Ubiquitous Myocardial Extensions Into the Pulmonary Artery Demonstrated by Integrated Intracardiac Echocardiography and Electroanatomic Mapping
Changing the Paradigm of Idiopathic Right Ventricular Outflow Tract Arrhythmias

Christopher F. Liu, MD; Jim W. Cheung, MD; George Thomas, MD; James E. Ip, MD; Steven M. Markowitz, MD; Bruce B. Lerman, MD

Background—Idiopathic ventricular arrhythmias of left bundle branch block inferior axis morphology are usually localized to the right ventricular outflow tract (RVOT), presumably below the pulmonic valve (PV). However, the PV location is usually not confirmed by direct visualization.

Methods and Results—Intracardiac echocardiography was used to visualize and tag the PV annulus, which was then integrated with 3-dimensional voltage maps of the RVOT. Distances were measured from the furthest extent of myocardial signal (bipolar voltage ≥1.5 mV) to the PV annulus. This was performed in 24 control patients and 24 prospective patients with RVOT arrhythmias. Myocardial signal beyond the PV was found in 92% of controls and 88% of RVOT arrhythmia patients (P=1.000). Average myocardial extension was further on the septal side than on the free wall side for control patients (5.6 mm; interquartile range [IQR], 3.6–7.7, versus 1.7 mm; IQR (−)0.1 to (+)4.0; P=0.002) and RVOT arrhythmia patients (5.7 mm; IQR, 2.7–7.7, versus 1.4 mm; IQR, (−)0.8 to (+)4.8; P=0.004). Eleven (46%) RVOT arrhythmia foci were localized beyond the valve in the pulmonary artery (median 8.2 mm above PV; IQR, 6.6–10.3 mm); these locations were confirmed as supravalvular by direct intracardiac echocardiography visualization.

Conclusions—Myocardial voltage extension into the pulmonary artery in humans is ubiquitous and can be demonstrated in vivo using 3-dimensional integrated intracardiac echocardiography to localize the PV. These extensions frequently serve as origins of presumed RVOT arrhythmias; intracardiac echocardiography localization of the PV allows reclassification of these as pulmonary arterial arrhythmias. (Circ Arrhythm Electrophysiol. 2014;7:691-700.)

Key Words: intracardiac imaging techniques ■ pulmonary artery ■ pulmonary valve ■ ventricular tachycardia

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ultrasound has been validated in animal models to be within 1.1±1.1 mm of actual location. This modality is often used in obtaining left atrial anatomy for ablation of atrial fibrillation as well as for localizing aortic cusps for ablation of ventricular arrhythmias originating from this region. Placement of the ICE catheter into the right ventricle allows direct visualization of the PV, and echocardiography can be used to tag the PV for integration into the 3-dimensional electroanatomic map. In this study, we assessed the relationship of myocardial tissue presence—as detected by voltage mapping—to the anatomic PV annulus, which is defined by 3-dimensional integrated echocardiographic mapping, in patients with RVOT-type ventricular arrhythmias and in a control group. In so doing, we sought to determine the presence of myocardial signal in the proximal PA and to localize the precise sites of origin of RVOT arrhythmias with respect to the PV.

**Methods**

**Study Population**

For the control group (group 1), we examined 24 consecutive patients with structurally normal hearts who presented for electrophysiology study and ablation of supraventricular arrhythmias; these patients were not known to have RVOT arrhythmias. In the RVOT arrhythmia group (group 2), we prospectively examined 24 consecutive patients undergoing catheter ablation of idiopathic ventricular arrhythmias originating from the region of the RVOT (left bundle branch block morphology, inferior frontal axis, precordial transition V3 or later). Only patients undergoing ablation were included. All procedures were performed following institutional guidelines of the Weill Cornell Medical Center/New York Presbyterian Hospital, and all patients gave written informed consent. The study was approved by the institutional review board of Weill Cornell Medical College.

**Voltage Mapping**

During sinus rhythm, we obtained a detailed 3-dimensional electroanatomic voltage map of the RVOT region in each patient. At least 30 points were taken from the tricuspid inflow to the proximal PA using either a 4-mm solid tip (Navistar) or 3.5-mm irrigated tip (Navistar Thermocool) catheter and the CARTO XP or CARTO3 system (Biosense Webster Inc, Diamond Bar, CA). The interelectrode distance was 2 mm. Signals were filtered at 30 to 500 Hz and were displayed at 200 mm/s. Maps were displayed with local bipolar voltage color thresholds 0.5–1.5 mV. Since the initial report by Marchlinski et al, bipolar voltage threshold of ≥1.5 mV has been used to delineate normal ventricular myocardium. Therefore, care was taken to collect the most superior (distal) points with bipolar voltage ≥1.5 mV; this was done circumferentially around the RVOT/PV region, and these most distal points were used to define the extent of normal myocardium by voltage. The maps were obtained in gated mode, and care was taken to ensure that all voltage points were gated with sinus rhythm beats, because PVCs are known to shift the location significantly compared with sinus rhythm.

**Echocardiography-Rendered Anatomic Mapping of the PV**

In each patient, a separate anatomic PV map using 3-dimensional integrated ICE was also created. The ICE-rendered anatomic map was created in a blinded fashion as the voltage map was hidden from view. A 10F phased-array ICE catheter (SoundStar; Biosense Webster Inc) was positioned in the heart from femoral venous access with the tip advanced across the tricuspid valve into the right ventricle, and the imaging plane was rotated to scan cranially, allowing direct visualization of the PV and PA. The ICE catheter and associated scanning sector were then rotated in small increments to scan the entire PV annulus. The visualized valve hinge points (±20 circumferential points for each patient) were tagged and registered to the 3-dimensional anatomic map via CartoSound (Biosense Webster Inc). The anatomic points were acquired using the same ECG gating (sinus rhythm QRS complex) as the voltage map. Respiratory gating was also applied.

**Voltage and Anatomic Correlation**

After the voltage and ultrasound maps were obtained independently, we superimposed the 2 respective maps for each patient. For purposes of analysis, the PV was divided into 6 cross-sectional segments: septal 1 (posterior), 2 (mid), 3 (anterior) and free wall (FW) 1 (posterior), 2 (mid), and 3 (anterior; Figure 1). This is similar to the previous scheme used by Dixit et al for classifying site of origin of RVOT arrhythmias. For each segment, we measured the shortest perpendicular distance from the ICE-defined anatomic annulus to the most distal electroanatomic point with bipolar voltage ≥1.5 mV (as surrogate for myocardial tissue). The distance was recorded as positive (+) if the voltage point was beyond the valve annulus (on the PA side), or negative (−) if the voltage point was proximal to the valve annulus (on the ventricular side). Figure 2 provides an example of this type of measurement for a point above the PV, with confirmed visualization by ICE. For purposes of comparing septal and FW sides, the average distances for the 3 septal segments and for the 3 FW segments in each patient were used.
Group 2: Electrocardiographic and Electrophysiological Characterization of Ventricular Arrhythmias

In patients with RVOT arrhythmias, after the initial voltage and ICE maps were created, an electrophysiology study was performed to assess for inducible sustained VT or increased ventricular ectopy. Following the baseline study, which included the introduction of up to triple ventricular extrastimuli from the right ventricular apex and RVOT, as well as burst pacing, the protocol was repeated during the infusion of isoproterenol. Isoproterenol was also infused alone (≤10 μg/min) to assess for increased ventricular ectopy or induction of VT. If sustained VT was induced, adenosine 12 mg was given in a rapid bolus and response of the VT was recorded. Analysis of the surface ECG during ventricular arrhythmia included QRS duration; R-wave amplitudes in leads II, III, and aVF; ratio of Q-wave amplitudes in leads aVR and aVL; ratio of R wave to S wave in lead V1; and the precordial R-wave transition zone (earliest lead with R>S).

Statistical Analysis

Continuous variables are expressed as mean±SD or as median (interquartile range [IQR]), depending on normality of distribution. Comparisons of continuous variables were made using 2-tailed Student t test for normal data and using the 2-tailed Mann–Whitney U test for non-normal data. Comparisons of categorical variables were made using Fisher exact test. Statistical calculations were performed using SPSS 16.0 (SPSS Inc, Chicago, IL). P value <0.050 was considered statistically significant.

Results

Twenty-four patients (17 men, 7 women; mean age, 50±16 years) with structurally normal hearts and supraventricular tachycardia comprise the control group (group 1). Twenty-four patients (10 men, 14 women; mean age, 50±19 years) underwent mapping and ablation of RVOT arrhythmias (group 2). The baseline characteristics of the 2 groups are shown in Table 1. Five patients in group 2 had left ventricular ejection fraction <50%.

Pulmonary Arterial Myocardial Extensions

In the control group, voltage maps were created with a mean of 57±20 contact points in the RVOT; anatomic PVs were defined using a mean of 30±6 circumferential annular anatomic electrocardiographic tags. The median distances (+ denotes myocardium extending above the valve) from the PV to the most superior site in each segment with a voltage ≥1.5 mV were: (+)3.9 mm (IQR, 0.6–6.6) in septal 1; (+)5.6 mm (IQR, 3.3–6.9) in septal 2; (+)6.9 mm (IQR, 4.5–11.1) in septal 3; 0 mm (IQR, –1.7 to (+)3.5) in FW 1; 0 mm (IQR, 0–3.5) in FW 2; and (+)2.9 mm (IQR, 0–7.6) in FW 3 (Figure 3A). Using the average distances, there was greater extension of myocardial voltage beyond the valve on the septal side (median average distance, 5.6 mm; IQR, 3.6–7.7) compared with the FW side (median average distance, 1.7 mm; IQR, –0.1).
In group 2 patients, voltage maps were created with a mean of 29±6 circumferential annular echo-cardiographic tags. The median distances from the PV to the RVOT sides (septal and free wall [FW] sides). Dark line indicates median; and box, interquartile range (IQR). (+) Distance denotes myocardium above valve; (−) distance denotes myocardium below valve.

As illustrated in Figure 3B, average myocardial extension was not significantly different between the control patients and RVOT arrhythmia patients. On the septal side, the median average myocardial voltage distance was 5.6 mm (IQR, 3.6–7.7) beyond the PV for control patients, compared with median average distance 5.7 mm (IQR, 2.7–7.7) for RVOT arrhythmia patients (P=0.726). On the FW side, the median average myocardial signal distance was 1.7 mm (IQR, −0.1 to (+)4.0) beyond the PV for control patients, compared with median average distance 1.4 mm (IQR, −0.8 to (+)4.8) for RVOT arrhythmia patients (P=0.672).

**Origin of RVOT Ventricular Arrhythmias (Group 2)**

Of the 24 patients who underwent mapping and successful catheter ablation in the RVOT region, 14 (58%) had their ventricular arrhythmia ablated on the septal side, whereas 10 patients had their arrhythmia ablated on the FW side. Eleven patients in the group (46%) had the origin of their ventricular arrhythmia mapped and ablated at myocardium that extended into the PA as visualized by ICE (group 2A). The median distance of the successful ablation site for these 11 patients was 8.2 mm (IQR, 6.6–10.3) beyond the valve (Figures 4–6; Movie in the Data Supplement). Seven of these sites were on the septal side, and 4 were on the FW side. Mean sinus rhythm voltage at the successful ablation site was 2.8±1.7 mV; 10 of the 11 (91%) successful ablation sites had sinus rhythm voltage ≥1.5 mV.

Of the remaining 13 patients in the RVOT group whose ventricular arrhythmias were not mapped above the valve (group 2B), 9 were ablated coincident with the PV annulus as visualized by ICE, and the remaining 4 were ablated below the PV (subvalvular RVOT). Mean voltage at the successful ablation site was 2.0±0.7 mV in group 2B.

During follow-up, all 24 patients in group 2 remained free of their targeted ventricular arrhythmia as determined by symptoms and 24-hour Holter monitoring (average follow-up of 14±11 months).

The patients with ventricular arrhythmias originating from supravalvular locations (group 2A) were found to have more extensive myocardium above the PV compared with the patients having arrhythmias originating from the valvar and subvalvular RVOT (group 2B; Figure 7A). On the septal side, the median average myocardial voltage distance was 7.0 mm (IQR, 5.0–8.3) beyond the PV for group 2A patients, compared with 2.7 mm (IQR, 0–6.0) for group 2B patients (P=0.007). On the FW side, the median average...
myocardial signal distance was 4.3 mm (IQR, 0.9–5.5) beyond the PV for group 2A patients, compared with 0 mm (IQR, −0.9 to +2.3) for group 2B patients (P = 0.059; Figure 7B).

The successful ablation sites were measured relative to the most distal extent of myocardium in their respective cross-sectional segments; (−) denotes distance below the extent of myocardial signal of 1.5 mV. In group 2A patients, the median distance from the ablation sites to the extent of myocardial signal was 0 mm, that is, the ablation site was precisely at the furthest extent of the myocardium (IQR, −3.9 to 0 mm). In group 2B patients, the median distance was also 0 mm (IQR, −1.7 to 0). These distances were not significantly different between 2A and 2B patients (P = 0.332), that is, whether the actual ablation site was supravalvular or subvalvular.

**Clinical and ECG Characteristics of Pulmonary Arterial Ventricular Arrhythmias**

The clinical and electrocardiographic properties associated with ventricular arrhythmias ablated above the PV are summarized in Table 2. The average QRS duration of the ventricular arrhythmias originating above the PV (group 2A) was 161±29 ms and was not significantly different from those originating at or below the valve (160±18 ms; P = 0.948). There was no significant difference with regard to R-wave amplitude in any of the inferior leads (P = 0.861–0.949), nor in the Q-wave ratio of aVR to aVL (P = 0.685) and the R/S ratio in lead V2 (P = 0.664). Precordial transition zone at V3 or earlier was seen in 3 of 11 patients in group 2A, compared with 7 of 13 patients in group 2B, but this did not reach statistical significance (P = 0.240).

**Electrophysiological Characteristics of Pulmonary Arterial Ventricular Arrhythmias**

The electrophysiological and ablation findings of the ventricular arrhythmias originating above the PV (group 2A) are summarized in Table 3. Two patients had inducible sustained VT, both of which terminated in response to adenosine. A third patient had bursts of nonsustained VT inducible on high-dose isoproterenol that terminated with adenosine. Catecholamine facilitation of the ventricular arrhythmias was seen in 8 of 11 patients with pulmonary arterial origin (group 2A), compared with 11 of 13 patients with subvalvular RVOT origin (group 2B; P = 0.630). Comparing group 2A and group 2B, there were no significant differences in the earliest activation times, best pace match leads, number of ablation attempts, or sinus rhythm voltage at the successful ablation sites (Table 4). No discrete multicomponent potentials were observed in any of the successful ablation sites of group 2A or 2B.

**Discussion**

The principal finding of this in vivo study is that myocardial tissue nearly always extends beyond the PV into the PA and can be the focal source of VT or PVCs in ≈50% of unselected...
patients with RVOT arrhythmias. To our knowledge, this is the first prospective study demonstrating the incidence of pulmonary arterial origin of idiopathic RVOT-type arrhythmias. There was no difference in the degree of pulmonary arterial myocardial extension between control patients and RVOT arrhythmia patients. Furthermore, there were no discrete ECG findings that distinguished the source of arrhythmia as above or below the PV. Our findings challenge conventional understanding of the anatomic relationship between myocardial tissue in the RVOT and the PA, as well as the precise source of RVOT arrhythmias in many patients. Because most cases of RVOT-type arrhythmias are thought to arise from the subvalvular aspect of the RVOT, ventricular arrhythmia originating from the PA is now shown to be more frequent than previously recognized.

The extension of myocardial tissue into the great vessels is commonly observed, as seen in the superior vena cava,
Idiopathic ventricular arrhythmias are frequently seen to originate from the aortic sinuses of valsalva and aortic root. This phenomenon is relatively easy to appreciate because these arrhythmias are usually mapped via the retrograde approach, and hence the location of the catheter is easily seen to be at or above the aortic valve fluoroscopically. In contrast, because the RVOT is routinely mapped with catheter advancement in the anterograde direction (from right ventricle to PA), there is no fluoroscopic landmark for identifying the PV. Hence, during conventional catheter mapping, local electrogram amplitude is typically used to define the location of the PV, and loss of local electrogram signal with catheter advancement into the distal RVOT is presumed to indicate passage beyond the PV into the PA. This method assumes that myocardium extends precisely to the level of PV and not beyond. Pulmonary angiography has been used in previous series to determine catheter location with respect to the PV. However, because the PV is tilted, standard angiographic views may not accurately reflect the relationship of the catheter to the valve.

ICE is often used in mapping and ablation of arrhythmias from the outflow tract region and has been previously described as a method to visualize the ablation catheter in
the PA. In our study, the integration of PV anatomy from ICE and myocardial electrophysiology from voltage mapping allowed us to demonstrate contiguity of RVOT myocardium with pulmonary arterial myocardium and additionally allowed quantitative measurement of these myocardial extensions. Autopsy studies focusing on the prevalence of myocardium extending beyond the PV have also shown that this is a common finding, occurring in ≤74% of hearts. In these series of autopsy specimens from patients without known arrhythmias, myocardium extended for average distances of 3.2 to 4.0 mm above the PV into the PA. These reports are consistent with the findings in our study.

We did not find the extension of myocardial signal into the PA to be statistically different between the control patients (group 1) and RVOT arrhythmia patients (group 2). This suggests that myocardial extension alone may not predict the occurrence of idiopathic ventricular arrhythmias. In both control patients and RVOT arrhythmia patients, we found that the myocardial signal extended a greater distance above the PV on the septal side compared with the FW side. We postulate that this may be because of tilting of the PV, that is, the septal side of the valve attaches more caudally, whereas the FW side attaches more cranially (closer to the extent of myocardium; Figure 4).

Although the phenomenon of ventricular arrhythmias originating from the PA has been appreciated, it is thought to be an uncommon finding. The paucity of reports describing this finding affirms its supposed novelty. However, our study shows that this phenomenon is more common than previously recognized. In our prospective cohort of consecutive RVOT arrhythmia patients, nearly half of the RVOT arrhythmias were ablated in the

Table 3. Electrophysiological Characteristics of RVOT Arrhythmias Ablated Above the Pulmonic Valve

<table>
<thead>
<tr>
<th>Patient</th>
<th>Clinical Arrhythmia</th>
<th>Isoproterenol Response (Increased Ectopy)</th>
<th>Inducible VT</th>
<th>R-Wave Amplitude in Inferior Leads, mV</th>
<th>Q-Wave Ratio in aVR/aVL</th>
<th>R/S Ratio in Lead V2</th>
<th>Successful Ablation Site</th>
<th>Distance of Ablation Site Above PV, mm</th>
<th>Ablation Site Voltage in Sinus Rhythm, mV</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>PVC</td>
<td>+</td>
<td>–</td>
<td>2</td>
<td>2.1</td>
<td>2</td>
<td>FW 2</td>
<td>8</td>
<td>4.4</td>
</tr>
<tr>
<td>7</td>
<td>PVC</td>
<td>+</td>
<td>–</td>
<td>2</td>
<td>2.1</td>
<td>2</td>
<td>FW 2</td>
<td>8</td>
<td>4.4</td>
</tr>
<tr>
<td>8</td>
<td>NSVT</td>
<td>+</td>
<td>Sustained</td>
<td>Isop plus AP or VP</td>
<td>Termination</td>
<td>–46</td>
<td>Septal 3</td>
<td>11</td>
<td>4.6</td>
</tr>
<tr>
<td>11</td>
<td>PVC</td>
<td>+</td>
<td>Nonsustained</td>
<td>Isop plus VES</td>
<td>Termination</td>
<td>–18</td>
<td>Septal 3</td>
<td>13</td>
<td>2.3</td>
</tr>
<tr>
<td>18</td>
<td>PVC</td>
<td>–</td>
<td>–</td>
<td>–18</td>
<td>11</td>
<td>4</td>
<td>Septal 2</td>
<td>6</td>
<td>2.8</td>
</tr>
<tr>
<td>19</td>
<td>NSVT</td>
<td>+</td>
<td>Sustained</td>
<td>Isop plus VP</td>
<td>Termination</td>
<td>–18</td>
<td>Septal 3</td>
<td>9</td>
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<tr>
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<td>NSVT</td>
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<td>11</td>
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<td>Septal 2</td>
<td>6</td>
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<tr>
<td>21</td>
<td>PVC</td>
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<td>–21</td>
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<td>Septal 3</td>
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<td>22</td>
<td>PVC</td>
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<td>Septal 2</td>
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<tr>
<td>23</td>
<td>PVC</td>
<td>+</td>
<td>–</td>
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<td>11</td>
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<td>FW 3</td>
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<tr>
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<td>PVC</td>
<td>+</td>
<td>–</td>
<td>–34</td>
<td>12</td>
<td>3</td>
<td>FW 3</td>
<td>4</td>
<td>1.5</td>
</tr>
</tbody>
</table>

AP indicates atrial pacing; EAT, earliest activation time; FW, free wall; Iso, isoproterenol; NSVT, nonsustained ventricular tachycardia (VT); PV, pulmonic valve; PVC, premature ventricular contraction; RF, radiofrequency; RVOT, right ventricular outflow tract; VES, ventricular extrastimuli; and VP, ventricular pacing.
SA. The ECG and ablation characteristics were not significantly different from the arrhythmias ablated in the subvalvular RVOT proper. This suggests that many of the currently understood RVOT-type (left bundle branch block, inferior axis) arrhythmias are in fact of pulmonary arterial origin. Indeed, of the 11 patients with RVOT-type arrhythmias ablated in supravalvular locations, 10 (91%) exhibited sinus rhythm voltage ≥1.5 mV that was contiguous with RVOT myocardium. Using conventional mapping criteria, these sites would have been assumed to be RVOT sites below the PV, and the pulmonary arterial locations would not have been recognized without visualization of the valve.

In the previous series reported by Sekiguchi et al., ventricular arrhythmias originating in the PA demonstrated distinguishing ECG characteristics— including larger R-wave amplitudes in the inferior leads, larger Q-wave ratio in aVL/aVR, and R/S ratio in V2—compared with arrhythmias originating in the subvalvular RVOT. These ECG differences were not observed in our series, likely because of methodological differences for identifying the pulmonary arterial arrhythmias. Whereas prior published series used pulmonary arteriogram for defining the site of ablation in selected patients, we used integrated real-time echocardiography for direct visualization of the PV and ablation sites in unselected patients. As is the case for the aortic valve, 3-dimensional integrated echocardiography is a more precise method for defining the PV, which is anatomically tilted and therefore difficult to circumferentially define via standard angiographic views. As a result, it is likely that our study demonstrates a more sensitive method for identifying points above the PV than methods used in prior studies.

Previous work in developmental biology has shown that the RVOT develops from a secondary source of cardiomyocytes distinct from the primary heart field in both avian and mammalian systems. It has also been shown that outflow tract myocardium and proximal pulmonary trunk vascular smooth muscle derive from the same group of cells in the arterial pole of the heart, and a combination of cellular processes, including apoptosis and absorption, contributes to disappearance of myocardium from the pulmonary trunk during embryonic development. When these processes are incomplete or aberrant, residual myocardial tissue may persist above the PV in the adult heart. These developmental variations likely account for the observations made in our study and the prior autopsy series, suggesting that the final myocardial boundary between the outflow tract and proximal PA is not always precisely fixed at the PV.

Interestingly, patients with pulmonary arterial arrhythmias (group 2A) had a greater degree of myocardial extension compared with patients with nonpulmonary arterial arrhythmias (group 2B), but there is inherent selection bias in this post hoc analysis, and this may well represent normal variation in the extent of myocardium beyond the valve. It is more revealing that in the overall RVOT-type arrhythmia patients (group 2), the median distance from the successful ablation sites to the furthest extent of myocardium in the respective segment was 0 mm, that is, the successful ablation site was precisely at the location where the myocardial signal terminates. This was regardless of whether the arrhythmia was ablated in the PA (group 2A) or in the RVOT proper (group 2B). It is possible that myocardial termination adjacent to vascular tissue tends to be more arrhythmogenic, perhaps because of structural reasons; this hypothesis merits further study and may implicate pulmonic valvular tissue in the pathogenesis of idiopathic ventricular arrhythmias from the RVOT and PA.

Limitations
Voltage mapping is subject to bias toward finding less tissue extension beyond the valve, because poor tissue contact and undersampling will tend to favor underestimating the extent of myocardial tissue. Therefore, these measurements may in fact be an underestimate of the true myocardial extension present. Furthermore, the bipolar voltage threshold of >1.5 mV was established for normal myocardium in the ventricle proper. Although autopsy studies have shown small strands of myocardium extending above the PV, their voltage characteristics are unknown. Some of these thinner strands may go undetected if their electrogram amplitude is <1.5 mV, the voltage map threshold. However, this scenario would again bias the data toward underestimating the true extent of supravalvular tissue, thus further strengthening the conclusion that the presence of myocardial sleeves above the PV is a common and underappreciated phenomenon.

Conclusions
Integrated voltage and echocardiographic mapping shows frequent extension of right ventricular myocardium beyond...
the PV into the PA in vivo; this extension is greater on the septal side than the FW side and occurs in humans without ventricular arrhythmias. A significant proportion of idiopathic RVOT-type ventricular arrhythmias originate from this PA myocardium. The pulmonary arterial origin of these arrhythmias can be more readily recognized with real-time echocardiographic localization of the PV.

Disclosures
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CLINICAL PERSPECTIVE
Idiopathic focal ventricular arrhythmias with left bundle branch block inferior axis morphology typically occur in patients with structurally normal hearts and are commonly mapped to the right ventricular outflow tract (RVOT). Other focal arrhythmias have been found to cluster around valvular tissue (tricuspid, mitral, and aortic valves) or myocardial extensions into large vessels attached to the heart (pulmonary veins, superior vena cava, aortic root). Although arrhythmias originating from myocardial extension into the pulmonary artery (PA) have been previously demonstrated in small retrospective series, the actual prevalence is unknown, because the pulmonic valve is not usually visualized during catheter ablation procedures. In this study, we prospectively performed detailed voltage mapping of the entire RVOT region and proximal PA and used 3-dimensional integrated intracardiac echocardiography to tag the pulmonic valve, thereby clearly delineating the boundary separating the RVOT and the PA. We found that, based on electrogram voltage, the vast majority of control patients (without ventricular arrhythmias) demonstrated at least 1 segment of myocardial extension beyond the pulmonic valve into the PA, which corroborates previous autopsy studies. Similar findings were seen in unselected patients with RVOT-type arrhythmias. Almost half of typical-appearing RVOT-type arrhythmias were ultimately ablated in PA myocardium, as confirmed by intracardiac echocardiography. The recognition of the frequent origin of ventricular arrhythmias in the PA suggests a possible causative role for the ubiquitous PA myocardial extensions in the pathogenesis of idiopathic RVOT ventricular arrhythmias.

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SUPPLEMENTAL MATERIAL

Supplemental Video. Patient 24. **Left:** Superimposed voltage map and ICE-defined pulmonic valve annulus (green). Site of successful ablation (dark red dot) is above the pulmonic valve.

**Right:** Ablation catheter is seen in ICE image to be well beyond the pulmonic valve in the pulmonary artery (ICE catheter in right ventricle imaging upward at pulmonary artery).