, the induction of sustained VT was not always attempted if frequent PVCs with a QRS morphology identical to that of the sustained VT were observed during the electrophysiologic study. In some cases, sustained VT terminated during mapping probably because of a mechanical effect (Fig. 4), and thereafter only PVCs were induced.

Comparison of the clinical, electrocardiographic, and electrophysiologic parameters in the VAs originating from the aortic root. The results of the clinical, electrocardiographic, and electrophysiologic parameters of the VAs with their determined origins are given in Table 2. There were no significant differences in the clinical characteristics or standard electrocardiographic parameters (such as the morphology [in leads I and aVL as well] or precordial transition) between the origin sites within the aortic root (Fig. 5). However, a right bundle branch block and right inferior axis QRS morphology was
observed only in the VAs with an LCC origin, and an R-wave in lead aVL was observed only with an origin in the NCC. The maximal amplitude of the R-wave in the inferior leads was significantly greater in the LCC VAs than in the RCC VAs (p < 0.05) (Fig. 5). The ratio of the R-wave amplitude in leads II and III (III/II ratio) was significantly greater for the VAs with an LCC origin than for those with an RCC origin (p < 0.01) and was significantly smaller for the 1 NCC origin than for the other sites (p < 0.0001) (Fig. 5). Comparing origins in the LCC and RCC, a III/II ratio of >0.9 predicted an LCC origin with a sensitivity of 100%, specificity of 64.2%, positive predictive accuracy of 80.0%, and negative predictive accuracy of 100%.

There were no significant differences in the electrophysiologic parameters such as local ventricular activation time relative to the QRS onset, amplitude of the atrial or ventricular electrograms at the successful ablation site, or duration or number of radiofrequency applications needed for successful ablation between the sites of origin in the aortic root. The local ventricular activation time relative to the QRS onset at the RV HB region was significantly greater for the VAs with LCC and L-RCC origins than for those with RCC and individual NCC origins (p < 0.0001). The amplitude ratios in the atrial and ventricular electrograms were significantly greater for the 1 NCC origin than for the other sites (p < 0.0001).

**Complications.** Sinus bradycardia followed by complete AV conduction block occurred in 1 patient with PVCs with an RCC origin, in whom an HB electrogram was not recorded at the successful ablation site. Both sinus node function and AV conduction recovered soon after termination of radiofrequency delivery in this patient. No other complications occurred. Coronary arteriograms demonstrated no change in the coronary artery flow or diameter after the ablation compared with baseline.

**Discussion**

This study revealed that VAs more commonly originate from the LCC than from the RCC and are rare in the NCC. Anatomic studies may provide the rationale for those findings (13,14). The aortic root and the mitral valve form direct contact with the ostium of the left ventricle (Fig. 6). The aortic root consists of 3 sinuses of Valsalva, 2 of which (the right and left sinuses) make direct contact with the ostium of the left ventricle. Spatially, the aortic root occupies a central location within the heart, with the NCC anterior and superior to the paraseptal region of the left and right atria close to the superior atroventricular junctions. In all normally structured human hearts, the NCC is adjacent
to the atrial myocardium on the epicardial aspect and does not directly contact the ventricular myocardium of either the right or the left ventricle (Fig. 5). Indeed, atrial tachycardias can be ablated within the NCC (15). Therefore, in normal human hearts, VAs should not arise from the NCC, and the present case of a successful ablation of the VA within the NCC is quite unusual. The electrogram recorded at the ablation site demonstrated a very small ventricular potential and a large atrial potential, as would be expected at this location. It seems likely that the ablation lesion may have extended to the posterior portion of the RCC, thereby ablating a portion of the ostium of the left ventricle. It is also possible that this case represented an anatomic variant in which the LV myocardium was more closely apposed to the NCC than normally.

This study included VAs arising from the L-RCC as originating from the aortic root because of several considerations. Because of the semilunar nature of the attachments of the aortic valvular cusps, there are 3 triangular extensions of the aortic root that reach to the level of the sinotubular junction of the aorta, located approximately 1 cm above the base of either cusp (Fig. 6). These extensions are bound by thin fibrous walls of the aorta between the expanded sinuses. At the base of the LCC and RCC, the ventricular myocardium of the ostium of the left ventricle comes in direct contact with the aorta (Fig. 6). It is quite likely that ventricular arrhythmias that can be ablated within the base of the LCC and RCC actually arise from the most superior portion of the ostium of the left ventricle. Although the ventricular myocardium at the L-RCC forms the ostium of the left ventricle at the same level as that connecting with the LCC and RCC, it is located beneath the true anatomic junction of those cusps (Fig. 6), as confirmed by our practical findings in the catheter ablation of the L-RCC VAs. Actually, the present study demonstrated that the clinical, electrocardiographic, and electrophysiologic characteristics of the L-RCC VAs were similar to those of the LCC or RCC VAs. The site of the ablation of those L-RCC VAs provides an insight into all arrhythmias arising from the LVOT. Although some arrhythmias can be ablated within the LCC or RCC, those should not be considered to be true ASC VAs; rather, those VAs, whether approached by a catheter from above or below the aortic valvular annulus, should be considered to arise from the ostium of the left ventricle.
Anatomic studies have revealed a close relationship between the RCC or NCC and the HB (5,15). The posterior part of the RCC is adjacent to the central fibrous body, which carries within it the penetrating portion of the HB. Anteriorly, the RCC is related to the bifurcating atroventricular bundle and the origin of the left bundle branch. The NCC lies superior to the central fibrous body through which the HB penetrates. The present study demonstrates that the local ventricular activation time relative to the QRS onset at the RV HB region might be an electrophysiologic clue for differentiating VA origins in the LCC and L-RCC from those in the RCC and NCC. In catheter ablation of those arrhythmias, the HB catheter may be a useful landmark for mapping within the RCC and NCC. During catheter ablation within the RCC and NCC, the proximity of the RCC and NCC to the HB should be kept in mind to avoid inadvertent damage to the AV conduction system.

The electrocardiographic parameters may not be helpful in differentiating between VAs with an origin in each aortic root site, probably because those sites were located contiguous to each other in the limited space. However, the present study may provide several electrocardiographic algorithms for differentiating VAs originating from the aortic root. An R-wave in lead aVL during the VAs may exclude an origin in the LCC, RCC, or L-RCC. A right bundle branch block QRS morphology during the VAs may suggest that the VAs never originate from the RCC, NCC, or L-RCC. For the 2 major sites of origin within the aortic root (LCC and RCC), the R-wave amplitude ratio in leads II and III may be the most helpful discriminating factor. The activation time in the HB region may be helpful for explaining the last 2 electrocardiographic characteristics.

Although the activation time at successful ablation sites did not differ significantly between the LCC and RCC VAs, in the HB region it was significantly earlier during RCC VAs than LCC VAs. These findings suggest that the activation from the RCC may propagate to the ventricular septum and right ventricle before the LV free wall, whereas that from the LCC may do so after part of the LV free wall activates. Although the LCC and the RCC are located adjacent to each other, the horizontal vectors of the activation from those sites may be in opposite directions.

Serious complications, such as chronic left main coronary artery occlusions (16) or aortic regurgitation, may be possible during catheter ablation of aortic root VAs. However, these complications may be prevented if radiofrequency ablation is performed using the technique described in the present study, where the only complication was transient sinus bradycardia followed by transient complete AV conduction block, which occurred during radiofrequency ablation in the RCC. Anatomic studies have demonstrated that the anteriorly situated RVOT passes slightly superior to and leftward of the aortic valve and there is an anterior epicardial fat pad containing parasympathetic ganglia between the pulmonary infundibulum and the RCC (5,14,17). Therefore, radiofrequency energy deliveries from the RCC may have a thermal effect on the anterior epicardial fat pad, resulting in vagal stimulation.

There are several novel findings of the present study compared with earlier reports. First, this is the first report to propose the new concept of VAs originating from the aortic root as a part of the ventricular ostium. This concept is supported by computerized tomography images illustrating a continuous anatomic opening into the left ventricle with
the attachments of the aortic root anteriorly and the mitral annulus posteriorly. The ostium of the left ventricle is divided centrally by the contiguous structures the NCC of the aorta and the anterior leaflet of the mitral valve. Second, this study confirms that the anatomic features of the aortic root explain the far higher prevalence of VAs from the LCC and RCC than from the NCC. Although there have been a few reports describing NCC VAs (18,19), none have included local electrograms at the successful ablation site. Furthermore, the 12-lead electrocardiograms during the proposed NCC VAs exhibited very similar characteristics to those of the RCC VAs in this study. Therefore, we believe that the NCC VAs in earlier reports might have actually originated from the RCC. The careful radiographic imaging used in the present study is likely to provide a more accurate description of the prevalence of the aortic root VAs. And third, whereas most of the earlier studies contrasted the electrocardiographic and electrophysiologic characteristics of the aortic root VAs with those arising in the RVOT or LVOT (5,6), the present study compared those features for VAs confined to the aortic root.

**Study limitations.** The fact that there was only 1 NCC origin precludes meaningful statistical comparisons with the LCC and RCC origins. However, the mere fact that only 1 of 44 origins in the aortic root was found in the NCC attests to the rarity of this location as a site for the aortic root VA origin.

**Conclusions**

This study revealed that the LCC is the most common ablation location of aortic root VAs, followed by the RCC and the L–RCC. The NCC is rarely the site of origin. The ratio of the R-wave amplitude in leads II and III and the local ventricular activation time relative to the QRS onset at the HB region were helpful for differentiating an origin within the LCC from that within the RCC.

**Reprint requests and correspondence:** Dr. Takumi Yamada, Division of Cardiovascular Disease, University of Alabama at Birmingham, VA B147, 1670 University Boulevard, 1530 Third Avenue South, Birmingham, Alabama 35294-0019. E-mail: takumi-y@fb4.so-net.ne.jp.

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**Key Words:** ventricular arrhythmia • aortic root • prevalence • characteristics • radiofrequency catheter ablation.